Biopsychosocial Factors in Chronic Spine-Related Pain: Contributions to Pain Intensity and Perceived Disability

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Biopsychosocial Factors in Chronic Spine-Related Pain: Contributions to Pain Intensity and Perceived Disability

A Dissertation

Submitted to the Graduate Faculty of the University of New Orleans in partial fulfillment of the requirements for the degree of

Doctor of Philosophy in Applied Biopsychology

by

Jonathan S. Ord

B.S. Evergreen State University, 2003
M.S. University of New Orleans, 2007

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ABSTRACT

Psychological and contextual factors play an important role in the development and maintenance of chronic spine-related pain, and effective treatment of pain-related conditions requires an understanding of how these factors contribute to pain and disability. The present study examined the relative contributions of spine pathology, psychological complications, and demographic factors to perceived pain intensity and disability in patients with chronic spine-related pain. Because most patients were assessed in the context of a compensable injury, exaggeration of symptoms and disability was systematically controlled for using multiple validity indicators. A high prevalence of psychological complications was observed in the present sample. Analysis indicated that psychological factors were not significantly related to pain intensity, but were significantly related to reported pain-related disability. Further, psychological factors were found to predict pain-related disability beyond demographics, medical findings, and pain intensity. Clinical implications of these findings are discussed.

Keywords: chronic pain; disability; outcome; psychological factors; biopsychosocial model
INTRODUCTION

The International Association for the Study of Pain (IASP) defines pain as “an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage” (Merskey & Bogduk, 1994, p. 210). Importantly, the IASP’s definition considers pain a subjective experience influenced by both psychological and contextual factors that is not necessarily dependent on tissue damage or specific nociceptive activation. Pain is usually termed chronic when symptoms do not follow the natural course of healing after injury or persist for longer than three months without biological value (Merskey & Bogduk, 1994). As described by Pappagallo and Werner (2008), “Acute pain ordinarily has a useful purpose, such as signaling damage or that something is wrong. By contrast, chronic pain has no such value, but is a disease in its own right, causing widespread suffering, distress, and disability” (p. 17).

The estimated prevalence of chronic pain in the U.S. is between 10% and 20% in the general population (Verhaak, Kerssens, Dekker, Sorbi, & Bensing, 1998; Von Korff et al., 2005) and 20% to 25% in primary care patients (Gureje, Von Korff, Simon, & Gater, 1998). An estimated 70% to 80% of adults experience an episode of spine-related pain (Andersson, 1998; Frymoyer, 1988) and approximately 29% of Americans report experiencing chronic spinal pain sometime in their life (Von Korff et al., 2005). Americans spend an estimated $70 billion per year in healthcare costs related to chronic pain (American Academy of Pain Management, 2003) and back pain alone is thought to contribute to over 100 million lost workdays yearly (Guo, Tanaka, Halperin, & Cameron, 1999). The total economic burden of pain-related disability in the
U.S. has been reported to exceed $150 billion a year (Gatchel & Okifuji, 2006; Mayer, Gatchel, & Polatin, 2000).

Pain-related symptoms and disability are often not explained by physiological findings alone (Boden, Davis, Dina, Patronas, & Wiesel, 1990; Boden, McCowin, et al., 1990; Jarvik et al., 2005). Research has increasingly demonstrated that the development and maintenance of chronic pain and pain-related disability is a complex process involving interactions of biological, psychological, and social factors (see Gatchel, Polatin, & Mayer, 1995; Gatchel, 2005; Proctor, Gatchel, & Robinson, 2000). Understanding the role of each of these factors in chronic pain is vital for development of effective treatment and rehabilitation programs (Gatchel, Polatin, Mayer, & Garcy, 1994).

**Medical Factors in Chronic Spine-Related Pain**

Physical pathology has clear contributions to acute spine-related pain (Adams, 2004). Common pain-related spine pathologies include vertebral disc compromise, facet joint disorders, vertebral fracture, or musculoligamentous injuries (see Appendix A for brief descriptions of selected conditions). Spinal pathologies are thought to generate pain through mechanical or inflammatory processes that result in the activation or potentiation of nociceptive transduction or the disruption of nerve function (Adams, 2004; Brisby, 2006; Saal, 1995). When nerve disruption is involved, spine-related pain may radiate down limbs or be referred to other parts of the body (Zimmerman, 2001).

There are a variety of medical diagnostic techniques used to identify spine pathologies (see Appendix B for a selected review of procedures). However, diagnostic findings are not particularly predictive of pain-related symptoms, recovery, or disability. For example, findings
from physical examination are only moderately correlated with self-reported back pain and
disability (Michel, Kohlmann, Raspe, 1997). Spinal abnormalities on imaging, such as disc
protrusions or foraminal stenosis, are neither sensitive nor specific predictors of pain, as many
asymptomatic patients present with these findings, while many symptomatic patients do not
(Boden & Davis et al., 1990; Boden & McCowin et al., 1990; Boos et al., 1995; Jarvik,
Hollingworth, Heagerty, Haynor, & Deyo, 2001; Jensen et al., 1994). Moreover, spine findings
are not significant prognostic indicators for the development of future back pain (Borenstein et
al., 2001; Jarvik et al., 2005).

Psychosocial complications are relatively common in chronic pain patients and their
clinical relevance is becoming increasingly clear (Bellamy, 1997). In fact, a number of studies
have suggested that, compared to physiological findings or injury characteristics, psychosocial
factors are often stronger predictors of the transition from acute to chronic pain and are more
closely associated with the degree of disability experienced (e.g., Bigos et al., 1991; Carragee,
Alamin, Miller, & Carragee, 2005; Carroll et al., 2008; Jarvik et al., 2005; Shaw, Pransky,
Patterson, & Winters, 2005; Turner et al., 2004). Thus, effective clinical management of pain-
related conditions requires an understanding of the contributions of psychological and social
factors.

**Psychological Factors in Chronic Pain**

The contributions of psychological processes to the development and maintenance of
chronic pain have been well-established (see Gatchel, 2004a; Keefe, Rumble, Scipio, Giordano,
& Perri, 2004; Linton, 2000). Psychopathology – defined here as any maladaptive behavior that
causes impairment, distress, or disability – is prevalent in patients with chronic pain, with
approximately two-thirds presenting with a diagnosable mental disorder (Dersh, Gatchel, Mayer, Polatin, & Temple, 2006). The following sections will review the characteristics and prevalence of selected psychological constructs in patients with chronic pain and discuss their collective role in contributing to pain-related symptoms and hampering recovery.

**Characteristics and Prevalence of Psychopathology**

While research has revealed a number of affective, cognitive, and behavioral issues relevant to chronic pain, four constructs that are particularly essential to the clinical management of pain-related conditions will be examined: depression, anxiety/fear, somatization, and catastrophizing. It should be mentioned that although a discussion of prevalence inherently involves categorization into presence or absence of a condition, these constructs will generally be viewed here as dimensional processes with a continuum of severities, rather than discrete diagnostic entities.

**Depression**

As a construct, depression is characterized by mood and emotional disturbances (e.g., excessive sadness) and negative cognitions (e.g., hopelessness). While clinical manifestations of depression can vary, common symptoms include anhedonia, sleep irregularities, fatigue, and difficulty concentrating according to the Diagnostic and Statistical Manual – fourth edition – text revision (DSM-IV-TR; American Psychological Association [APA], 2000).

There is a strong association between depression and chronic pain, with studies showing a near linear relationship between self-reported pain and depressive symptoms (Carroll, Cassidy, & Cote, 2000; Currie & Wang, 2004). In the general population, estimates place the prevalence of major depression in persons reporting chronic pain at approximately 20% (Von
Korff et al., 2005). However, in patients seeking treatment at pain clinics or rehabilitation programs the prevalence of major depression is often over 50% (Dersh et al., 2006; Mayer, Towns, Neblett, Theodore, & Gatchel, 2008).

The relationship between depression and chronic pain is complex and reciprocal as: (a) there is some overlap between symptoms (e.g., sleep disturbances or reduced activity levels); (b) they may share physiological mechanisms, such as NE and 5-HT dysregulation (Bair, Robinson, Katon, & Kroenke, 2003); (c) the presence of either predicts future development of the other (Gureje, Simon, & Von Korff, 2001); and (d) comorbidity complicates treatment for both conditions (Moultry & Poon, 2009). Depression may be a particularly important predictor of pain-related disability (Alshuler, Theisen-Goodvich, Haig, & Geisser, 2008), with studies suggesting that depression may serve as a moderator for the relationships between other psychological vulnerabilities (discussed below) and self-perceived disability (Boersma & Linton, 2005, 2006).

**Anxiety/ Fear**

Anxiety refers to a generalized uneasiness or worry that is not associated with a particular stimulus (Rachman, 1998). This may include anticipation of unknown threats or concern about threats perceived to be uncontrollable or unavoidable. Approximately 15% of patients with chronic pain present with a diagnosable anxiety disorder (Dersh et al., 2006; Polatin, Kinney, Gatchel, Lillo, & Mayer, 1993). Anxiety can have a number of physiological effects, including increased sympathetic arousal (Cuthbert et al., 2003), and has been shown to have a significant effect on the perceived intensity of painful stimuli (Colloca & Benedetti, 2007).
Fear is a related construct that occurs in the presence of a specific, identifiable, immediate threat; often leading to escape or avoidance behaviors (Rachman, 1998). A fear-avoidance model of chronic pain-related disability has been proposed whereby physiological, behavioral, and cognitive elements contribute to a reinforcing cycle of fear and anxiety towards pain-related stimuli (Vlaeyen, Kole-Snijders, Boeren, & van Eek, 1995; Vlaeyen & Linton, 2000). Patients can experience fear of pain itself, reinjury, or specific activities such as movement (i.e., kinesiophobia). Avoidance of activities, in turn, can contribute to the development and maintenance of functional disability (Leeuw et al., 2007; Woby, Watson, Roach, & Urmston, 2004a).

**Somatization**

Somatization refers to a predisposition to use physical symptoms as a means of coping with emotional problems, resulting in a tendency to manifest and focus on physical complaints when dealing with stressful life events (Lamberty, 2008). As Gatchel (2004b) describes, “physical symptoms may be easier to accept as causing current unhappiness and discontent than admitting that some psychological reason is contributing to it” (p. 204). Somatization can be viewed as a maladaptive personality trait or coping style with elements of negative illness behaviors, such as symptom magnification, and excessive preoccupation or worry about illnesses (i.e. hypochondriasis). Somatization appears to play a particularly important role in the development and perception of medically unexplained pain-related symptoms (Block, Vanharanta, Ohnmeiss, & Guyer, 1996; McBeth, Macfarlane, Benjamin, & Silman, 2001).

Diagnoses based on the DSM-IV-TR are not particularly useful for establishing the prevalence of somatization in chronic pain considering that nearly all chronic pain patients
meet criteria for somatoform pain disorder while almost none meet criteria for somatization
disorder (Polatin et al., 1993; Dersh et al., 2006). However, studies examining profile patterns
on the Minnesota Multiphasic Personality Inventory-2 (MMPI-2; Butcher, Dahlstrom, Graham,
Tellegen, & Kaemmer, 1989) suggest that scale elevations associated with somatization may be
seen in more than half of patients with chronic pain (Riley, Robinson, Geisser, & Wittmer, 1993;
Nordin, Eisemann, & Richter, 2005; Porter-Moffitt et al., 2006).

Pain Catastrophizing

Pain catastrophizing refers to a tendency to exaggerate, focus on, and emphasize
negative aspects of painful conditions (Turner & Aaron, 2001). Catastrophizing is a complex
process that has at various times been characterized as a coping strategy, perception and
appraisal process, or set of beliefs (Sullivan et al., 2001). Factor analysis has revealed three
primary components of pain catastrophization: magnification, rumination, and helplessness
(Osman et al., 1997). Magnification refers to a tendency to exaggerate the threat of pain
sensations (e.g., “it will get worse and something serious may happen”); rumination refers to a
persistent tendency to focus on painful stimuli (e.g., “I can’t stop thinking about it”); and
helplessness refers to a feeling of being overwhelmed and lacking control over the pain (e.g.,
“It’s terrible and never going to get better”).

Catastrophizing tendencies are thought to reflect relatively persistent life-course traits
(Sullivan, Bishop, & Pivik, 1995). While no formalized criteria exist for diagnosing “pain
catastrophizing,” persons with chronic pain generally show higher levels of catastrophization
than those without chronic pain (Buer & Linton, 2002). Catastrophizing appears to play a
particularly important role in the perceived intensity of painful experiences and associated emotional distress (Sullivan et al., 2001).

**Contributions to Symptoms and Recovery**

Psychopathology serves as is an important prognostic indicator of cases that transition from acute to chronic pain (Carragee et al., 2005, Dersh, Gatchel, & Polatin, 2001; Keefe et al., 2004; Linton, 2000; Pincus, Burton, Vogel, & Field, 2002). Psychopathology can increase perceived pain intensity, hamper rehabilitation efforts, and magnify perceived disabilities; all of which serve to reinforce and perpetuate pain-related dysfunction (Gatchel & Dersh, 2002; Holzberg, Robinson, Geisser, & Gremillion, 1996; Leeuw et al., 2007).

**Pain Perceptions**

Psychological processes can have a direct impact on the experience of pain. Depression and catastrophization are associated with heightened pain intensity and lower pain thresholds (Averill, Novy, Nelson, & Berry, 1996; Sherman et al., 2004; Sullivan, Stanish, Waite, Sullivan, & Tripp, 1998; Turner, Jensen, Warms, & Cardenas, 2002). Somatization is associated with reporting pain and sensitivity in more body areas (McBeth, Macfarlane, Benjamin, Morris, & Silman, 1999; Sherman et al., 2004). Negative emotional states, such as anger and sadness, can increase the intensity and unpleasantness of a painful stimulus and provoke larger autonomic responses (Rainville, Bao, & Chretien, 2005). Anxious expectations of painful stimuli have been shown to directly facilitate biological pain transmissions (Colloca & Benedetti, 2007) and catastrophizing is associated with heightened sympathetic reactions (Edwards & Fillingim, 2005) and greater central nervous system sensitization in response to pain (Edwards, Smith, Stonerock, & Haythornthwaite, 2006).
Treatment & Rehabilitation

Psycyhopathology also influences the effectiveness of pain interventions and rehabilitation programs. Factors related to depression, anxiety, somatization, catastrophizing, fear-avoidance, and personality disorders are all prognostic for delays in returning to work following a back injury (Bigos et al., 1991; Gatchel, Polatin, & Kinney, 1995; Trief, Grant, & Fredrickson, 2000; Turner et al., 2006; Turner et al., 2007). Fear-avoidance beliefs are associated with fewer benefits from conservative pain interventions (Al-Obaidi, Beattie, Al-Zoabi, & Al-Wekeel, 2005) and poor outcome is strongly associated with factors like substance dependence or multiple comorbid psychopathologies, even in an interdisciplinary rehabilitation program (Dersh et al., 2007; Gatchel, Mayer, & Eddington, 2006; Maier & Falkai, 1999).

The effectiveness of more invasive medical procedures is also influenced by psychopathology. For example, higher pre-surgical anxiety is associated with slower recovery and more complications post-surgery (Kiecolt-Glaser, Page, Marucha, MacCallum, & Glaser, 1998). Even when patients are carefully selected on the basis of objective physical findings, pre-surgical psychological risk factors are a significant predictor of poor outcome (Voorhies, Jiang, & Thomas, 2007). In fact, a study of therapeutic injections to relive lower back pain found that while the treatment was effective in patients with low levels of psychopathology, patients with high psychopathology actually reported a mean worsening of pain following treatment (Wasan et al., 2009).

Self-Perceived Disability

Psycyhopathology also plays an important role in shaping self-perceived limitations and disabilities (Gatchel, 2004b). Concurrent psychopathology is associated with higher overall
levels of self-perceived disability (Alschuler et al., 2008; Mannion et al., 2001; Schiphorst Preuper et al., 2008). Moreover, depression, anxiety, somatization, catastrophizing, and fear-avoidance beliefs are all prognostic indicators of future self-reported disability (Dionne et al., 1997; Sullivan, Stanish, Sullivan, & Tripp, 2002; Trief et al., 2000; Woby et al., 2004a).

Importantly, changes in psychological distress and fear-avoidance beliefs over the course of recovery are shown to contribute to changes in self-rated disability even after controlling for factors such as pain intensity (Mannion et al., 2001; Woby, Watson, Roach, & Urmston, 2004b).

Self-perceived disability is a particularly important indicator because it not only predicts pain chronicity (Gatchel, Polatin, & Kinney, 1995), but current functional capacities as well (Alschuler et al., 2008). Thus, reducing self-perceived disability is essential to ending the escalating cycle of psychological and pain-related symptoms and restoring functional abilities. In fact, a study of physical and psychological contributions to outcome following a program of functional restoration by Hildebrandt, Pfingsten, Saur, and Jansen (1997) concluded that “the most important variable in determining a successful treatment of chronic low back pain is the reduction of subjective feelings of disability in patients” (p. 990).

**Etiology: Consequence or Cause?**

While the association between chronic pain and psychopathology is well-established, there has been some debate in the literature regarding the causal implications of this relationship (e.g., Dersh, Mayer, Theodore, Polatin, & Gatchel, 2007; Fishbain, Cutler, Rosomoff, & Rosomoff, 1997). More explicitly, the question is to what degree psychopathology is a consequence, or a cause, of chronic pain. A prospective study by Gureje et al. (2001) suggested that depression and anxiety predict the onset of pain to the same degree that pain predicts the
onset of depression or anxiety. Studies by Polatin et al. (1993) and Mayer et al. (2008) suggest that the onset of depression, in particular, may occur after the onset of chronic pain; but other examined psychopathologies did not show this effect.

There are reasons to believe that pre-existing psychological risk factors explain much of the relationship between psychopathology and pain-related symptoms and disability. Support for this view comes from research demonstrating that early childhood trauma and adversity predicts the onset of back pain in adulthood (Kopec & Sayre, 2005), poor outcome following back surgery (Schofferman, Anderson, Hines, Smith, & Keane, 1993; Schofferman, Anderson, Hines, Smith, & White, 1992) and retirement due to disability (Harkonmaki et al., 2007). Additionally, development of chronic pain is predicted by measures of psychiatric disorders (Hotopf, Mayou, Wadsworth, & Wessely, 1998) and personality (Bigos et al., 1991), even decades after assessment (Applegate et al., 2005).

A diathesis-stress model is emerging as an explanation for this relationship whereby pre-existing semi-dormant characteristics are exacerbated by the various stresses associated with injury and illness, leading to diagnosable psychopathology and difficulties recovering (Dersh, Polatin, & Gatchel, 2002). These preexisting vulnerabilities to psychological complications and chronic pain may be mediated by various psychological and biological mechanisms, such as attachment (Meredith, Strong, & Feeney, 2005, 2006, 2007) or hypothalamic-pituitary-adrenal axis dysfunction (Anderson, Orenberg, Chan, Morey, & Flores, 2008; McBeth et al., 2005; McBeth et al., 2007). However, regardless of the possible etiological pathways, consideration of concurrent psychological complications is important in patients with chronic pain (Gatchel et al., 1994; Hildebrandt et al., 1997).
Sociodemographic Factors in Chronic Pain

Certain sociodemographic factors have also been linked with the development and maintenance of chronic spine-related pain and dysfunction (Chibnall & Tait, 2009). Not surprisingly, the development of chronic pain is predicted by factors such as older age (Turner, 2006; Tate, 1992) or obesity (Hagen, Tambs, & Bjerkedal, 2002). Perhaps not as obvious are the contributions of factors like education, socioeconomic status, occupation, and medico-legal context (Chibnall & Tait, 2009; Rubin, 2007). However, these relationships are complex and the variables are interrelated, with some undoubtedly acting as correlates for underlying causal risk factors. Despite these uncertainties, these correlates can still serve as useful predictors of chronic pain-related dysfunction.

Education

Low education has been identified as a prognostic indicator of work-related disability (Breslin et al., 2008; Hagen, Holte, Tambs, & Bjerkedal, 2000). A review by Dionne et al. (2001) found that lower education is associated with longer pain duration following back injury and a higher rate of recurrence. Even after controlling for age, pain duration, sex, and incentive status, lower education was significantly associated with higher self-perceived disability (Roth & Geisser, 2002). This relationship may be explained by the finding that lower education is associated with more misconceptions about back pain (Goubert, Crombez, & De Boudeauhuij, 2004), as Roth and Geisser found that the relationship between education and disability was mediated almost entirely by maladaptive pain beliefs and coping strategies (e.g., catastrophizing).
**Occupation and Socioeconomic Status**

Physical work load and job satisfaction both are prognostic indicators of back pain-related work absences and disability (Bigos et al., 1991; Hagen et al., 2002; Hoogendoorn et al., 2002; Shaw et al., 2005). Lower wage compensation is associated with longer back pain chronicity (Volinn, Van Koevering, & Loeser, 1991) and unskilled workers are two to three times more likely to retire due to disability than professionals (Hagen et al., 2000). Hagen et al. concluded that this relationship may be partly due to a social class effect, rather than just physical job demands, as the relationship between professional level and disability retirement remained consistent at higher levels of levels of the socioeconomic scale.

**Compensation and Litigation**

It is not uncommon for chronic pain cases to be seen in the context of personal injury litigation, workers compensation, or disability determinations. Patients seen in a compensatory context report significantly more pain, depression, and disability than patients not involved in compensation (Chibnall & Tait, 1994; Rainville, Sobel, Hartigan, & Wright, 1997; Rohling, Binder, & Langhinrichsen-Rohling, 1995). In fact, a longitudinal study by Overland et al. (2008) found that reports of pain, anxiety, depression, sleep disturbances, and somatic symptoms increased steadily as a financial disability determination neared, only to steadily decrease after the determination was made. Further, compensation status is associated with overall decreased treatment efficacy (Gatchel, Polatin, & Mayer, 1995; Rainville et al., 1997; Rohling et al., 1995) including worse surgical outcomes (Harris, Mulford, Solomon, van Gelder, & Young, 2005), even for clearly defined spinal pathology (Atlas et al., 2000; Atlas et al., 2006). As an example of the systemic effects of compensatory context, recent changes to a “no fault” compensation system
in Canada were found to result in a lower incidence of lower-back pain and whiplash injuries following accidents and better prognosis for recovery (Cameron et al., 2008; Cassidy, Carroll, Cote, Berglund, & Nygren, 2003).

**Validity of Clinical Presentation**

Clinical presentation can be impacted by a number of factors that provide incentive or motivation for patients to exaggerate or minimize certain symptoms, psychological complications, or disabilities (Rogers, 2008). For patients with chronic pain, external motivations may include financial compensation (discussed above) or drug-seeking (Hansen, 2005; Longo, Parran, Johnson, & Kinsey, 2000), while internal motivations may include a need for attention or symptom validation (Blackwell & Gutmann, 1987).

Measures of pain, perceived disability, and psychopathology rely on self-report and are thus dependent on patient cooperation and honesty. In addition, most measures are relatively face-valid; meaning intentional or unintentional manipulation of results by the examinee is possible. Thus, it is important to consider the validity of a patient’s neuropsychological presentation when drawing conclusions about test results, particularly in cases that are medically unexplained (Binder, 2005). As stated in a recent position paper from the National Academy of Neuropsychology, “Adequate assessment of response validity is essential in order to maximize confidence in the results of neurocognitive and personality measures and in the diagnoses and recommendations that are based on the results” (Bush et al., 2005, p. 419).

Symptom validity is not an inconsequential factor for research on chronic pain, as recent findings suggest that a sizeable minority (25% to 45%) of chronic pain patients seen in a compensatory context over-report psychological complications to a degree that makes their
presentations invalid (Greve, Ord, Bianchini, & Curtis, 2009). Despite these concerns, a review of the literature found no studies that systematically controlled for presentation validity while examining the relationship between psychopathology and perceived disability in patients with chronic pain.

**Purpose**

This study sought to explore relationships between medical findings, psychological complications, sociodemographic factors, pain intensity, and self-perceived disability in patients with chronic spine-related pain. More specifically, this study addressed: (a) the prevalence of complications related to depression, anxiety, somatization, and pain catastrophizing; (b) the relationship between these psychopathologies and reported pain intensity and disability; and (c) whether these psychological factors predict pain and perceived disability beyond selected medical and sociodemographic factors. Importantly, the validity of patients’ neuropsychological presentations was evaluated and controlled for in order to provide a more accurate assessment of psychological functioning and perceived disability.

**Hypotheses**

*Prevalence of Psychopathology*

Significant elevations in the prevalence of depression, anxiety, somatization, and pain catastrophizing were expected relative to normative non-chronic pain samples. Given their strong relationship with the development of chronic pain, elements of depression, somatization, and pain catastrophizing were expected to be particularly common. Most patients were expected to exhibit multiple comorbid psychopathologies. Patients without medical findings were expected to show higher rates of psychological complications compared
to patients with documented medical findings. Higher rates of psychological complications were also expected to be observed in patients showing more evidence of symptom exaggeration.

**Pain Intensity and Perceived Disability**

For all measured psychological factors, higher scores were expected to be associated with higher reported pain and disability. Each measured psychological factors was expected to significantly predict reported pain intensity beyond demographics and spine-related medical findings. Psychological factors were expected to be even more strongly associated with reported disability. Further, each psychological construct was expected to predict reported disability beyond demographics, medical findings, and pain intensity. Psychopathology’s relationship with pain and disability was expected to be particularly strong in the absence of spine pathology.
METHODS

Participants

Data were collected retrospectively from the files of 346 consecutive patients with chronic pain seen for psychological evaluation at a clinic in southeast Louisiana. Cases were then excluded if (a) they did not present with a prominent spine-related injury or pain complaint, (b) they were not between the ages of 18 and 65, or (c) they did not complete all of the measures discussed below. 138 cases met all criteria and were included in the analyses.

A comprehensive review of medical records was performed to characterize injuries and pathological findings. The majority of cases involved musculoskeletal injury and 73.9% had spine-related findings including: degenerative discs or joints (38.4%), bulging or protruding discs (57.2%), herniated discs (5.8%), and/or neural impingement (5.8%). Surgical procedures involving discectomies or vertebral fusions were observed in 26.1% of the sample and decompression or laminectomies were observed in 13.0%.

All but one case was evaluated in the context of a workers’ compensation (80.4%) or personal injury (18.8%) claim and about half were represented by an attorney. Mean time between injury and evaluation was 43.3 months (SD = 33.4). Average verbal rating of current pain (0-10) was 6.4 (SD = 1.9). A summary of patient demographics and injury characteristics for the entire sample can be found in Table 1.
Table 1
Patient Demographics and Injury Characteristics

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Sample (N = 138)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age M (SD)</td>
<td>45.0 (9.2)</td>
</tr>
<tr>
<td>Education M (SD)</td>
<td>12.1 (2.2)</td>
</tr>
<tr>
<td>Sex</td>
<td></td>
</tr>
<tr>
<td>Male %</td>
<td>63.0</td>
</tr>
<tr>
<td>Female %</td>
<td>37.0</td>
</tr>
<tr>
<td>Race</td>
<td></td>
</tr>
<tr>
<td>White %</td>
<td>69.6</td>
</tr>
<tr>
<td>Black %</td>
<td>29.0</td>
</tr>
<tr>
<td>Other %</td>
<td>1.4</td>
</tr>
<tr>
<td>Medico-Legal Status</td>
<td></td>
</tr>
<tr>
<td>Incentive %</td>
<td>99.3</td>
</tr>
<tr>
<td>Attorney Represented %</td>
<td>50.0</td>
</tr>
<tr>
<td>Workers Compensation %</td>
<td>80.4</td>
</tr>
<tr>
<td>Personal Injury %</td>
<td>18.8</td>
</tr>
<tr>
<td>Months Since Injury M (SD)</td>
<td>43.3 (33.4)</td>
</tr>
<tr>
<td>Current Pain Rating (0-10) M (SD)</td>
<td>6.4 (1.9)</td>
</tr>
<tr>
<td>Spine Findings</td>
<td></td>
</tr>
<tr>
<td>Degenerative Disc %</td>
<td>38.4</td>
</tr>
<tr>
<td>Disc Bulge %</td>
<td>57.2</td>
</tr>
<tr>
<td>Disc Herniation %</td>
<td>5.8</td>
</tr>
<tr>
<td>Impingement %</td>
<td>5.8</td>
</tr>
<tr>
<td>Any Spine Findings</td>
<td>73.9</td>
</tr>
<tr>
<td>Spine Surgery</td>
<td></td>
</tr>
<tr>
<td>Discectomy / Fusion %</td>
<td>26.1</td>
</tr>
<tr>
<td>Decompression / Laminectomy %</td>
<td>13.0</td>
</tr>
</tbody>
</table>

Measures

Psychopathology

Minnesota Multiphasic Personality Inventory-2

The Minnesota Multiphasic Personality Inventory-2 (MMPI-2; Butcher, Dahlstrom, Graham, Tellegen, & Kaemmer, 1989) consists of 567 true/false questions designed to measure social and personal maladjustment. The MMPI-2 was used to measure the constructs of depression (scale 2 [D]), somatization (scales 1 [Hs] and 3 [Hy]), and anxiety (scale 7 [Pt]).
Uniform T-scores were used for the analysis and all normative comparisons were made to the sample reported by Butcher et al. Patients who showed inconsistent or random responding by scoring above 80 on either the Variable Response Inconsistency scale (VRIN) or the True Response Inconsistency scale (TRIN) were excluded from the study due to the inability to interpret MMPI-2 results (Butcher et al.).

**Pain Catastrophizing Scale**

The Pain Catastrophizing Scale (PCS; Sullivan, Bishop, & Pivik, 1995) was used to measure the construct of catastrophization which includes a hypervigilance, threat magnification, and feeling of helplessness related to pain. The PCS consists of 13 statements related to pain that are each rated (0-4) as to the degree felt during painful experiences. Final scores were converted to gender corrected T-scores using normative data from Sullivan et al. A copy of this measure is included in Appendix C.

**Pain-Related Disability**

Pain-related disability was measured using the Pain Disability Index (PDI; Pollard, 1984). The PDI is a short self-report questionnaire designed to measure the consequences of chronic pain on daily life (Chibnall & Tait, 1994; Tait, Pollard, Margolis, Duckro, & Krause, 1987). Patients are asked to rate (0-10) the overall impact of pain on their lives in seven domains of daily activities: family/home responsibilities, recreation, social activity, occupation, sexual behavior, self-care, and life-support activities. A raw score (0-70) is calculated by summing the reported disability from each domain. A copy of this questionnaire is presented in Appendix D.
Spine Severity

A spine severity scale was created to serve as a rough linear approximation of the degree or severity of spine-related medical findings. Based on a review of medical records each case was assigned a score of 0 to 4 as follows: no findings = 0; degenerative disc(s) or joint(s) = 1; bulging or protruding disc(s) = 2; herniated disc(s) = 3; and 4) neural impingement(s) = 4. Note that spine severity scores were not cumulative; patients received the highest single score for which findings were observed.

Exaggeration

To help control for symptom exaggeration a composite variable was created using a diverse set of well-validated indicators of performance and symptom validity. Indicators were chosen from three domains related to disability in patients with pain: psychological, cognitive, and functional findings. For the psychological and cognitive domains, each of three groups of indicators was assigned a score of 0, 1, or 2, with higher scores indicative of greater exaggeration. For the functional domain, each of four types of validity-related findings was scored as a 0 or 1. Scores were then added together to create a final composite score.

This process is similar to that used to develop the Meyers Index for the MMPI-2 (Meyers, Millis, & Volkert, 2002), but was expanded to include contributions from the cognitive and functional domains. In addition to standard validity indicators from the MMPI-2, exaggeration was measured using the Portland Digit Recognition Test (PDRT; Binder, 1993), Test of Memory Malingering (TOMM; Tombaugh, 1996), Reliable Digit Span (RDS; Greiffenstein, Baker, & Gola, 1994), rated effort on a Functional Capacity Exam (FCE), and inconsistencies between functional findings and self report. Table 2 presents the indicators and cutoffs used. A
more extensive explanation of the chosen validity indicators along with a rational for their use can be found in Greve et al. (2009).

Table 2

<table>
<thead>
<tr>
<th>Domain</th>
<th>Measure</th>
<th>Score&lt;sup&gt;a&lt;/sup&gt;</th>
<th>Cutoff(s)/Criteria</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cognitive</td>
<td>PDRT</td>
<td>1</td>
<td>Easy &lt; 25 or Hard &lt; 21 or Total &lt; 46</td>
</tr>
<tr>
<td></td>
<td></td>
<td>2</td>
<td>Easy &lt; 22 or Hard &lt; 16 or Total &lt; 40</td>
</tr>
<tr>
<td></td>
<td>TOMM</td>
<td>1</td>
<td>Trial 2 or Retention &lt; 49</td>
</tr>
<tr>
<td></td>
<td></td>
<td>2</td>
<td>Trial 2 or Retention &lt; 45</td>
</tr>
<tr>
<td></td>
<td>WAIS-III</td>
<td>1</td>
<td>Reliable Digit Span &lt; 7</td>
</tr>
<tr>
<td></td>
<td></td>
<td>2</td>
<td>Reliable Digit Span &lt; 6</td>
</tr>
<tr>
<td>Psychological</td>
<td>MMPI-2</td>
<td>1</td>
<td>Infrequency or Infrequency-back &gt; 80</td>
</tr>
<tr>
<td></td>
<td></td>
<td>2</td>
<td>Infrequency or Infrequency-back &gt; 90</td>
</tr>
<tr>
<td></td>
<td></td>
<td>1</td>
<td>Infrequency-psychopathology &gt; 80</td>
</tr>
<tr>
<td></td>
<td></td>
<td>2</td>
<td>Infrequency-psychopathology &gt; 90</td>
</tr>
<tr>
<td></td>
<td></td>
<td>1</td>
<td>Symptom Validity Scale (FBS raw) &gt; 28</td>
</tr>
<tr>
<td></td>
<td></td>
<td>2</td>
<td>Symptom Validity Scale (FBS raw) &gt; 32</td>
</tr>
<tr>
<td>Functional</td>
<td></td>
<td>1</td>
<td>Poor effort on a Functional Capacity Exam</td>
</tr>
<tr>
<td></td>
<td></td>
<td>1</td>
<td>Inconsistent Functional Findings</td>
</tr>
<tr>
<td></td>
<td></td>
<td>1</td>
<td>Inconsistent findings during physical exam</td>
</tr>
<tr>
<td></td>
<td></td>
<td>1</td>
<td>Inconsistencies in self report</td>
</tr>
</tbody>
</table>

Note. MMPI-2 = Minnesota Multiphasic Personality Inventory-2; PDRT = Portland Digit Recognition Test; TOMM = Test of Memory Malingering; WAIS-III = Wechsler Adult Intelligence Scale-III.
<sup>a</sup> Each indicator can meet criteria for a score of 1 or 2, not both.

For validation of the exaggeration composite, all cases were also classified as Not Malingering (N = 27), Possible Malingering (N = 61), and Malingering (N = 50) according to criteria for Malingered Pain-Related Disability (MPRD) from Bianchini, Greve, & Glynn (2005) using methods, indicators, and cutoffs described in Greve et al. (2009).

The symptom exaggeration composite was used as a covariate in some analyses and to categorically classify patients in others. When feasible, cases were classified into no exaggeration (scores < 2; N = 59), some exaggeration (scores of 2 or 3; N = 52), and high
exaggeration (scores > 3; N = 27) groups. However, for certain analyses, the “no” and “some” exaggeration groups were combined into a “low” exaggeration group to avoid over truncating this group and potentially removing meaningful score variance.
RESULTS

Preliminary Analyses

All variables were examined to insure that there were no major threats to statistical analysis. Variables were distributed normally, with the exception of the exaggeration composite (skew = 1.31; kurtosis = 1.85) and PDI (skew = -1.40; kurtosis = 2.45). The skew in the exaggeration composite was expected given the nature of the variable and was not anticipated to threaten the analyses. The skew in PDI scores was of more concern considering its role as the primary outcome variable. A decision was made to transform PDI scores using a square root transformation and the resulting PDI variable was normally distributed (skew = -.41; kurtosis = .54). The transformed PDI variable was used for all correlation- and regression-based analyses.

The exaggeration composite variable was further examined to insure that it was functioning as intended. The mean symptom exaggeration score was 2.23 (SD = 2.24) and mean scores by domain were .90 (SD = 1.15) for psychological indicators, .88 (SD = 1.40) for cognitive indicators, and 0.45 (SD = .67) for functional indicators. Exaggeration scores were compared according to MPRD classification status and significant differences ($F[2] = 53.852; p < .01$) across the Not Malingering ($m = 0; SD = 0$), Possible Malingering ($m = 1.77; SD = 1.15$), and Malingering ($m = 4.00; SD = 2.48$) groups were found. These findings suggest that the exaggeration composite is effectively separating valid from invalid clinic presentations. As expected, higher exaggeration was associated with higher scores on psychological measures ($p < .01$), pain rating ($p = .02$), and PDI ($p < .01$).

Mean scores for all examined variables are presented in Table 3 for the entire sample and broken down by presence or absence of spine findings and degree of exaggeration. In the
entire sample, all psychological scales (PCS, Hs, D, Hy, and Pt) showed significant mean T-score elevations above their respective normative samples ($p < .01$). Patients without spine findings had slightly higher mean scores on all measures compared to patients with spine findings, though only the difference in Pt was significant ($p = .05$). Patients displaying higher levels of exaggeration had significantly higher mean scores on all measures ($p < .01$), with the exception of pain rating which approached significance ($p = .07$).

Table 3

<table>
<thead>
<tr>
<th>Measure</th>
<th>Sample</th>
<th>Spine Findings</th>
<th>Exaggeration</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>N</td>
<td>No</td>
<td>Yes</td>
</tr>
<tr>
<td>PCS</td>
<td>M</td>
<td>73.6</td>
<td>74.4</td>
</tr>
<tr>
<td></td>
<td>SD</td>
<td>15.0</td>
<td>13.2</td>
</tr>
<tr>
<td>Hs</td>
<td>M</td>
<td>81.8</td>
<td>82.6</td>
</tr>
<tr>
<td></td>
<td>SD</td>
<td>9.4</td>
<td>10.1</td>
</tr>
<tr>
<td>D</td>
<td>M</td>
<td>81.7</td>
<td>83.5</td>
</tr>
<tr>
<td></td>
<td>SD</td>
<td>13.1</td>
<td>10.8</td>
</tr>
<tr>
<td>Hy</td>
<td>M</td>
<td>82.9</td>
<td>84.5</td>
</tr>
<tr>
<td></td>
<td>SD</td>
<td>15.4</td>
<td>15.8</td>
</tr>
<tr>
<td>Pt</td>
<td>M</td>
<td>72.8</td>
<td>77.0</td>
</tr>
<tr>
<td></td>
<td>SD</td>
<td>15.1</td>
<td>12.9</td>
</tr>
<tr>
<td>Exaggeration</td>
<td>M</td>
<td>2.2</td>
<td>2.7</td>
</tr>
<tr>
<td></td>
<td>SD</td>
<td>2.2</td>
<td>2.4</td>
</tr>
<tr>
<td>Pain Rating</td>
<td>M</td>
<td>6.4</td>
<td>6.8</td>
</tr>
<tr>
<td></td>
<td>SD</td>
<td>1.9</td>
<td>2.1</td>
</tr>
<tr>
<td>PDI</td>
<td>M</td>
<td>51.6</td>
<td>52.2</td>
</tr>
<tr>
<td></td>
<td>SD</td>
<td>12.1</td>
<td>11.6</td>
</tr>
</tbody>
</table>

Note. D = Depression (Scale 2); Hs = Hypochondriasis (Scale 1); Hy = Hysteria (Scale 3); PCS = Pain Catastrophizing Scale; PDI = Pain Disability Index; Pt = Psychasthenia (Scale 7).
Relationships among demographics, psychological variables, and symptom exaggeration were examined using Pearson correlations in the entire sample. Older age was found to be associated with less exaggeration ($r = -.213; p = .01$). Females were associated with higher education ($r = .279; p < .01$) and education was negatively correlated with PCS scores ($r = -.223; p < .01$). PCS was most strongly related to D ($r = .435; p < .01$) and Pt ($r = .380; p < .01$). High correlations were found among MMPI-2 variables, particularly Hs and Hy ($r = 0.719; p < .01$) and D and Pt ($r = 0.716; p < .01$). All psychological measures were positively correlated with the exaggeration composite score ($p < .01$). Table 4 presents full results of the correlational analysis. Note that correlations involving pain rating and PDI are discussed in their respective sections.

Table 4

<table>
<thead>
<tr>
<th></th>
<th>Age</th>
<th>Ed</th>
<th>Gender$^a$</th>
<th>PCS</th>
<th>Hs</th>
<th>D</th>
<th>Hy</th>
<th>Pt</th>
<th>Exag</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>-</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ed</td>
<td>.032</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Gender</td>
<td>.166</td>
<td>.279**</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PCS</td>
<td>-.153</td>
<td>-.229**</td>
<td>-.164</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hs</td>
<td>.092</td>
<td>-.008</td>
<td>-.094</td>
<td>.214*</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>D</td>
<td>-.070</td>
<td>-.134</td>
<td>-.049</td>
<td>.435**</td>
<td>.498**</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hy</td>
<td>.127</td>
<td>.009</td>
<td>-.056</td>
<td>.117</td>
<td>.735**</td>
<td>.550**</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pt</td>
<td>-.064</td>
<td>-.137</td>
<td>-.156</td>
<td>.380**</td>
<td>.509**</td>
<td>.756**</td>
<td>.402**</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Exag</td>
<td>-.213*</td>
<td>.056</td>
<td>.001</td>
<td>.322**</td>
<td>.429**</td>
<td>.483**</td>
<td>.378**</td>
<td>.480**</td>
<td></td>
</tr>
</tbody>
</table>

Note. D = Depression (Scale 2); Ed = Education; Exag = Exaggeration composite; Hs = Hypochondriasis (Scale 1); Hy = Hysteria (Scale 3); PCS = Pain Catastrophizing Scale; PDI = Pain Disability Index; Pt = Psychasthenia (Scale 7).

$^a$ Females are coded in the positive direction.

* Correlation is significant at the 0.05 level (2-tailed).

** Correlation is significant at the 0.01 level (2-tailed).
Prevalence of Psychopathology

*Individual Scale Elevations*

The prevalence of elevations on PCS, Hs, D, Hy, and Pt (defined for these purposes as T-scores greater than 70) were examined to compare the relative rates of psychological complications. Almost all of the patients (97.9%) had at least one elevated score, and each scale was elevated by at least half of the sample. Table 5 presents a summary of observed prevalence rates as well as the expected rates based on normative samples.

Table 5
*Frequencies of Elevations on Psychological Measures*

<table>
<thead>
<tr>
<th>Measure</th>
<th>Sample</th>
<th>Spine Findings</th>
<th>Exaggeration</th>
<th>Normative Expectation</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>No</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td>N 138</td>
<td>65.9</td>
<td>69.4</td>
<td>64.7</td>
<td>59</td>
</tr>
<tr>
<td>PCS</td>
<td>65.9</td>
<td>69.4</td>
<td>64.7</td>
<td>59</td>
</tr>
<tr>
<td>Hs 87.0</td>
<td>83.3</td>
<td>88.2</td>
<td>79.4</td>
<td>79.7</td>
</tr>
<tr>
<td>D 81.2</td>
<td>86.1</td>
<td>79.4</td>
<td>61.0</td>
<td>94.2</td>
</tr>
<tr>
<td>Hy 81.2</td>
<td>86.1</td>
<td>79.4</td>
<td>35.7</td>
<td>88.5</td>
</tr>
<tr>
<td>Pt 55.8</td>
<td>72.2</td>
<td>50.0</td>
<td>27.1</td>
<td>71.2</td>
</tr>
</tbody>
</table>

*Note.* Scores were considered elevated if they were more than 2 standard deviations above normative means (T-scores > 70); D = Depression (Scale 2); Hs = Hypochondriasis (Scale 1); Hy = Hysteria (Scale 3); PCS = Pain Catastrophizing Scale; PDI = Pain Disability Index; Pt = Psychasthenia (Scale 7).

^a Statistical approximation based on normative data from Sullivan et al. (1995).

^b Based on Uniform T-score conversions of normative data from Butcher et al. (1989).

Chi-square and Monte Carlo goodness-of-fit procedures indicated prevalence rates were significantly higher than normative samples on all measures, even in the no exaggeration group \((p < .01)\). Patients without spine findings tended to have higher rates of elevations, though the rates were only significantly higher on Pt \((\chi^2[1] = 5.328; p = .03)\); Hs showed the opposite trend, but the difference in scores was not significant \((\chi^2[1] = .564; p = .31)\). Patients displaying higher levels of exaggeration had significantly higher rates of elevations on PCS \((\chi^2[2] = 19.005; p < .01)\).
.01), D ($\chi^2[2] = 27.733; p < .01$), Hy ($\chi^2[2] = 12.749; p < .01$), and Pt ($\chi^2[2] = 36.634; p < .01$), and marginally higher rates of elevations on Hs ($\chi^2[2] = 5.384; p = .07$).

**Multiple Scale Elevations**

Comorbidity among psychological complications was also examined. Since exaggeration was found to be a potential confound, only patients in the low exaggeration group were included in this analysis. Psychological complications were rarely observed in isolation, with 89.2% of cases elevating more than one scale and over half elevating at least four of the five examined scales. Patients elevating two scales were most likely to elevate Hs (70.0%) and Hy (75.0%); patients elevating three scales were most likely to elevate Hs (93.3%), D (86.7%), and Hy (73.3%); and patients elevating four scales were most likely to elevate Hs (92.9%), D (100%), Hy (85.7%), and PCS (67.9%).

Table 6 presents characteristics of patients in the low exaggeration group broken down by number of observed scale elevations. No significant age, education, or spine severity differences were observed across these groups. Chi-square analysis did reveal significant gender differences ($\chi^2[5] = 13.528; p = .02$), with females more likely to show two elevations and males more likely to show four elevations. Significant differences were also observed across PDI scores ($F[5] = 2.813; p = .02$), with more psychological complications being associated with more reported disability. Differences across pain rating scores approached marginal significance ($F[5] = 1.915; p = .10$); however, no clear trend was apparent as the lowest pain ratings were reported by patients with three scale elevations. Figure 1 presents a grouped summary of pain disability ratings by number of scale elevations.
Table 6
Characteristics of Patients in the Low Exaggeration Group by Number of Comorbid Psychological Complications

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Number of Scale Elevations</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>0</td>
</tr>
<tr>
<td>Sample (N = 111)</td>
<td>%a</td>
</tr>
<tr>
<td>Males</td>
<td></td>
</tr>
<tr>
<td>Females</td>
<td></td>
</tr>
<tr>
<td>Spine Findings</td>
<td></td>
</tr>
<tr>
<td>No Spine Findings</td>
<td></td>
</tr>
<tr>
<td>Spine Severity</td>
<td>M</td>
</tr>
<tr>
<td></td>
<td>SD</td>
</tr>
<tr>
<td>Pain Rating</td>
<td>M</td>
</tr>
<tr>
<td></td>
<td>SD</td>
</tr>
<tr>
<td>Pain Disability Index</td>
<td>M</td>
</tr>
<tr>
<td></td>
<td>SD</td>
</tr>
</tbody>
</table>

*a* All percentages are presented within row.

Figure 1. Mean Pain Disability Index scores by number of elevations on psychological measures.
Pain Intensity

*Relationships with Examined Variables*

Pearson correlations were calculated to examine how self-rated pain intensity relates to demographics (age, education, and gender), medical findings (spine severity), psychological factors (PCS-T, Hs, D, Hy, and Pt), and symptom exaggeration. In the entire sample, pain ratings were significantly correlated with gender ($r = .281; p < .01$) and symptom exaggeration ($r = .194; p = .02$). Of the psychological measures, only D ($r = .162; p = .06$) approached a significant relationship with pain ratings. Full results are presented in Table 7.

Table 7

<table>
<thead>
<tr>
<th></th>
<th>Entire Sample</th>
<th>Spine Findings</th>
<th>Exaggeration</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>N</td>
<td>No</td>
<td>Yes</td>
</tr>
<tr>
<td><strong>Age</strong></td>
<td>r .066</td>
<td>.064</td>
<td>.081</td>
</tr>
<tr>
<td><strong>Education</strong></td>
<td>r .032</td>
<td>.097</td>
<td>.024</td>
</tr>
<tr>
<td><strong>Gender</strong></td>
<td>r .281**</td>
<td>.358*</td>
<td>.256**</td>
</tr>
<tr>
<td><strong>Spine Severity</strong></td>
<td>r -.106</td>
<td>--</td>
<td>-.007</td>
</tr>
<tr>
<td><strong>Exaggeration</strong></td>
<td>r .194*</td>
<td>.135</td>
<td>.200*</td>
</tr>
<tr>
<td><strong>PCS</strong></td>
<td>r .073</td>
<td>.415*</td>
<td>-.044</td>
</tr>
<tr>
<td><strong>Hs</strong></td>
<td>r .035</td>
<td>-.159</td>
<td>.109</td>
</tr>
<tr>
<td><strong>D</strong></td>
<td>r .162</td>
<td>.024</td>
<td>.195*</td>
</tr>
<tr>
<td><strong>Hy</strong></td>
<td>r -.016</td>
<td>-.102</td>
<td>.008</td>
</tr>
<tr>
<td><strong>Pt</strong></td>
<td>r .147</td>
<td>.019</td>
<td>.165</td>
</tr>
</tbody>
</table>

Note. D = Depression (Scale 2); Hs = Hypochondriasis (Scale 1); Hy = Hysteria (Scale 3); PCS = Pain Catastrophizing Scale; Pt = Psychasthenia (Scale 7).
* Correlation is significant at the 0.05 level (2-tailed).
** Correlation is significant at the 0.01 level (2-tailed).

When correlations were examined separately in patients with and without spine findings, some differences in relationships were observed. Pain ratings in patients without spine findings showed a stronger relationship with PCS ($r = .415$) than in patients with spine findings.
(r = -.044), and the difference between these correlations was significant when analyzed using a Fischer r-to-z transformation (z = 2.42; p = .02). Conversely, pain ratings in patients with spine findings showed a stronger relationship with depression (r = .195) than was observed in patients without spine findings (r = .024); however, the difference between these correlations was not significant (z = .86; p = .39).

Differences in correlations were also observed when comparing patients in the low and high exaggeration groups. For example, pain rating and education were significantly correlated in the high exaggeration group (r = .421), but not in the low exaggeration group (r = -.046); and this difference in correlations was significant (z = 2.19; p = .03). Similarly, all of the examined psychological variables were more strongly correlated to pain rating in the high exaggeration group (|r| = .206 to .385) compared to the low exaggeration group (|r| = .042 to .162), however, only the difference on Hy (z = 2.24; p = .03) was significant. Although not significant, it is interesting to note that higher pain ratings were associated with lower severity of spine findings in the high exaggeration group (r = -.211; p = .29).

Prediction of Pain Intensity

A series of stepwise regressions was performed to examine the contributions of the selected psychological variables to predicting pain ratings beyond demographics, spine findings, and exaggeration. First, using the entire sample, demographics, spine severity, and exaggeration were entered in the first step; this step significantly predicted pain ratings (r = .356; F[5] = 3.844; p < .01), with gender (b = .282; t = 3.274; p < .01) and exaggeration (b = .202; t = 2.396; p = .02) being the only significant predictors. Each of the psychological variables was
then individually entered in the second step; none were found to significantly add to the prediction of pain ratings.

Another series of regressions was performed using only cases in the low exaggeration group and excluding the exaggeration composite variable. Demographics and spine severity were entered in the first step which significantly predicted pain ratings ($r = .298; F[4] = 2.589; p = .04$), with gender ($b = .289; t = 2.937; p < .01$) being the only significant predictor. When each psychological variable was entered individually in the second step only Pt significantly added to prediction of pain ratings ($r^2 \text{ change} = .046; F \text{ change} = 5.542; p = .02$). Full results of the second step changes for both series of regressions are present in Table 8.

**Table 8**

*Second Step Change Statistics for Psychological Measures Individually Added to the Second Step of a Regression Predicting Pain Rating Scores*

<table>
<thead>
<tr>
<th>Included</th>
<th>Step 1</th>
<th>Measure</th>
<th>$r^2 \text{ change}$</th>
<th>$F \text{ change}$</th>
<th>$p$</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Entire Sample</strong></td>
<td>Age, Education, Gender, Spine Findings, and Exaggeration</td>
<td>PCS</td>
<td>.003</td>
<td>.483</td>
<td>.488</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Hs</td>
<td>.001</td>
<td>.184</td>
<td>.669</td>
</tr>
<tr>
<td></td>
<td></td>
<td>D</td>
<td>.008</td>
<td>1.156</td>
<td>.284</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Hy</td>
<td>.009</td>
<td>1.315</td>
<td>.254</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Pt</td>
<td>.010</td>
<td>1.496</td>
<td>.224</td>
</tr>
<tr>
<td><strong>Low Exaggerators</strong></td>
<td>Age, Education, Gender, and Spine Findings</td>
<td>PCS</td>
<td>.015</td>
<td>1.738</td>
<td>.190</td>
</tr>
<tr>
<td>(N = 111)</td>
<td></td>
<td>Hs</td>
<td>.000</td>
<td>.010</td>
<td>.919</td>
</tr>
<tr>
<td></td>
<td></td>
<td>D</td>
<td>.024</td>
<td>2.786</td>
<td>.098</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Hy</td>
<td>.009</td>
<td>1.011</td>
<td>.317</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Pt</td>
<td>.046</td>
<td>5.542</td>
<td>.020</td>
</tr>
</tbody>
</table>

*Note.* D = Depression (Scale 2); Hs = Hypochondriasis (Scale 1); Hy = Hysteria (Scale 3); PCS = Pain Catastrophizing Scale; Pt = Psychasthenia (Scale 7).
Perceived Disability

Relationships with Examined Variables

Pearson correlations were calculated to examine how PDI scores relate to demographics, spine findings, psychological factors, symptom exaggeration, and pain rating. In the entire sample, PDI scores were significantly correlated with symptom exaggeration ($r = .350; p < .01$), pain rating ($r = .202; p = .02$), Hs ($r = .350; p < .01$), D ($r = .350; p < .01$), Hy ($r = .385; p < .01$), and Pt ($r = .354; p < .01$). Full results are presented in Table 9.

Table 9
Pain Disability Index Correlations with Demographics, Exaggeration, Pain Rating and Psychological Measures

<table>
<thead>
<tr>
<th></th>
<th>Entire Sample</th>
<th>Spine Findings</th>
<th>Exaggeration</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>No</td>
<td>Yes</td>
</tr>
<tr>
<td>N</td>
<td>138</td>
<td>36</td>
<td>102</td>
</tr>
<tr>
<td>Age</td>
<td>r .100</td>
<td>-.043</td>
<td>.151</td>
</tr>
<tr>
<td>Education</td>
<td>r -.100</td>
<td>.233</td>
<td>-.214*</td>
</tr>
<tr>
<td>Gender</td>
<td>r -.094</td>
<td>-.167</td>
<td>-.070</td>
</tr>
<tr>
<td>Spine Severity</td>
<td>r -.028</td>
<td>--</td>
<td>-.019</td>
</tr>
<tr>
<td>Exag</td>
<td>r .350**</td>
<td>.493**</td>
<td>.300**</td>
</tr>
<tr>
<td>Pain Rating</td>
<td>r .202*</td>
<td>-.019</td>
<td>.282**</td>
</tr>
<tr>
<td>PCS</td>
<td>r .217*</td>
<td>.385*</td>
<td>.171</td>
</tr>
<tr>
<td>Hs</td>
<td>r .350**</td>
<td>.566**</td>
<td>.273**</td>
</tr>
<tr>
<td>D</td>
<td>r .350**</td>
<td>.518**</td>
<td>.309**</td>
</tr>
<tr>
<td>Hy</td>
<td>r .385**</td>
<td>.563**</td>
<td>.326**</td>
</tr>
<tr>
<td>Pt</td>
<td>r .354**</td>
<td>.366*</td>
<td>.353**</td>
</tr>
</tbody>
</table>

Note. D = Depression (Scale 2); Hs = Hypochondriasis (Scale 1); Hy = Hysteria (Scale 3); PCS = Pain Catastrophizing Scale; Pt = Psychasthenia (Scale 7).
* Correlation is significant at the 0.05 level (2-tailed).
** Correlation is significant at the 0.01 level (2-tailed).

When correlations were examined separately in patients with and without spine findings, some differences were again observed. In patients without spine findings there was a positive correlation between PDI and education ($r = .233$), while the opposite was observed in
patients with spine findings ($r = -.214$); a difference that was significant ($z = 2.26; p = .02$). Pain rating was significantly correlated with PDI in patients with spine findings ($r = .282$), but not in patients without spine findings ($r = -.019$), though this difference was not significant ($z = 1.54; p = .12$). In addition, PDI scores were more strongly correlated to psychological variables in patients without spine findings ($r = .366$ to $.566$) than in those with spine findings ($r = .171$ to $.353$); however, only the difference on Hs approached significance ($z = 1.80; p = .07$).

Though none were significant, some differences in correlations were observed between low exaggerators and high exaggerators. For example, PDI had little correlation with spine severity in the low exaggeration group ($r = .057$), but tended towards a negative correlation in the high exaggeration group ($r = -.240$). PDI showed a stronger correlation to PCS in the low exaggeration group ($r = .233$) compared to the high exaggeration group ($r = -.099$). Conversely, PDI was more strongly correlated with all of the MMPI-2 scales in the high exaggeration group ($r = .441$ to $.559$) compared to the low exaggeration group ($r = .230$ to $.320$).

**Prediction of the Pain Disability Index**

A series of stepwise regressions was performed to examine the contribution of the selected psychological variables to predicting PDI scores beyond demographics, spine findings, exaggeration, and pain rating. First, each of the psychological variables was individually entered in the second step of a regression in four different conditions:

1) Using the entire sample, age, education, gender, spine severity, and exaggeration were entered in the first step; this step predicted a significant portion of the variance in PDI ($r = .424; F[5] = 5.784; p < .01$), with age ($b = .205; t = 2.497; p = .01$) and exaggeration ($b = .401; t = 4.910; p < .01$) being the only significant predictors.
2) The above condition was repeated with the addition of pain rating in the first step; this step predicted a significant portion of the variance in PDI ($r = .451; F[6] = 5.571; p < .01$), with age ($b = .194; t = 2.383; p = .02$), exaggeration ($b = .368; t = 4.457; p < .01$), and pain rating ($b = .164; t = 1.969; p = .05$) being the only significant predictors.

3) Using only low exaggerators, age, education, gender, and spine severity were entered in the first step; this step did not predict a significant portion of the variance in PDI ($r = .249; F[4] = 1.755; p = .14$), with education ($b = -.165; t = 1.676; p = .10$) being the only predictor approaching marginal significance.

4) The above condition was repeated with the addition of pain rating in the first step; this step predicted a significant portion of the variance in PDI ($r = .318; F[5] = 2.364; p = .05$), with pain rating ($b = .207; t = 2.137; p = .04$) being the only significant predictor.

Results indicated that Hs, D, and Hy significantly (or marginally) increased model prediction of PDI in all conditions. Pt added significantly to prediction in conditions that did not include pain ratings in the first step. PCS added significantly to prediction in conditions that only included low exaggerators. Full results of the second step changes for each psychological variable in each condition are presented in Table 10.
Table 10

Second Step Change Statistics for Psychological Measures Individually Added to the Second Step of a Regression Predicting Pain Disability Index Scores in Four Different Conditions

<table>
<thead>
<tr>
<th>Included</th>
<th>Measure</th>
<th>( r^2 ) change</th>
<th>F change</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Entire Sample (N = 138)</td>
<td>PCS</td>
<td>0.008</td>
<td>1.256</td>
<td>.265</td>
</tr>
<tr>
<td></td>
<td>Hs</td>
<td>0.029</td>
<td>4.793</td>
<td>.030</td>
</tr>
<tr>
<td></td>
<td>D</td>
<td>0.032</td>
<td>5.259</td>
<td>.023</td>
</tr>
<tr>
<td></td>
<td>Hy</td>
<td>0.051</td>
<td>8.753</td>
<td>.004</td>
</tr>
<tr>
<td></td>
<td>Pt</td>
<td>0.030</td>
<td>4.932</td>
<td>.028</td>
</tr>
<tr>
<td>Low Exaggerators (N = 111)</td>
<td>PCS</td>
<td>0.006</td>
<td>1.028</td>
<td>.312</td>
</tr>
<tr>
<td></td>
<td>Hs</td>
<td>0.031</td>
<td>5.260</td>
<td>.023</td>
</tr>
<tr>
<td></td>
<td>D</td>
<td>0.027</td>
<td>4.559</td>
<td>.035</td>
</tr>
<tr>
<td></td>
<td>Hy</td>
<td>0.059</td>
<td>10.423</td>
<td>.002</td>
</tr>
<tr>
<td></td>
<td>Pt</td>
<td>0.025</td>
<td>4.156</td>
<td>.044</td>
</tr>
<tr>
<td></td>
<td>PCS</td>
<td>0.042</td>
<td>4.946</td>
<td>.028</td>
</tr>
<tr>
<td></td>
<td>Hs</td>
<td>0.032</td>
<td>3.712</td>
<td>.057</td>
</tr>
<tr>
<td></td>
<td>D</td>
<td>0.050</td>
<td>5.851</td>
<td>.017</td>
</tr>
<tr>
<td></td>
<td>Hy</td>
<td>0.077</td>
<td>9.363</td>
<td>.003</td>
</tr>
<tr>
<td></td>
<td>Pt</td>
<td>0.037</td>
<td>4.282</td>
<td>.041</td>
</tr>
<tr>
<td></td>
<td>PCS</td>
<td>0.033</td>
<td>3.964</td>
<td>.049</td>
</tr>
<tr>
<td></td>
<td>Hs</td>
<td>0.033</td>
<td>3.931</td>
<td>.050</td>
</tr>
<tr>
<td></td>
<td>D</td>
<td>0.037</td>
<td>4.507</td>
<td>.036</td>
</tr>
<tr>
<td></td>
<td>Hy</td>
<td>0.089</td>
<td>11.388</td>
<td>.001</td>
</tr>
<tr>
<td></td>
<td>Pt</td>
<td>0.023</td>
<td>2.717</td>
<td>.102</td>
</tr>
</tbody>
</table>

Note. D = Depression (Scale 2); Hs = Hypochondriasis (Scale 1); Hy = Hysteria (Scale 3); PCS = Pain Catastrophizing Scale; Pt = Psychasthenia (Scale 7).

Next, a stepwise regression incorporating all available predictors was performed to assess (a) whether exaggeration predicts PDI beyond demographics, spine findings, and pain rating; and (b) the joint ability of the selected psychological factors to predict PDI beyond all other examined factors. Demographics, spine severity, and pain ratings were entered in the first step which significantly predicted PDI scores \( r = .287; F[5] = 2.373; p = .042 \), with pain rating \( b \)
=.240; \(t = 2.751; p < .01\) being the only significant predictor. Exaggeration was entered in the second step which significantly increased prediction of PDI \(\left( r^2 \text{ change } = .121; F \text{ change } = 19.864; p < .01 \right)\). PCS, Hs, D, Hy, and Pt were entered in the final step which also significantly increased prediction \(\left( r^2 \text{ change } = .072; F \text{ change } = 2.502; p = .03 \right)\). In the final model, pain rating \((b = .177; t = 2.137; p = .04)\) and Hy \((b = .272; t = 2.162; p = .03)\) remained significant predictors, while exaggeration was marginally significant \((b = .191; t = 1.935; p = .06)\).

Finally, a similar stepwise regression was performed using only cases in the low exaggeration group and excluding the exaggeration composite variable. Demographics, spine severity, and pain rating were entered in the first step which significantly predicted PDI scores \((r = .318; F[5] = 2.364; p = .05)\), with pain rating \((b = .207; t = 2.137; p = .04)\) being the only significant predictor. PCS, Hs, D, Hy, and Pt were entered in the second step which produced a significant increase in prediction \(\left( r^2 \text{ change } = .118; F \text{ change } = 3.029; p = .01 \right)\). In the final model, only pain rating \((b = .224; t = 2.308; p = .02)\) and Hy \((b = .388; t = 2.653; p < .01)\) remained significant predictors, although PCS approached significance \((b = .186; t = 1.823; p = .07)\).
DISCUSSION

Understanding how psychosocial factors contribute to functional disability in pain-related conditions is important for informing treatment and rehabilitation decisions. The primary purpose of this study was to assess the prevalence of psychological complications in a chronic pain sample and examine whether these psychological factors contribute to spine-related pain and disability beyond sociodemographics and medical findings. This study is believed to be the first to assess the relative contributions of biological, psychological, and social factors to perceived disability in patients with chronic spine-related pain while systematically controlling for the validity of clinical presentation.

While this study did not directly assess the presence or absence of diagnosable disorders, rates of clinically relevant scale elevations were examined. Given the literature on a relationship between psychopathology and chronic pain, it was hypothesized that the present sample would show elevated rates of psychological complications. As expected, patients showed elevated rates of complications on all of the examined psychological constructs, even after controlling for exaggeration. Rates of somatization were most prevalent, followed by depression, catastrophizing, and then anxiety. Comorbidity among psychological complications was common and most patients presented with multiple scale elevations.

Psychological complications were hypothesized to be associated with higher reported pain. However, findings did not support this prediction, as none of the examined psychological measures was significantly related to pain intensity. Psychological complications were also expected to predict pain intensity beyond demographics and spine-related medical findings. This prediction was not supported for catastrophizing, somatization, or depression. However,
findings did indicate that anxiety provided a small but significant increase in the prediction of pain rating beyond demographics and medical findings in patients with low levels of exaggeration, accounting for an additional 5% of the variance in pain ratings.

It was also hypothesized that psychological complications would be associated with higher reported pain-related disability, and results generally supported this expectation. When exaggeration was controlled for statistically, somatization, depression, and anxiety significantly added to prediction of reported disability beyond demographics, spine-related medical findings, and reported pain intensity, explaining an addition 3% to 6% of disability score variance. Catastrophizing did not add to prediction of reported disability. When exaggeration was controlled for by excluding high exaggerators, all of the psychological measures significantly added to prediction of disability beyond demographics and spine-related medical findings. In addition, somatization, disability, and catastrophizing predicted reported disability beyond demographics, spine-related medical findings, and pain intensity, explaining an additional 3% to 9% of disability variance.

Prevalence of Psychological Complications

Overall, rates of psychological complications observed in the entire sample were comparable to results from other studies examining patients seeking treatment for chronic pain, such as those reported by Dersh et al. (2006) and Mayer et al. (2008). While rates of scale elevations in the present sample were slightly higher than rates of diagnosed psychopathology reported by those studies, this disparity is likely due to methodological differences, as Dersh et al. and Mayer et al. used more stringent criteria for diagnosis.
Psychological complications were found to be less prevalent in patients demonstrating lower levels of symptom exaggeration. However, even in patients showing no indications of exaggeration, rates of psychological complications were considerably elevated compared to normal non-pain samples. Magnitudes of scale elevations in non-exaggerating patients were consistent with findings from a study by Porter-Moffitt et al. (2006) that examined a sample of mostly non-incentive chronic pain patients.

A consistent trend has emerged in the literature indicating that rates of psychological complications are considerably higher in patients actively seeking treatment for chronic pain compared to persons with chronic pain drawn from the general population. For example, Demyttenaere et al. (2007) reported a psychological disorder prevalence rate of 16% in persons with chronic pain drawn from the general population; while Dersh et al. (2006), using the same diagnostic criteria, found a prevalence rate of 65% in patients seeking treatment for occupational spine disorders. This difference, while consistent, has been difficult to interpret given the social- and incentive-related influences that are inherent in psychological assessments of compensable injuries, which make up large portions of most clinic-based samples.

Results from this study confirm this trend and, importantly, suggest that exaggeration alone cannot account for the wide gap between clinic-based observations and population-based observations. For example, even in patients showing no indications of exaggeration, rates of catastrophizing were much higher in this sample compared to rates reported by Buer and Linton (2002) for a large sample of persons with moderate chronic pain drawn from the general population. Similarly, observed rates of depression in non-exaggerating patients from the
present sample were much higher than rates reported by Currie and Wang (2004) for a population-based sample of persons with chronic pain, even those reporting severe pain.

A number of factors may be contributing to this discrepancy between clinic-based and population-based estimates. For example, despite attempts by researchers to examine comparable samples, there may be subtle demographic, social, or injury-related differences between those seeking treatment for chronic pain and those with chronic pain in the general population. Contextual factors may also play a role, as persons suffering a work- or compensation-related injury can encounter a number of additional difficulties that may contribute to bitterness and emotional distress (Beardwood, Kirsh, & Clark, 2005). In addition, consulting behaviors may explain part of this relationship, as persons with psychological disorders and psychosocial complications are more likely to seek treatment (Aaron et al., 1996; Barksy, Orav, & Bates, 2005; Barsky, Wyshak, & Klerman, 1986; Kersh et al., 2001) and would thus be over-represented in clinical populations. Further study of these factors could help clinicians understand the unique psychological make-up of patients seeking treatment for chronic pain.

Psychological Contributions to Pain Intensity

Psychological factors were not observed to be significantly associated with pain ratings in this study. These findings are contrary to a number of studies that have reported a relationship between these psychological constructs and pain intensity. For example, as discussed above, many experiments have linked acute pain sensitivity and intensity to psychological factors, particularly somatization, catastrophization, and anxiety. However, disparities between these studies and the present study may be partially due to the
complexities of studying pain in real clinical populations, as opposed to laboratory-induced pain. In clinical populations, psychological contributions to the experience of pain are likely to be obscured by injury-related and contextual factors. It is also possible that psychological factors simply influence acute and chronic pain differently.

More difficult to explain are disparities between the present findings and other studies conducted in chronic pain populations. Depression, somatization, and anxiety were all found to explain relatively low amounts of pain rating variance (1-3%), regardless of whether exaggeration was controlled for or not. While catastrophization was found to account for a relatively high amount of the variance in reported pain in patients without spine findings (17%), it accounted for at most 1% of pain variance in the entire sample. In contrast, Sullivan’s (2001) review of studies conducted in clinical chronic pain samples concluded that catastrophizing accounts for 7% to 31% of variance in pain experience. It is not clear if injury-related differences between the present sample and those studies reported by Sullivan et al. would be enough to explain this large difference.

Some studies from the general population have also suggested a relationship between psychological complications and pain intensity. For example, Currie and Wang (2004) reported a near linear increase in diagnoses of major depression across mild, moderate, and severe pain groups. However, of note is a similar study by Carroll, Cassidy and Cote (2000) that examined depressive symptoms in chronic pain drawn from the same general population, but separated pain grades according to the degree of reported disability. In these results, pain intensity was only weakly related to depressive symptoms; contrastingly, pain-related disability showed a robust relationship with depressive symptoms. These findings were similar to the findings
observed in this study and suggest that some of the previously observed associations between depression and pain may be due in part to a failure to clearly differentiate pain intensity and pain-related disability.

It should also be mentioned that the lack of an observed relationship between psychological factors and pain intensity in this study may be partly explained by some of the limitations in how pain intensity was measured. Pain was only measured at one point in time while patients were sedentary. The resulting pain rating variable had limited variance, which can negatively impact the measurement of statistical relationships. In addition, the perception of pain intensity, like all subjective phenomena, is influenced by past experiences and interpreted relative to recent levels (Ellermeier, Westphal, & Heidenfelder, 1991). This would suggest that intra-individual pain ratings would be expected to change over time, even for comparable levels of pain. Measuring pain levels at multiple times and during different levels of activities would likely have resulted in a more accurate rating of pain and may have allowed for a clearer assessment of the relationship between pain intensity and psychological factors.

**Psychological Contributions to Perceived Disability**

One of the purposes of this study was to validate the biopsychosocial model of chronic pain and confirm the unique contribution of psychological factors to predicting pain-related disability. In agreement with the reviewed literature, psychological factors were found to have a significant relationship with pain-related disability. The observed relationship between psychological factors and reported disability was slightly weakened by controlling for the effects of exaggeration, but generally remained significant beyond demographics, medical findings, and pain rating. It is interesting to note that of the examined domains (i.e., biological,
psychological, and social), psychological factors, particularly aspects of somatization, were consistently found to be the strongest individual predictors of reported disability.

Findings from this study were generally in agreement with the reviewed literature. For example, despite using different measures, the strength of the observed relationship between depressive symptoms and reported disability ($r = .33$) was very similar to what was reported by Alschuler et al. (2008; $r = .31$) in a similar sample of patients with chronic pain. Also, in agreement with Sullivan et al. (2002), psychological complications were observed to have a stronger relationship with disability than with pain intensity. Moreover, the present findings agreed with studies employing other indicators of disability, such as failure to return to work (e.g., Trief et al., 2000; Turner et al., 2004) or retirement due to disability (e.g., Harkomaki et al., 2007).

Because this study only examined concurrent relationships between psychological complications and perceived disability, it does not speak to etiological questions concerning causation. However, present findings were similar to studies employing prospective designs to examine how psychological complications predict future disability. For example, Boersma and Linton (2006) found that psychological complications, specifically, fear-avoidance, catastrophizing, and depression, significantly predicted reported disability at a 7-month follow-up. Other prospective studies, Bigos et al. (1991) and Gatchel et al. (1995) for example, have found that elevations on Scale 3 from the MMPI-2 – the scale that was most related to disability in this study – significantly predict future back-related disability. Similarly, in a study of persons from the general population, McBeth et al. (2001) found that features of somatization predicted the onset of chronic widespread pain at a 12-month follow-up.
The implications of this causative relationship are important because they potentially guide treatment decisions and priorities. Taken together with findings from studies more directly addressing etiological questions (reviewed in more detail on pp. 10-11), to a large extent the present findings are thought to be driven by psychological contributions to disability. However, it should be mentioned that the etiological nature of this relationship is still being debated (e.g., Dersh et al., 2007; Fishbain et al., 1997); and, to date, no studies could be found that prospectively examined the relationship between psychological factors and pain-related disability while addressing issues related to symptom validity.

**Demographic Factors**

Many studies have reported relationships between demographics and pain-related disability. This study specifically examined the contributions of age, education, and gender to pain intensity and disability. In agreement with the general literature (e.g., Hagen et al., 2000), older age tended to be associated with higher reported disability, though the effect was relatively small. Age range truncation likely contributed to the weakness of this finding, as most cases fell between the ages of 35 and 55 years old.

Gender was found to be associated with pain ratings, with females reporting higher levels of pain. This pattern has been reported in other literature on chronic spine pain (e.g., Walton, Pretty, MacDermid, & Teasell, 2009) and is generally thought to reflect actual differences in experienced pain as opposed to just differences in reporting (Ellermeier & Westphal, 1995; Fillingim, King, Ribeiro-Dasilva, Rahim-Williams, & Riley, 2009).

Some gender differences were also observed regarding patterns of comorbid psychological elevations. Further examination suggested a subset of females who were more
likely to “spike” pairs of scales, particularly Hy/Hs or PCS/D; while in males there was a subset that tended to “spike” Hy/D/Hs along with either PCS or Pt. This pattern was in agreement with gender differences observed by Prokop, Bradley, Margolis and Gentry (1980) on MMPI-2 scales using cluster analytic techniques in a comparable sample of pain patients.

Given that, despite the use of gender corrected T-scores, some differences between genders were observed, all primary analyses were also run separately by gender as a post-hoc examination. Results were not presented for these analyses as no meaningful differences were observed and group sizes were too small to allow for a sufficiently detailed examination. While the underlying nature of the observed gender differences was resistant to these analyses, examination in larger samples with more suitable techniques (e.g., cluster analysis) may prove more effective.

Similar to findings by Roth and Geisser (2002), lower education was associated with higher disability. While the possibility that this finding was due to chance could not be ruled out in the present study, the consistency of this finding in the literature (e.g., Breslin et al., 2008; Dionne et al., 2001; Hagen et al., 2000) suggests a real effect. This finding also reflects the reality that chronic pain patients with lower education are likely to have fewer resources and work-related options available to them and would thus be expected to experience more actual disability than comparably injured patients with higher education (Westman et al., 2006). Interestingly, patients without spine findings and patients who were exaggerating showed the opposite trend, with higher education being associated with higher disability.

Also in agreement with Roth and Geisser (2002), education was not associated with reported pain intensity. While some studies have reported a link between lower education and
higher reported pain (e.g., Goubert et al., 2004) the present findings do not support that conclusion. Lower education was only associated with higher pain in patients who were found to be exaggerating symptoms, suggesting that failure to control for symptom validity in these earlier studies may have played a role in the observed relationship.

Medical Findings

One purpose of this study was to examine how psychological and demographic factors relate to pain intensity and disability in the context of spine-related medical findings. Towards this goal, differences between patients with spine findings and without spine findings were examined. Contrary to what would be expected, patients without spine findings reported slightly higher pain and disability. As a partial explanation for this pattern, it was expected that prevalence rates of psychological complications would be higher in those without spine findings. While a trend in this direction was generally observed, only anxiety (Pt) was significantly more prevalent in patients without spine findings. Unexpectedly, aspects of somatization measured by Scale 1 (Hs) were slightly more prevalent in patients with spine findings; however, this difference is thought to be explained by the tendency for real physical illness to contribute to elevations on this scale (Greene, 1999).

The contributions of spine pathology to pain intensity and disability were examined by assigning a severity rating according to the degree of documented medical findings. Results suggested that severity of spine findings was not associated with reported pain intensity or disability. This finding is most likely explained by the fact that the vast majority of the sample was composed of patients whose medical records indicated pathology no worse than a disc bulge. While more severe spine pathologies would be expected to contribute to pain and
disability, findings from a number of studies have reported little relationship between symptom severity and these milder forms of spine pathology (e.g., Boden & Davis et al., 1990; Boden & McCowin et al., 1990; Boos et al., 1995; Jarvik et al., 2001; Jensen et al., 1994).

It should also be mentioned that while the results of this study did not support a strong relationship between medical findings and pain or disability at the group level, this does not imply that interpretation of medical records at the individual level is not relevant. A primary issue in the assessment of patients with chronic pain is whether or not symptoms are disproportionately long-lasting or severe in the context of the injury and/or pathology. The individual importance of medical findings is likely diluted somewhat by the necessarily coarse categorization of spine findings for the purposes of this study and by the tendency for patients referred for psychological evaluation, from which this sample was drawn, to have more medically unexplained symptoms.

**Exaggeration**

The exaggeration composite created for this study was intended to capture both the breadth and severity of symptom exaggeration across the entire psychological evaluation. Considering the indicators used, this composite is thought to primarily reflect intentional exaggeration of symptoms and impairment. Supporting this contention, validation of the composite indicated that it was strongly correlated to malingering pain-related disability status classified according to criteria from Bianchini et al. (2005). As hypothesized, symptom exaggeration had a large impact on measured psychopathology, particularly anxiety and depression, and on reported disability. Exaggeration was found to have a smaller impact on reported pain intensity.
One contribution this study makes to the literature is an examination of psychosocial contributions to pain disability while accounting for the effects of symptom exaggeration. Given that exaggeration tends to increase measured levels of both psychopathology and disability, it is a reasonable concern that failure to account for exaggeration in previous research could be artificially inflating or confusing the relationship between psychological complications and disability. However, findings from this study suggest that the relationship between psychopathology and pain-related disability cannot be fully explained by symptom exaggeration. While controlling for exaggeration did slightly weaken the relationship between psychological factors and reported disability, the selected psychological constructs were still found to be significantly related to disability.

Examination of high and low exaggerators separately revealed some differences between them. For example, in patients who were exaggerating, more severe spine findings tended to be associated with lower pain and disability. Also observed in high exaggerators was the finding that measured levels of catastrophizing and anxiety had the opposite relationship with reported pain intensity than expected; higher scores were associated with lower reported pain. A closer inspection of the data suggested that a subset of high exaggerators presented with very high reported pain, but suppressed scores on the catastrophizing and anxiety scales. These findings highlight the difficulty of treating symptom validity as a unidirectional influence towards exaggeration. In practice, patients may be motivated to present in a variety of ways depending on their situation and conceptions about pain and disability, and these differences can result in a variety of effects on neuropsychological testing (Rogers, 2008).
Functional Impairment and Disability

It is important to consider how this study relates to functional impairments and disability in patients with chronic pain. Impairment is defined by the American Medical Association’s (AMA) Guides to the Evaluation of Permanent Impairment as “a loss, loss of use, or derangement of any body part, organ system, or organ function” (Cocchiarella & Anderson, 2001, p. 2). While some changes have recently been made in this regard, the AMA has not classically viewed pain itself as an impairment. Instead, pain is typically considered a symptom that can potentially lead to impairments by impacting physical, cognitive, or psychological functions. Related to this, the AMA defines disability as an "alteration of an individual's capacity to meet personal, social, or occupational demands because of an impairment" (p. 600). Thus, disability refers to the functional limitations in daily life that result from impairments caused by injury or illness.

Establishing the overall degree of disability an individual experiences – let alone the disability experienced in any single domain – is complicated by numerous personal and contextual factors. For example, when it comes to work-related disability, two individuals with the same impairment in lifting capacity could have drastically different levels of disability depending on their job demands. Due to these inherent difficulties, determining disability often requires a synthesis of information from measures involving functional performance and self-report. Thus, to understand how this study relates to true functional disability, it is important to consider how these findings potentially relate to physical impairment.

Some studies have suggested there are only moderate correlations between perceived disability and functional performance in patients with chronic pain (Reneman, Geertzen,
Groothoff, & Brouwer, 2008; Schiphorst Preuper et al., 2008). However, there is clear evidence that psychological complications are associated with real impairments in physical capacities (Geisser, Robinson, Miller, & Bade, 2003). For example, a study by Alschuler et al. (2008) found that depression was a significant predictor of both perceived and real physical impairment, even after controlling for demographics and pain intensity. Alschuler et al. observed that physiologic effort, as measured by heart rate, partially mediated the relationship between depression and physical function, but the relationship remained significant even after controlling for physiologic effort.

Alschuler et al.’s (2008) findings paralleled the findings in this study, where exaggeration was found to partially – but not fully – account for the relationship between psychological complication and disability. Taken together, the results from the present study and those from Alschuler et al. provide mutual support for the assertion that psychological factors impact both perceived and functional disability, even after accounting for exaggeration and reduced effort. Put more broadly, these findings suggest that psychologically-related poor outcomes in chronic pain may be at least partly explained by actual reductions in capacity in addition to other factors such as transitory behavioral changes and/or disability misperceptions. These findings lend support to chronic pain models that suggest behavioral factors may contribute to lasting physiologically-based impairments and disability (e.g., the fear-avoidance model, Leeuw et al., 2007).

Clinical Implications for Assessment and Treatment

 Clinically, the principal issue addressed in this study is whether psychological assessment, including measurement of psychopathology, personality, and symptom validity,
provides clinically useful information for interpreting reported pain-related disability. This issue is important for informing health care decisions regarding appropriate assessments, treatments, and rehabilitation of patients with chronic pain. Results support the utility of psychological assessment for these purposes, particularly in the absence of medical findings to explain reported pain and disability. Psychopathology was common in this population and was the strongest individual predictor of disability.

Assessment and consideration of psychosocial factors has important implications for the selection of appropriate treatments for a patient with chronic pain. For example, consideration of psychosocial risk factors can help prevent unnecessary invasive treatments (e.g., surgery), which can pose their own serious risks, including death (Eisendrath & McNeil, 2004). Consideration of psychosocial risk factors can also help prevent over-prescribing and over-use of potentially addictive narcotic pain medications and related adjuvants, such as anxiolytics or muscle relaxants (Longo et al., 2000).

In cases where invasive treatments and/or medication are called for, addressing psychological complications is still important for improving outcome (Block et al., 2003; Polatin & Dersh, 2004). As described by Block et al., “Spine surgery’s ultimate effectiveness . . . depends on much more than the surgeons’ diagnostic acumen and technological skill. Psychological factors exert very strong influences – ones that can improve, or inhibit, the patient’s ultimate recovery . . . surgical results can be greatly augmented by the inclusion of psychological components in the assessment and preparation of patients for spine surgery, as well as in post-operative rehabilitation” (p. 4). Supporting this position, a review by Guzman et al. (2001) determined that intensive multidisciplinary biopsychosocial rehabilitation with a functional
restoration approach, which includes systematic management of psychological risk factors, has been shown to provide better functional outcomes for patients with chronic back pain compared to outpatient non-multidisciplinary treatments.

**General Issues & Considerations**

Several methodological considerations regarding this study are important to mention. First, this sample is primarily composed of patients who were referred for psychological evaluation as part of a worker’s compensation or personal injury claim. Thus, this is a selected group of patients referred from a particular sub-population of chronic pain patients. Of all persons who suffer from chronic pain, these cases represent a relatively small sub-population of patients, who are much more likely to have psychosocial complications. As such, reported rates of psychological complications should only be considered representative of this population of patients.

Similarly, this study specifically examined patients with spine-related pain. While the results likely speak to other types of musculoskeletal pain, more studies will be necessary in other patient populations before these findings can be generalized to other types of chronic pain. This is particularly true for types of chronic pain that appear to result from different etiological mechanisms, such as fibromyalgia or chronic widespread pain.

Another issue that should be mentioned is that the relatively small sample size should be considered when interpreting results. For the primary analyses of hypothesized relationships, modest group sizes would be expected to result in less precision and more concern about type II errors. For the subgroup and post-hoc analyses, the smaller sample sizes and multiple comparisons create concern about both type I and type II errors. Replication in
larger samples would help confirm observed findings, including weaker effects that could not be differentiated from chance.

Finally, all of the measures used in this study relied on self-report and thus would be expected to share methodological variance. While this study attempted to account for the effects of exaggeration across these measures, other factors such as disclosure or social desirability may be systematically influencing the selected self-report measures. Further studies employing methods that can help identify and correct for shared methodological variance (e.g., path modeling), or studies employing alternative methods of measuring pain or disability (e.g., physiologic), could help control for these potential confounds.

**Summary**

This study examined the contributions of biopsychosocial factors to pain intensity and disability in patients with chronic spine-related pain. As hypothesized, psychological complications were prevalent and had a strong relationship with reported disability, predicting it beyond demographics, medical findings, and pain intensity. These findings support the importance of psychological assessment for patients with chronic pain-related disability. The results of this study, along with many other recent studies, continue to demonstrate the need to consider psychosocial factors when addressing functional disability in patients with chronic pain.
REFERENCES


APPENDICES

Appendix A: Common Pain-Related Spine Pathologies

The following anatomical descriptions are based on information from multiple sources including Adams, 2004; Block, Gatchel, Deardorff, and Guyer (2003), Giles and Singer (1997), and Filler (2004).

**Disc Bulge and Herniation**

The spine is composed of vertebrae segments that are connected to each other by a complex consisting of two facet joints and an intervertebral disc. The disc is composed of a nucleus surrounded by layers of fibrous cartilage called the annulus and interfaces with the vertebral bodies at the disc endplate. Over time vertebral discs may naturally begin to bulge outward beyond the vertebral body margins as they lose elasticity and expand horizontally. If the disc continues to degenerate or is exposed to traumatic mechanical stress a herniation may occur as the annular fibers tear and allow the disc nucleus to protrude against or through the annulus wall. In these conditions, pain may result directly from the annulus tears, from irritation caused by the release of chemicals from the nucleus, or by compression of the nerve root.

**Facet Joint Syndrome**

The facet (or zygapophyseal) joints may cause pain directly through arthritic processes, or indirectly by impinging on nearby structures such as the nerve root. The processes responsible for the development of facet joint pain often co-occur with disc degeneration and thus distinguishing the specific etiology of pain symptoms is difficult.
**Musculoligamentous Injuries**

Muscle sprains and strains are relatively common consequences of strenuous physical activity. Sprains are injuries involving ligaments, which are bands of cartilage that connect bones and hold them in alignment. Sprains are usually caused by trauma that displaces a joint resulting in stretching or tearing of the associated ligament(s). Strains are injuries involving muscles or the tendons that attach muscles to bone. Strains are usually caused by a quick movement that over-stretches or over-contracts a muscle resulting in damage or tearing to the muscle or tendon. Treatment and recovery depend on the severity of injury. Mild to moderate injuries will typically heal with self-care and rest while severe cases may require immobilization or surgery.

**Radiculopathy / Sciatica**

Radiculopathy refers to a disruption of the nerve root that can result in pain as well as sensory or motor disturbances. An important feature of radiculopathy is that symptoms are often referred to the limb associated with the disrupted nerve. Radiculopathy involving the sciatic nerve, often referred to as sciatica, typically manifest unilaterally in the lower back and legs.

**Spondylolysis & Spondylolisthesis**

Spondylolysis refers to a stress fracture of the pars interarticularis, the narrow bridge between the upper and lower facet joint of a vertebrae. A condition known as spondylolisthesis can occur if the fracture is bilateral and the vertebrae slip out of alignment. Most consider spondylolysis to represent a fatigue fracture resulting from chronic mechanical stress rather than a single traumatic event, though trauma may worsen a condition. Spondylolysis is
relatively common, particularly in athletes, and most cases are asymptomatic. When pain is present it is thought to be caused by nerve root compression, intervertebral disc pain, or facet joint pain.

**Spondylosis**

Spondylosis is a condition caused by age-related disc degeneration that causes a number of pathological processes that can ultimately result in a narrowing of the spinal canal. One mechanism is the formation of osteophytic bars along the ventral spinal canal caused by increased mechanical stress. Pain may result from compression or irritation of the cauda equine or nerve root.

**Stenosis**

Spinal stenosis refers to a narrowing of the spinal canal, nerve root canal, or foraminal openings from which nerve roots exit the canal. Symptoms typically occur when these nerve fibers become impinged. The condition can be congenital, but is more commonly acquired through degenerative processes. Cervical stenosis is associated with radiating arm pain, numbness, and paresthesia. Lumbar stenosis is associated with lower back pain and radiating bilateral or unilateral leg pain. More severe cases may present with other symptoms including myelopathy (spinal cord dysfunction).
Appendix B: Diagnostic Procedures

The following descriptions of diagnostic procedures are based on information from multiple sources including Giles and Singer (1997) and Filler (2004).

Diagnostic Injections

Injection of local anesthetics, steroids, neural blockades, or even irritants can be useful for determining the source of spinal-pain symptoms. A typical procedure involves the injection of the agent into a target location after which changes in the patient’s pain symptoms are noted. These techniques can help identify the source of pain symptoms allow for differentiation between local vs. referred pain, somatic vs. visceral pain, and peripheral vs. central etiologies. While diagnostic injections can offer the advantage of pinpointing a specific cause of symptoms, it should be noted that these procedures rely on the patient’s accurate report of symptoms and some have been criticized for having poor specificity in patient populations with external incentives.

Injections can also be used to introduce contrast materials to enhance standard imaging techniques. Arthograms involve the injection contrast agents into a joint to better image interior soft tissues. In a myelogram, contrast agent is introduced into the dura surrounding the spinal cord and nerves which allows for a detailed view of nerve arrangement and impingements. Discograms involve the injection of contrast material into the nucleus of an intervertebral disc to highlight any defects in the disc’s structural integrity. Often discograms also serve as a diagnostic injection due to the mildly irritating nature of the contrast material. If the injection elicits symptoms that are similar to those normally experienced it is considered an
indication that the targeted disc is responsible. However, as with all diagnostic injections, reliance on patient report can call the accuracy of the procedure into question.

**Electrodiagnosis**

Electrodiagnostics involves the study of human physiology using devices that produce and measure electrical current in the body. An electromyogram (EMG) uses a needle to directly measure the electrical activity of a muscle during different stages of activity. Abnormal electrical activity can indicate nerve and muscle pathologies. A nerve conduction study (NCS) delivers an electrical charge to a peripheral nerve while a recording electrode is placed in the innervated muscle. This arrangement allows for the determination of the nerve conduction velocity which is a sensitive indicator of nerve damage. A NCS also has the advantage of being able to isolate the specific site of nerve damage or impingement by stimulating the nerve at various locations along its path.

**Imaging**

Radiography and magnetic resonance imaging (MRI) are the most common forms of imaging. Radiography involves the use of x-ray to view internal tissue and is particularly useful for examining bony structures in the body. The three main categories of radiography are: 1) static images, 2) fluoroscopy, and 3) computed tomography (CT). Static images are classic x-ray snapshots on film. Fluoroscopy is the use of x-rays to provide real-time dynamic internal imaging – a technique often used to guide the placement of instruments during surgical procedures. CT scanning uses x-rays to collect numerous image slices which are then assembled into a detailed 3-dimensional structural view. MRI uses strong magnetic fields to provide what is essentially an image of water distribution in the body. MRI is particularly suited to examining
soft tissue structures and the high definition images allow for very accurate identification of
spinal disc herniation and nerve root compression.

**Physical Examination**

Physical examination can aid in diagnosis as well as provide information about the type
of degree of functional limitations. Musculoskeletal aspects examined may include gait,
posture, sensitivity to palpation, range of motion, and strength. Neurological aspects examined
may include focal CNS signs, motor disturbances, reflexes, and muscle tone.
Appendix C: The Pain Catastrophizing Scale

We are interested in your thoughts and feelings related to pain and distress. While responding to the following 13 items, please think of your physical pain. With this experience in mind, rate the frequency with which you experienced each of the following thoughts and feelings. Use the following scale (from 0 to 4) to indicate how frequently you had each thought or feeling during your experience of pain, and mark your frequency rating on the blank line to the left of the item.

0 1 2 3 4
(not at all) (all the time)

1. I worry all the time about whether the pain will end
2. I feel I can't go on
3. It's terrible and I think it's never going to get any better
4. It's awful and I feel that it overwhelms me.
5. I feel I can't stand it anymore
6. I become afraid that the pain may get worse
7. I think of other painful experiences
8. I anxiously want the pain to go away
9. I can't seem to keep it out of my mind
10. I keep thinking about how much it hurts
11. I keep thinking about how badly I want the pain to stop
12. There is nothing I can do to reduce the intensity of the pain
13. I wonder whether something serious may happen
Appendix D: The Pain Disability Index

The rating scales below are designed to measure the degree to which several aspects of your life are presently disrupted by chronic pain. In other words, we would like to know how much your pain is preventing you from doing what you would normally do, or from doing it as well as you normally would. Respond to each category by indicating the overall impact of pain in your life, not just when the pain is at its worst.

For each of the 7 categories of life activity listed, please circle the number on the scale which describes the level of disability you typically experience. A score of 0 means no disability at all, and a score of 10 signifies that all of the activities in which you would normally be involved have been totally disrupted or prevented by your pain.

1. **Family / home responsibilities**
   This category refers to activities related to the home or family. It includes chores, or duties performed around the house (e.g., yard work) and errands or favors for other family members (e.g., driving the children to school).

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2. **Recreation**
   This category includes hobbies, sports, and other similar leisure time activities.

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3. **Social activity**
   This category refers to activities which involve participation with friends and acquaintances other than family members. It includes parties, theater, concerts, dining out, and other social functions.

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4. **Occupation**
   This category refers to activities that are a part of or directly related to one's job. This includes non-paying jobs as well, such as that of a housewife or volunteer worker.

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5. **Sexual behavior**
   This category refers to the frequency and quality of one's sex life.

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6. **Self-care**
   This category includes activities which involve personal maintenance and independent daily living (e.g., taking a shower, eating, getting dressed, etc.).

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7. **Life-support activity**
   This category refers to basic life-supporting behaviors such as eating, sleeping, and breathing.

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Appendix E: Institutional Review Board Approval

Jonathan Ord

From: Robert D Laird [rlaird@uno.edu]
Sent: Friday, October 23, 2009 10:46 AM
To: Kevin Wood Greve; Jonathan Steven Ord
Subject: IRB Approval 06Dec09

University Committee for the Protection
of Human Subjects in Research
University of New Orleans

Campus Correspondence

Principal Investigator: Kevin Greve
Co-Investigator: Jonathan Ord
Date: October 23, 2009
Protocol Title: "Biopsychosocial factors in chronic spine-related pain: Contributions to pain intensity and perceived disability"
IRB#: 06Dec09

The IRB has deemed that the research and procedures are compliant with the University of New Orleans and federal guidelines. The above referenced human subjects protocol has been reviewed and approved using expedited procedures (under 45 CFR 46.116(a) category (8)).

Approval is only valid for one year from the approval date. Any changes to the procedures or protocols must be reviewed and approved by the IRB prior to implementation. Use the IRB number listed on this letter in all future correspondence regarding this proposal.

If an adverse, unforeseen event occurs (e.g., physical, social, or emotional harm), you are required to inform the IRB as soon as possible after the event.

Best wishes on your project!

Sincerely,

Robert D. Laird, Ph.D., Chair
UNO Committee for the Protection of Human Subjects in Research
VITA

Jonathan Ord attended Evergreen State College and received a B.S. in December of 2003. In the fall of 2005, Jonathan began working with Dr. Greve at the University of New Orleans in the Applied Biopsychology Doctoral program. Jonathan received his M.S. in the fall of 2007 and was awarded the Andrew S. Wensel Distinguished Graduate Student Award in April, 2010. He is currently working on a series of research projects related to measures of psychological and functional performance in patients with chronic pain.