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Measurement of pain, pain disability, and pain beliefs of morbidly obese adults

Brooke Louise Barbera
Louisiana State University and Agricultural and Mechanical College

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MEASUREMENT OF PAIN, PAIN DISABILITY, AND PAIN BELIEFS
OF MORBIDLY OBESE ADULTS

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Brooke Louise Barbera
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M.A., Louisiana State University, 2008
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To my family

for their encouragement and support of my education

To Chadwick

with love and gratitude

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ABSTRACT

Both obesity and pain are pervasive public health problems, contributing to significant

disability in the United States and worldwide. Studies have demonstrated a positive

association between obesity and pain, with increasing BMI related to increasing levels of pain

intensity; however, the mechanisms underlying this relationship are not well understood. The

present study assessed the presence of pain, pain-related disability, and pain beliefs in a

morbidly obese (BMI ≥ 40 kg/m^2) sample participating in a medically-supervised, nonsurgical

weight loss intervention. After controlling for demographic variables and depression, pain

was reported in 36.7% of the sample (N = 390). Results indicated that greater level of pain

severity at the outset of treatment was significantly associated with poorer weight loss

treatment outcome at one year [$F(3, 205) = 20.50, p < .001$]. Pain beliefs, specifically

catastrophizing, did not contribute to this relationship. The presence of pain was also

significantly related with decrements in health-related quality of life, after controlling for the

impact of age, BMI, and depression [$F(4, 204) = 14.61, p < .001$]. The findings of the present

study provide preliminary evidence that pain severity is an important indicator of treatment

outcomes for morbidly obese individuals trying to lose weight by nonsurgical methods.

Further research is needed to more clearly understand prognostic implications of the

comorbidity of pain on obesity treatment, as well as the impact of incremental weight loss on

pain outcomes.

INTRODUCTION

The Obesity Epidemic

Obesity is a pervasive and costly public health phenomenon, heralded frequently as a worldwide epidemic. Globally, the number of overweight and obese people is increasing dramatically (Haslam and James, 2005). In the United States, the prevalence of obesity has risen in every state over the last 15 years (Centers for Disease Control and Prevention, 2010; Flegal, Carroll, Ogden, & Curtin, 2010). The simple definition of obesity as excess body fat is easy to comprehend, but obesity is not as easy to measure due to the wide variability in human body composition and shape. The World Health Organization (WHO) has adopted a definition for overweight and obesity using the body mass index (BMI = weight/height^2 in kg/m^2). Obesity is defined as a BMI greater than 30 kg/m^2 (WHO, 2011). Recent data from the National Health and Nutrition Examination Survey (NHANES) estimates that 68.3% of American adults were classified as overweight (BMI $\geq$ 25 kg/m^2), and 33.9% were obese (Flegal, et al., 2010). Extreme or morbid obesity, defined as BMI $\geq$ 40 kg/m^2, is remarkably prevalent in the United States, occurring in 4.2% of men and 7.2% of women though 2008 (Flegal, et al., 2010), rising from 2.8% and 6.9%, respectively, in 2004 (Ogden, Carroll, Curtin, McDowell, Tabak, Flegal, 2006). Over the last 20 years, more than 300 million people were classified as obese worldwide, and the incidence of morbid obesity has doubled (Deitel, 2003).

The health burden associated with obesity is significant, as it contributes to the development of numerous chronic medical illnesses (Bray, 2004). Obesity is associated with coronary artery disease, hypertension, diabetes, congestive heart failure, sleep apnea, hyperlipidemia, stroke, gallbladder disease, and a variety of cancers (Bray, 2004; Crawford, Cote, Couto, Daskiran, Gunnarsson, Haas, et al., 2010). Furthermore, gastroesophageal reflux

disease, nonalcoholic fatty liver disease, osteoarthritis, and psychological disturbances have increased prevalence in obese patients and contribute to overall morbidity (Bray, 2004; Crawford, et al., 2010). Obesity takes a great toll on American health and mortality, and it remains the second leading preventable cause of mortality with an estimated 400,000 deaths occurring annually (Mokdad, Marks, Stroup, & Gerberding, 2004).

Definitions of Obesity

The most elementary definition of obesity is excess adipose tissue in the body; however, the pathophysiology of obesity is more complicated. Obesity is a problem of metabolic dysfunction, affecting all major systems of the human body (Redinger, 2007). Weight gain and obesity are described in the literature as the result of many factors, each individually complex: genetic, physical, behavioral, and psychological. Defining the obesity epidemic as solely a problem of imbalance of energy consumption (intake) to energy expenditure (activity) fails to recognize the organ system and immune dysfunction that are both consequences of and exacerbated by excessive body weight. Even further, immune dysfunction related to obesity has created toxic metabolic environments that are not able to ward off further disease, instead making the body readied for future illness. The multitude of comorbid conditions listed above is evidence of this multi-organ dysfunction. Although varying from study to study, approximately 30 to 40% of the variance in BMI can be attributed to genetics and 60 to 70% to environment (Pi-Sunyer, 2002). A widely-referenced analogy for this complexity is from Dr. George Bray, "genes load the gun and a permissive or toxic environment pulls the trigger" (Bray, 2007, pg. 32). Indeed, the rapidity at which this epidemic is expanding worldwide indicates the strong interaction of environment with the underlying susceptibility of the population.

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Bray described two pathophysiological categories for diseases related to obesity: the increase in the number of fat cells or the increase in their size (Bray, 2004). Increased fat mass in the body is affected by a number of factors, although androgen and estrogen hormones have the greatest effect in early life and adolescence. Later age-related accumulation of fat is more likely related to gender and associated changing levels of cortisol and testosterone. The increase in fat mass affects the human body's degree of insulin resistance, which can enhance the risk of diabetes, gall bladder disease, hypertension, and cardiovascular disease. Increased fat mass is also associated with osteoarthritis and sleep apnea, as well as social stigma, which can lead to changes in behavioral responses (Bray, 2004). Individual fat cell enlargement is also important in understanding overweight. The fat cell is a type of endocrine cell that directly creates metabolic change through the release of peptides (Bray, 2004). Perhaps the most significant of these is leptin, an adipose-released hormone that regulates the inhibition of appetite through the hypothalamus (Caro & Considine, 2004). In simplistic terms, leptin is the body's indicator of energy balance. Although the precise dynamics of leptin are still being studied, it is understood as having a direct influence on the body's metabolism.

Treatment of Obesity

Obesity as a complex biological, environmental, psychological, and social phenomenon similarly requires the implementation of multiple strategies in treatment. In recent decades, the treatment for obesity has taken many forms, studied as individual components and taken together in multiplicity: reduced energy consumption, increased energy expenditure, cognitive-behavioral therapy, medications aimed at reducing adipose tissue or reducing the body's ability to absorb fats, and surgery to alter the body's digestive processes. The application of one or more of these strategies in a clinical setting is highly dependent on the obese individual. Wadden and

colleagues (2002, 2012) have suggested a treatment selection algorithm based on BMI classification to help guide choice of treatment components.

According to the "BEST Treatment" assessment developed by Wadden and Phelan (2003), the most effective non-surgical weight management interventions use a combined approach of diet, physical activity, and behavior modification (Wadden, Webb, Moran, Bailer, 2012). Intensive medical approaches combining various elements of group behavior therapy, individual behavior therapy, structured diet, very low calorie diets, physical activity regimens and medications have been shown in small studies to produce weight loss that approximates 16% or more from baseline (Pi-Sunyer, et al., 2007; Wadden, Berkowitz, Sarwer, Prus-Wisniewski, Steinberg, 2001; Wadden, et al., 2012). Treatment studies have demonstrated that body weight can be reduced in 6 months, although natural processes of maintenance are hard to achieve as study samples repeatedly show regain after the 6 month mark (Wadden, et al., 2012). The more successful treatments have aimed at what is termed "lifestyle modification," in that participants are educated on skills that are expected to continue well after the study period is over, with the hopes that continued weight loss and management will continue over time (Wadden, Butryn, Wilson, 2007). Lifestyle modification is the closest approximation of treatment that can broaden not only to a way of eating and energy balance, but also healthy behaviors that aim to target some of the comorbidities of obesity and overweight (Wadden, Butryn, Wilson, 2007; Wadden, et al., 2012).

Standards for defining treatment success tend to differ by modality and BMI status. The United States Institute of Medicine (IOM) published the long-held definition of a clinically significant weight loss among nonsurgical treatment types as 10% loss of initial body weight maintained for one year (IOM, 1995). This definition has been maintained by prominent obesity

researchers in defining treatment success (Ryan, Johnson, Myers, Prather, McGlone, Rood, et al., 2011; Wing & Hill, 2001; Wing & Phelan, 2005). This level of weight loss is associated with improvement in related co-morbidities, such as hypertension, dyslipidemia, and insulin insensitivity (National Institutes of Health and the National Heart, Lung, and Blood Institute, 2000). The United States Food and Drug Administration (USFDA) considers pharmacotherapy as effective if treated patients lose 5% initial body weight (United States Department of Health and Hospitals, 2007). However, these guidelines are not often applied in extreme obesity (BMI $\geq$ 40 kg/m^2), as nonoperative treatment strategies have not been demonstrated as effective in U.S. samples (Wadden & Osei, 2003).

Treatment of Morbid Obesity. Extreme or morbid obesity is typically associated with more serious health complications than lesser degrees of excess weight, and its treatment is similarly more complex. Surgery is currently the only form of treatment for morbid obesity that has been demonstrated to produce durable (i.e., 10 years' duration) weight loss exceeding sixteen percent of baseline weight (Martins, Strømmen, Stayne, Nossum, Mårvik, Kulsend, 2011; Sjöström, 2000). Surgical therapy for obesity includes several different types of operations, including more recently developed laparoscopic procedures to reduce postoperative infections and complications (Tessier & Eagon, 2008). Although the NHLBI states doubt regarding the effectiveness of "more conservative" (i.e., non-operative) procedures for extreme obesity in their obesity treatment guidelines (2000), operative treatment is not possible for a large majority of morbidly obese individuals. Many comorbidities of obesity, conditions which may be improved with surgical treatment, may be severe enough health complications that actually preclude surgical candidacy. Furthermore, bariatric procedures are costly, creating a financial burden as a major medical procedure requiring extensive recovery and follow-up. Bariatric surgical

procedures of all types can also create lifelong metabolic abnormalities, with both psychological and social implications. Given that the number of surgical procedures performed in the U.S. represents only a small fraction of the population with extreme obesity (Zhao & Encinosa, 2007), continued research of non-operative methods of treatment for this group are necessary.

Pain as a Comorbidity

Despite a large body of research examining the multitude of obesity comorbidities, one concomitant problem that is often overlooked is pain. Like obesity, pain is a complex phenomenon, with biological, psychological, and social factors affecting an individual's development and experience of pain. The International Association for the Study of Pain (IASP, 2011, p. 25) published a widely accepted definition of pain as, "An unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage … Pain is always subjective … It is unquestionably a sensation in a part or parts of the body, but it is also always unpleasant and therefore also an emotional experience." This definition highlights pain as a sensory process, acknowledging the action of the body's sensory organs and receptors to convert physical stimuli and energy into neural impulses sent to the brain. However, the IASP emphasizes pain as a subjective experience and perceptive process, highlighting the importance of the brain's organization and interpretation of these neural impulses. Pain is an individual experience with severity defined across a broad continuum (IASP, 1994; IASP, 2011). Pain sensation, transmission, modulation, and interpretation are functions of the central nervous system, and when abnormalities in these processes occur, pain is regarded as a neurologic disease (Board on Health Sciences Policy, 2011).

Assessment of Pain. Given the IASP definition of pain as a subjective experience, the assessment of pain is inherently different than that of weight. While excess fat mass is a

quantifiable condition that can be seen, weighed, and measured without the input of the individual, an outsider cannot detect an individual's pain through their own senses. Although clinical findings (i.e., broken bone or tissue damage) or pain-related behaviors (i.e., grimacing or changes in mobility) may be observed, these do not correlate well with the severity of pain reported by patients (Melzak & Katz, 1994; Miaskowski, Bair, Chou, D'Arcy, Hartwick, Huffman, et al., 2008). Because pain often occurs in the absence of observable injury, the long-standing gold standard for pain assessment has been self-report (Cleeland, 1989; Greve, Bianchini, Ord, 2012; Melzak & Katz, 1994). However, assessment by this method must be cautious given that self-reporting can be influenced by numerous factors including cognitive abilities, mood, sleep disturbance, and medication usage (Peter & Watt-Watson, 2002). Secondarily, pain may be measured by the observance of a number of pain-related behaviors, including the monitoring of medication usage (Greve, Bianchini, Ord, 2012; Melzak & Katz, 1994).

The importance of pain assessment is underscored by its economic impact. Like obesity, pain is a pervasive and expensive public health problem, with chronic pain affecting an estimated 116 million American adults (Board on Health Sciences Policy, 2011). Arthritic pain is the leading cause of disability in the United States (National Center for Chronic Disease Prevention and Health Promotion, 2011). Pain represents an estimated $635 billion in costs from medical treatment and lost productivity (Board on Health Sciences Policy, 2011), as well as reduced job performance (Stewart, Ricci, Chee, Morganstein, Lipton, 2003). The lack of attention to pain in obese populations is perhaps not surprising given that underassessment of pain is extremely common, even despite its being the number one reason people seek medical attention (Berry &

Dahl, 2000). In fact, the most common reason for the undertreatment of pain is the failure of clinicians to assess pain and pain relief (Miaskowski, et al., 2008).

Co-occurrence of Obesity and Pain. Increasing levels of pain have been observed across the continuum of BMI classification, from healthy BMI to extreme obesity (Stone & Broderick, 2012). Results from a community-based twin registry confirmed the relationship between weight and a variety of painful conditions (e.g., low back pain, headache, fibromyalgia, chronic widespread pain; Wright, Schur, Noonan, Ahumada, Buchwald, Afari, 2010). Specifically, overweight and obese twins were more likely to report these conditions than normal weight twins after controlling for age, gender, and depression (Wright, et al., 2010). In a survey sample of over 3,500 individuals living in the southeast U.S., Hitt and colleagues (2007) reported that BMI was positively associated with increased pain over the last month. Importantly, this association was observed across the spectrum of BMI classification, even after controlling for age, gender, race, education, and the presence of healthcare coverage (Hitt, McMillan, Thornton-Neaves, Koch, Cosby, 2007). Furthermore, individuals classified with extreme obesity (BMI $\geq$ 40 kg/m^2) were 2.3 times more likely than those with lesser degrees of obesity to report moderate to severe pain (Hitt, et al., 2007). The most recent survey of obesity and pain in a sample of over one million U.S. adults, conducted by The Gallup Organization, found that BMI and pain yesterday were reliably associated when demographic variables were controlled. This association was found in both men and women, and became stronger in older adults (Stone & Broderick, 2012). Similarly, results from 407 participants in the Einstein Aging Study demonstrated that central (i.e., abdominal) obesity had the strongest independent association with pain over the last three months (Ray, Lipton, Zimmerman, Katz, Derby, 2011). Ray and colleagues noted that the

relationship between obesity and pain in older adults was independent, not explained by coexisting markers of insulin resistance or inflammation, osteoarthritis, or neuropathy.

Of the studies that assess for the presence of pain in obese populations, compelling numbers have emerged. Fontaine and Barofsky (2001) found that of the 312 overweight participants seeking weight management in their university setting, over half (56%) of the participants reported moderate or severe pain. The most common types of pain were low back pain and joint pain. In another study, Fontaine and colleagues also observed that 56% of their "treatment-seeking" sample of obese persons had co-existing chronic pain, with low back pain being the most prevalent pain condition in this group (Fontaine, Bartlett, Barofsky, 2000). When compared to their non-treatment-seeking but similarly obese counterparts, pain was still observed in nearly one-third of participants (Fontaine, et al, 2000). In one of the largest samples to date, the Swedish Obese Subjects (SOS) study documented that of their 6,238 obese registry subjects, the percentage reporting pain in at least one of five locations was 57.9% for men and 68% in women (Peltonen, Lindroos, Torgeson, 2003).

Pain Conditions Associated with Obesity. Although an analysis of all possible pain conditions is outside of the scope of the present dissertation, there are a number of conditions that have been examined as related to increased weight: osteoarthritis, low back pain, and chronic widespread pain (Janke, Collins, Kozak, 2007), as well as fibromyalgia (Okifugi, Donaldson, Barck, Fine, 2010). Perhaps the most logical association of pain resulting from overweight and obesity is musculoskeletal pain, as it may be intuitive that increasing pressure placed on the body's joints, muscles, and connective tissue by excess weight would cause pain. Reciprocally, reduced activity resulting from premorbid musculoskeletal disorder could lead to

weight gain. Two musculoskeletal conditions, osteoarthritis and low back pain, are the most frequently examined in relation to obesity (Janke, et al., 2007).

Osteoarthritis is the most common joint disorder, characterized by pain and stiffness in the joints, inflammation, tenderness, and limitation of joint movement (Tietel & Zieve, 2011). Although the course of osteoarthritis varies, it is often both progressive and irreversible. Treatment can range from use of over-the-counter medications such as Tylenol or non-steroidal anti-inflammatory drugs to the injection of corticosteroids or artificial joint fluid (Tietel & Zieve, 2011), as well as surgical joint replacement (Janke, et al., 2007). Osteoarthritis of the knee and hip tend to be the most disabling sites, contributing to excess health care utilization independent of demographic variables and comorbidities including obesity (Wright, Katz, Cisternas, Kessler, Wagenseller, Losina, 2010). Research evidence demonstrates that high BMI is a risk factor for the development and progression of osetoarthritis in these areas. Analyses from the earliest NHANES data showed that adults with BMI ≥ 30 kg/m^2 had a four times greater prevalence of osteoarthritis of the knee than individuals with BMI ≤ 30 kg/m^2 (Anderson & Felson, 1988). Later research not only concurs with these results (Felson, 1990), but indicates further increasing prevalence of osteoarthritis of the knee and hip in the context of the obesity epidemic (Wang & Beydoun, 2007; Losina, Walensky, Reichman, et al., 2011). The American College of Rheumatology treatment guidelines recommend weight loss for overweight and obese individuals with osteoarthritis (American College of Rheumatology Subcommittee on Osteoarthritis, 2000). Among normal weight individuals, moderate increases in BMI have been demonstrated as significantly related to knee osteoarthritis, indicating that even modest levels of overweight may increase the risk for developing the disorder (Holmberg, Thelin, & Thelin, 2005).

Low back pain is also a chronic pain condition, and a common health problem of many adults (Brooks, 2006). Yet, definitions of low back pain lack uniformity (Hoy, March, Brooks, et al., 2010), and research regarding the nature of the relationship between low back pain and obesity lacks conclusive evidence (Janke, et al., 2007; Sellinger, Clark, Shulman, Rosenberger, Heapy, Kerns, 2010). Several studies have identified increased body weight as an independent risk factor for the development of chronic back pain in both women and men (Brown, Mishra, Kenardy, et al., 2000; Fransen, Woodward, Norton, et al., 2002; Lake, Power, Cole, 2000; Sellinger, et al., 2010; Shiri, Karppinen, Leino-Arjas, Solovena, Viikara-Juntura, 2010; Webb, Brammah, Lunt, Urwin, Allison, Symmons, 2003), suggesting a possible dose-response relationship. Similarly, a large cross-sectional study of over 15,000 participants with common spine disorders and related pain found that increased BMI was positively associated with increased disability and severity of pain symptoms (Fanuele, Abdu, Hanscom, Weinstein, 2002), but also with more comorbidities when compared to a nonobese sample. Longitudinal studies, however, do not show a causal relationship between overweight & obesity and low back pain (Janke, et al., 2007; Shiri et al., 2010). In their review, Janke and colleagues (2007) suggested that the difficulty in understanding the relationship can be explained by poorly definitions of low back pain, mediation by lifestyle factors, and an overall weak relationship that is likely stronger in much higher BMI (≥ 30 kg/m^2) classifications, rather than across the spectrum. More recently, neuroengineering studies have made attempts to explain the obesity-low back pain relationship, demonstrating that significant gait disturbance (Cimolin, Vismara, Galli, Zaina, Negrini, Campodaglio, 2011) and spinal mobility impairment (Vismara, Menegoni, Zaina, Galli, Negrinir, Capodaglio, 2010) are found in obese adults with low back pain, when compared to obese individuals without low back pain.

Physical Disability and Quality of Life. The comorbidity of physical pain and obesity may act in a reciprocal fashion to increase physical disability. For example, the presence of pain may contribute to weight gain and/or impaired weight maintenance through increased disability and inactivity. Marcus (2004) observed that amongst a sample of chronic pain patients, 63% were found to be overweight or obese. In this sample, the "impact" of pain (as measured by reduced physical functioning and increased frequency of disability) was greater in patients with increased BMI, with the greatest impact noted in obese patients (Marcus, 2004). Alternatively, overweight may increase the risk for pain-related illness as described above through increased load on the joints, lower back, or lower limbs. Coggon and colleagues (2001) attributed 24% of required surgical corrections of osteoarthritis to obesity. Other researchers have postulated that it is the sedentary lifestyle of overweight individuals that may contribute to increased risk of low back pain (Leboeuf-Yde, Kyvik, Bruun, 1999; Shiri, et al., 2010).

While obesity studies typically have not focused on the overt assessment or functional impact of pain in their samples, pain has emerged as important covariate within quality of life data. Several studies incorporating the use of the Medical Outcomes Survey have unanimously found that even mildly elevated BMI is associated with increased bodily pain, and increased BMI and the presence of pain predict impaired quality of life on the physical domains of the survey (Doll, Petersen, & Stewart-Brown, 2000; Fine, Colditz, Coakley, et al., 1999; Yancy, Olsen, Westman, Bostworth, & Edelman, 2002). However, pain itself may take a unique toll on quality of life in obese persons. Fontaine, Barofsky and colleagues (1997, 2001) were the first to suggest that pain contributed independently to decrements in health-related quality of life (HRQL). They found when compared to obese persons not reporting pain, obese persons reporting pain were more significantly impaired on all domains of the Medical Outcomes Survey

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(SF-36), with the strongest effects appearing on the physical components scale. Moreover, these differences held even after controlling for sociodemographic factors, BMI, and depression. In support of these findings, Heo et al. (2003) conducted an analysis of the 1999 Behavioral Risk Factor Surveillance Survey. They concluded that the relationship between high BMI and decreased HRQL was very strongly mediated by joint pain. These two findings suggest that the unique and independent contribution of pain to HRQL in obese persons is an important question to explore.

Mechanisms of the Obesity-Pain Relationship. Although many studies demonstrate a positive association between weight status and pain, investigation of the underlying relationship is lacking. Mechanisms hypothesized for this link include mechanical-structural, metabolic, and behavioral (Janke, et al., 2007; Stone & Broderick, 2012). Given the complex pathophysiology of obesity and pain individually, it is likely that a combination of these factors is responsible for the relationship between the conditions. Mechanical and structural links have been suggested above, with the most discernible of those being excess weight creating excess load onto the body's joints. Chronic pressure on the joints may have further adverse effects on bones, connective tissue, and muscle (Fabris de Souza, Faintuch, Valezi, et al., 2005). Other hypothesized mechanical-structural links include gait disturbance (Cimolin et al., 2011), limited range of spinal movement (Vismara, et al., 2010), and decreased ambulation and conditioning (Ray, et al., 2011; Yamakawa, Tsai, Haig, Miner, Harris, 2004). Physiologic processes involving insulin resistance and inflammation are also hypothesized. Obese individuals have an increased risk for various metabolic disorders, indicating that they may have an increased vulnerability to diabetes-related neuropathic pain (Janke, et al., 2007). Sandell (2009) implicates leptin, a pro-inflammatory hormone released by adipose tissue, which is independently associated with insulin

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resistance and cardiovascular disease. This hypothesis suggests that having excess fat leads to the pathophysiologic processes of inflammation and related pain (Bray & Bellanger, 2006). Behavioral hypotheses suggest that lifestyle and psychosocial factors (e.g., diet, activity level, overall health status, smoking) may indicate shared pathways to both obesity and pain. Pain is also a likely risk factor for weight gain, resulting from reduced activity levels and muscle deconditioning (Janke, et al., 2007).

Pain Beliefs. Considering the IASP definition of pain as a subjective experience, and the reliance on self-report measures in the assessment of pain, it is important to understand the role of cognitive factors in the individual's experience of pain. Pain beliefs are thoughts that influence one's physical, psychological, and behavioral reactions to pain. Catastrophizing is a pain belief that is reported to correlate with self-report measures of pain intensity (Sullivan, Thorn, Haythornthwaite, Keefe, Martin, Bradley, Lefebvre, 2001). It is the tendency of those in pain to have an exaggerated and negative response to pain experiences and pain stimuli (Sullivan, Bishop, & Pivik, 1995; Sullivan, et al., 2001), and the term "catastrophizing" was first used by Albert Ellis, the founder of rational-emotional therapy (Ellis, 1962). Catastrophizing is considered an important antecedent of pain intensity, and has been identified as a marker for particularly poor response to medical treatments (Sullivan et al., 2001; Turner & Aaron, 2001). However, to date, no published study has explored how the presence of catastrophizing may affect individuals' responsiveness to weight loss interventions.

SUMMARY AND RATIONALE

The purpose of the present proposal was to assess the presence of pain, pain-related disability, and pain beliefs in a morbidly obese sample participating in a medically-supervised, nonsurgical weight loss intervention. It is important to research this comorbidity for several reasons. Several studies have demonstrated a positive association between obesity and pain, with increasing BMI related to increasing levels of pain intensity. Although this relationship is observed across the continuum of BMI classification (Stone & Broderick, 2012), individuals classified with extreme obesity are significantly more likely to report moderate to severe pain (Hitt,et al., 2007). Given the commonality of pain in obese populations, particularly those that are seeking weight management (Fontaine, Bartlett, Barofsky, 2000; Heo, Pietrobelli, Wang, Heymsfield, Faith, 2010), it is suggested that greater attention to comorbid pain conditions is warranted.

While available evidence suggests a likely relationship between obesity and pain, there is much to learn about the nature of the association. The comorbidity of physical pain and obesity may act in a reciprocal fashion to increase physical disability. Individuals with extreme obesity, regardless of comorbid diagnoses, have a high level of physical disability (Marcus, 2004). In the present study, it is presumed that the presence of self-reported pain will be related to a greater "impact" on disability, as measured by reduced physical functioning and increased frequency of disability, when compared to extremely obese individuals not reporting pain. This relationship is especially important to understand for participants seeking weight loss treatment, as there is a paucity of research directed toward understanding how pain may increase disability in this unique sample (Heo, et al., 2010). Conversely, the ways in which medical interventions for obesity do or do not assist in the management and impact of pain also remain unknown.

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Although treatment guidelines for pain conditions (i.e., osteoarthritis) recommend weight loss, there is much to understand about the impact on pain over the course of weight loss intervention.

Comorbidity of bodily pain and obesity may have important prognostic implications for obesity treatment, and may directly mediate the effect of weight loss interventions. The presence of pain may contribute unique treatment challenges in both surgical and non-surgical patients. Dixon and colleagues (2001a) demonstrated that in bariatric surgery patients, the presence of preoperative pain predicted less weight loss at one year. In another surgical study, preoperative pain also predicted greater improvement in quality of life indicators post-surgery (Dixon, Dixon, O'Brien, 2001b). Although it appears that those with pain may have the most to gain from surgery, it may be difficult for many to achieve this goal with coexisting pain. Given that the number of surgical procedures performed in the U.S. represents only a small fraction of the population with extreme obesity (Zhao & Encinosa, 2007), continued research of non-operative treatment for this group is necessary.

With respect to non-surgical interventions, pain appears to present a significant treatment obstacle. Evidence from studies conducted with older adults with osteoarthritis has shown strong support for the position that knee pain is an independent mediator for the effectiveness of physical interventions (Rejeski, Ettinger, Martin, Morgan, 1998) and also of diet and exercise interventions (Rejeski, Focht, Meisser, Morgan, Pahor, Pennix, 2002). The Swedish Obese Subjects study found no beneficial effects for their non-surgical weight loss intervention on the impact of pain on work environment (Peltonen, Lindroos, Torgeson, 2003). However, in the SOS study obese persons had more "work-restricting pain" than the general population (Peltonen, et al., 2003), and interference by pain in other domains was not assessed. Further, the SOS study observed that only the surgical obesity treatment reduced the long-term risk of

developing work-restricting musculoskeletal pain, and also increased the likelihood of

recovering from such pain (Peltonen, et al., 2003). As the present study is using an intensive

medical intervention as its active treatment component, it appears particularly important to

understand the potential contributing effects of pain on the efficacy of this type of intervention.

A valuable contribution of this study is to understand how self-reported pain at the outset of

treatment may affect success in a medical intervention for morbid obesity.

Aside from the sole presence of pain, another factor that may disrupt the efficacy of

medical weight loss interventions is pain beliefs. As stated above, there are currently no

published studies that investigate how pain beliefs, specifically catastrophizing, may impact

individuals' responsiveness to weight loss interventions. The present study relied heavily on self-

report measures of participants' pain, thus it was important to control for the potential effects of

cognition on individual's experience of pain within the study.

In the Louisiana Obese Subjects Study (LOSS), patients randomized to the intensive

medical intervention (consisting of group therapy, medication, diet, and toolbox treatments) were

compared with usual care participants, allowing an empirical examination of these questions. If

the presence of pain or pain catastrophizing significantly reduced the efficacy of non-surgical

interventions, this would lend compelling support for the incorporation of empirically supported

pain treatments (such as cognitive behavioral therapy) in obesity rehabilitation programs. Thus,

the following aims were proposed for examination:

Primary Aim

1. The primary aim of this study was to assess how pain may affect the outcome of the intensive

    medical weight loss treatment. It was hypothesized that the presence of pain, pain severity,

and pain interference would predict less weight loss overall and poorer response to treatment one year after receiving the intensive medical treatment.

Secondary Aims

2. A secondary aim of this study was to determine how pain beliefs, specifically pain catastrophizing, may affect treatment outcome. Participants who reported higher levels of pain catastrophizing were expected to have the poorest response to the intensive medical intervention.

3. A basic aim of this study was to evaluate and record the pain that these morbidly obese individuals were experiencing. Pain was hypothesized to be reported within a significant percentage of this sample. Following the direction of previous research, demographic variables and self-reported depression symptomatology were controlled.

4. Another aim of this study was to assess the functional interference of self-reported pain in participants' daily functioning and quality of life. It was hypothesized that when compared to participants not experiencing pain, greater levels of disability would be reported by participants experiencing pain on a measure of health-related quality of life (SF-36).

METHOD

Participants

All participants for the current project were recruited as part of a study entitled "Louisiana Obese Subjects Study (LOSS): Pragmatic Clinical Evaluation of Treatments for Severe Obesity for State Group Benefits Members in Louisiana," funded by the Louisiana Office of Group Benefits (OGB). Participants of the current study included 390 morbidly obese individuals who qualified for state medical insurance benefits through OGB at the time of screening and randomization. Participants were initially seen at one of eight clinical sites in Louisiana. Males and non-pregnant females aged 20-60 years, with BMI $\geq$ 40 kg/m^2 but $\leq$ 60 kg/m^2 were recruited for the parent study. Participants were excluded for factors that might limit adherence to interventions or affect the conduct of the trial, including but not limited to: recent hospitalization for psychiatric illness or substance abuse, history of eating disorder, current pregnancy or nursing, prior bariatric surgery, or bowel resection. Diagnosis or current treatment for several medical disorders (e.g., HIV, Hepatitis C, tuberculosis, severe congestive heart failure, cancer) or mental illness (e.g., psychotic or bipolar disorders or current major depressive episode) that would make an individual unsuitable for an intensive medical weight loss intervention were also basis for exclusion. To maximize high participant retention across study sites, the following methods were employed: use of incentive programs, supplemental materials, personal contact by study staff, mailed reminders, motivational interviewing, participant goal setting, and problem solving/overcoming barriers. Incentive programs included free medical screening and store and gas gift cards for attendance at follow-up study visits.

Measures

   Medical Outcomes Survey (SF-36). The Medical Outcomes Study Short Form Health Survey (SF-36; Ware, Snow, Kosinski, Gandek, 1993) is a self-administered questionnaire that assesses eight domains of functioning: physical functioning, role limitations due to physical problems, vitality, bodily pain, social functioning, role limitations due to emotional problems, mental health, and general health perception. The eight scales have also been shown to form two distinct higher-order clusters due to the physical and mental health variance they have in common. Factor analytic studies have confirmed physical and mental health factors that account for 80-85% of the reliable variance in the eight scales (Ware, et al., 1993). Three scales (physical functioning, role limitations due to physical problems, and bodily pain) correlate the most highly with the physical component and contribute to the scoring of the Physical Component Summary measure. The mental health, role limitations due to emotional problems, and social functioning scales contribute to the scoring of the Mental Component Summary measure (Ware, Kosinski, Keller, 1994). The other two scales have noteworthy correlations with both component measures. The SF-36 possesses sound psychometric properties (Stewart & Hays, 1992) and has been used in a variety of health care contexts to assess health-related quality of life across the multiple domains described above (Ware, 2000; Ware & Kosinski, 2001).

   Due to the extensive use of the SF-36, research has demonstrated that the scales on the Physical Component Summary measure are most responsive to treatments that change physical morbidity (Ware, 2000; Ware & Kosinski, 2001), including studies of weight loss treatments (Kolotkin, et al., 2009). The present study applied the principles of other weight loss treatment studies and limited the factors of health-related quality of life explored here (i.e., severe comorbid illness, depression). Furthermore, the present study aimed to examine those domains

most relevant to pain-related quality of life, specifically: physical functioning, role limitations due to physical problems, and bodily pain (Ware & Kosinski, 2001). Therefore, the Physical Component Summary measure provided the best estimate of functional health and disability for the present study.

Brief Pain Inventory (BPI). The BPI, in the short form (Cleeland, 1989; Keller, Bann, Dodd, Schien, Mendoza, Cleeland, 2004), is a nine-item self-report measure. The BPI is a condition-specific quality of life measure that provides detailed information about the impact of pain on daily functioning in several domains, including occupational, interpersonal, and emotional realms. The BPI was developed to provide a quick and efficient means of measuring pain presence, location, intensity, and the extent to which pain interferes in the lives of pain sufferers (Keller et al., 2004). Using a 1-10 scale, respondents rate their worst, least, average, and current pain intensity, and also rate the degree to which pain interferes with seven domains of functioning (general activity, mood, walking ability, normal work, relations with others, sleep and enjoyment of life). The BPI can be computed to generate two subscale scores: Pain Severity and Pain Interference (Cleeland, 1989; Keller, et al., 2004). The BPI has been used extensively in cancer pain populations, and has been translated into many different languages and is used throughout the world. The BPI was also validated in two chronic, nonmalignant pain populations (Keller et al., 2004; Tan, Jensen, Thornby, Shanti, 2004). Internal consistency is acceptable, with an $\propto$ coefficient of .85 for the intensity scale and .88 for the interference scale. The BPI has also been noted to have good convergent validity with other general measures of health related quality of life, such as the bodily pain and physical role functioning scales of the SF-36 (Keller, et al., 2004). Thus, the BPI is a reliable, valid, and sensitive to change instrument for assessing

pain intensity and interference in patients with nonmalignant pain and for the general population who may have chronic intractable pain.

Pain Catastrophizing Scale (PCS). The PCS is a thirteen-item self-report instrument that describes different thoughts and feelings individuals may experience when they are in pain. The PCS asks participants about the extent to which they ruminate, magnify, and experience helplessness with regard to their pain symptoms. Participants reflect on each of the questions and respond on five-point scales with the endpoints of (0) "not at all" to (4) "all of the time." Examples of statements that participants are asked to rate are, "My pain is awful and I feel it overwhelms me" and "When I am in pain I feel I can't go on." The PCS yields three subscale scores of rumination, magnification, and helplessness, as well as a total score. The PCS has been shown to have adequate to excellent internal consistency with $\propto$ coefficients of: rumination = 0.87, magnification = 0.66, helplessness = 0.78 and Total PCS = 0.87 (Sullivan, Bishop, & Pivik, 1995). The PCS has also been used extensively in both chronic and experimental pain populations (Osman, Barrios, Kopper, Hauptmann, Jones, O'Neil, 1997), and has been translated into several languages (Turner & Aaron, 2001).

Center for Epidemiological Study and Depression Scale (CES-D). Developed by Radloff (1977), the CES-D is a 20-item self-report measure of depressive symptomatology. Respondents rate the frequency of occurrence in the past week of each symptom on a four-point scale ranging from "rarely or none of the time (less than 1 day)" to "most of the time or all of the time (5-7 days)." On the CES-D, scores range from 0 to 60, with higher scores indicating more symptoms of depression. CES-D scores of 16 to 26 indicate mild depression and scores of 27 or greater may indicate major depression (Martens, et al., 2006; Radloff, 1977). A score of "16" or greater indicates increased risk for the diagnosis of depression (Radloff, 1977). The CES-D has

been extensively used in primary care and community based studies. In studies of depression prevalence among primary care and arthritic pain patients, a score of "19" has been suggested as a more optimal sensitivity point to avoid false-positive identification of depression (Martens, Parker, Smarr, Hewett, Ge, Slaughter, Walker, 2006). However, in the present study, a score of "16" required that a clinical psychologist review the participant's medical and psychiatric history to give approval for study entry, as a more stringent control of the study's exclusion criteria.

Body Weight. The formula for calculation of BMI = weight [kg] divided by height [m^2] (WHO, 2011). All weight information provided in this study is reported in kilograms. Height and weight were measured via standardized protocol using annually calibrated measuring devices and scales at each clinical site.

Procedure

Overall LOSS Design. The primary aim of the LOSS study was to observe the effect of an intensive medical management program versus usual care for class III obesity (BMI $\geq$ 40kg/m^2) on weight loss, total medical costs and on measures of health risks associated with weight loss. LOSS was structured as a pragmatic clinical trial (PCT; Tunis, Stryer, & Clancy, 2003). As such, the interventions chosen were designed to mimic medical practice patterns of care. LOSS was characterized by three major study periods: screening and randomization, active weight loss in years 1-3, and observation in years 4-5 (see Figure 1). All individuals with medical insurance supplied by OGB were invited to participate in informational sessions. Screening and randomization was conducted across two visits. Randomization employed stratification for sex, BMI, and age, and was designed to yield comparable samples with regard to baseline prognostic factors. Screening, randomization, and the observation period in years 4-5 were structured similarly for all participants, regardless of group assignment.

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                    ┌─────────────────────────────────┐
                    │     597 Attended screening      │
                    │ visits at eight clinical sites  │
                    │ (Alexandria, Baton Rouge,       │
                    │ Hammond, Lafayette, Lake         │
                    │ Charles, Monroe, New Orleans,    │
                    │ and Shreveport)                  │
                    └─────────────────────────────────┘
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597 Attended screening visits at eight clinical sites (Alexandria, Baton Rouge, Hammond, Lafayette, Lake Charles, Monroe, New Orleans, and Shreveport)

132 Excluded; Did not meet entry criteria

465 Randomized

75 Excluded; 3 Had weight loss surgery 72 New Orleans site post-Katrina and Monroe site

200 Intensive Medical Intervention

190 Usual Care

Figure 1. Louisiana Obese Subjects Study Recruitment and Retention

Participants agreed to randomization to one of two treatment conditions in years 1-3 of the study: usual care condition or intensive medical approaches. The usual care group received initial instruction in the use of the Mayo Clinic Weight Management website but otherwise received usual care from their primary care provider. For study purposes, usual care participants were also monitored annually with a health screening examination at the clinical site. The active weight loss period for the intensive medical management condition was structured into three phases. Phase 1 had a duration of approximately three months, during which participants

followed a prescribed very low calorie diet (VLCD). Following the initial VLCD phase, Phase 2 of the intensive medical intervention utilized a highly structured diet, and introduced weight loss medication and regular behavioral therapy schedule that spanned four months. Phase 3 began in month 8 and lasted to month 36. Phase 3 began the maintenance program which was instituted to maintain weight loss over the long term.

Clinical Sites. LOSS was conducted at seven of the eight original clinical sites in the state of Louisiana (Baton Rouge, Lafayette, Lake Charles, Alexandria, Monroe, Hammond, and Shreveport). As the LOSS study began recruitment and screening in summer 2005, the New Orleans site was directly impacted by Hurricane Katrina. The remaining clinical sites absorbed most of the New Orleans-based participant base for continued study involvement. However, the participants from the New Orleans site were excluded from study analysis due to catastrophic exposure and life change that could not be accounted for in the context of the present study. Participants from the Monroe site were also excluded due to problems with adherence to study protocol by the clinical site staff (e.g., failure to schedule study visits). Lastly, three participants were excluded because they underwent weight loss surgery during the study period on their own, independent from the LOSS study and OGB (see Figure 1).

Current Study. Treatment outcome will be operationalized as percentage change of initial body weight (IBW), per IOM standards for clinically significant weight loss in non-operative treatment studies. Thus, the primary endpoint of the current study was the same as that of the original LOSS study. The current study aimed to capture the impact of self-reported pain, pain intensity, pain severity, and pain catastrophizing on treatment outcome. Measurement of the above constructs occurred at baseline/ randomization and month 12 of the LOSS study for all participants.

Power Analyses

Power analyses were conducted for the parent study, and given that both the parent and the current studies use the same primary endpoint, these calculations are applicable here (Ryan, et al., 2010). Power analyses were conducted under the usual assumptions of ANOVA, and with a type 1 error rate of 5%, or alpha set at 0.05, 2-tailed for all analyses. For the original LOSS study, two hypotheses were addressed: 1) that weight loss for the intervention group exceeded 20% of initial body weight, and 2) that weight loss for the intervention group exceeded that of the usual care group. As such, power calculations were carried out for weight change from baseline in kilograms to the end of the intervention period and to the end of the study. The estimate of standard deviation for weight loss employed in the power calculations was taken to be 16 kilograms. This value, an estimate provided by the authors of the Swedish Obese Subjects study (Sjöström, 2000; Sjöström, Lissner, Wedel, Sjöström, 1999), was chosen because it produced a highly conservative approach to power analyses. With sample size of 240, a difference in weight loss (Intensive Medical Intervention - Usual Care) of as little as 3.46 kg would be required for 80% power for the test of treatment effect. Allowing for a 25% dropout rate, the parent LOSS study calculated that sample size of 180 required a minimum difference between groups (Intensive Medical Intervention - Usual Care) of 4.2 kg for 80% power.

RESULTS

Sample Characteristics and Descriptive Analyses

Baseline characteristics of the 390 randomized participants are depicted in Table 1. The mean age of the population was 47 years, and they were predominantly white (75%) and female (83%). At randomization, the coordinating center randomly assigned participants to intensive medical intervention (IMI) or usual care (UC) by applying minimization allocation with stratification by age, sex, and BMI to achieve allocations that were comparable with specified baseline prognostic factors. For stratification, age and BMI were dichotomized as follows: age (in years) 20 or older but younger than 40, or 40 or older, up to and including 60; and BMI of 40 or higher but less than 45, or 45 and higher, up to and including 60. As expected, there were no group differences in age, sex, BMI, and race.

Table 1

Baseline Characteristics of Participants

	IMI (n = 200)	UC (n = 190)
Age, mean (SE), in years	47.2 (0.6)	47.1 (0.6)
Male, total number (%)	33 (16.5)	31 (16.3)
White	29 (14.5)	26 (13.7)
Black	4 (2.0)	5 (2.2)
Female, total number (%)	167 (83.5)	159 (83.7)
White	120 (60.0)	121 (63.7)
Black	46 (23.0)	38 (20.0)
Hispanic	1 (0.5)	0 (0.0)
Weight, median (IQR), kg	126.2 (23.2)	128.4 (28.6)
BMI, median (IQR)	45.6 (7.9)	46.6 (8.5)
Male	44.9 (7.2)	46.4 (9.6)
Female	45.7 (8.0)	46.8 (8.4)

IQR = interquartile range

Common comorbid medical conditions to obesity reported at screening are reported in Table 2. Occurrence of congestive heart failure and coronary artery disease is relatively low, as severe congestive heart failure and the occurrence of a cardiovascular event in the past year prior

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to screening were exclusion criteria for LOSS. Hypertension (84.6%) was the comorbid condition reported by the most participants. Pre-existing pain conditions (e.g., fibromyalgia, neuropathy, osteoarthritis) were not recorded consistently at the screening visit to provide estimates for this sample. A basic aim of the study was to evaluate and record the pain reported by participants. At the time of randomization, scores on the BPI indicated that 36.7% of the sample (N=378) reported the presence of "pain today." This was a direct response to the question of whether the participant was experiencing pain that was beyond 'everyday' pain on that day. By gender, 39% of males and 37% of females reported pain today (see Table 2). BPI at randomization was not available for twelve participants. An independent sample t-test was conducted to compare pain today and completer status. There was no a significant group difference (completers versus non-completers) in self-reported presence of pain (t (376) = -.55, p=.58).

Table 2

Health-Related Sample Characteristics

	N	%
Congestive Heart Failure	16	4.1
Coronary Artery Disease	30	7.7
Diabetes Mellitus	80	20.5
Insulin use	21	5.4
Hypertension	330	84.6
Hyperlipidemia	125	32.1
Pain Today	143	36.7
Males	25	39
Females	120	36.8

Due to the potential adverse impact on compliance, the current study made extra efforts to control for depression (Wright, et al., 2010), as the case of any randomized participant with CES-D scores at 16 or greater was reviewed by a clinical psychologist. Recall that on the CES-

D, a score of 16 or greater indicates an increased risk for the diagnosis of depression, although minimal impact (Martens, et al., 2006; Radloff, 1977). Of the 390 participants who completed the CES-D at randomization, only eleven participants (2.8%) had scores greater than 16, and of these, three (0.7%) were greater than 27. Scores of 27 or greater may indicate major depression (Martens, et al., 2006). The highest CES-D score reported was 32. A review of study records indicated that protocol was followed for these participants, whose charts were reviewed.

Correlations of Predictor and Criterion Variables

Preliminary analyses were conducted to determine if there were intercorrelations amongst the set of predictors (i.e., the Physical Component Summary subscale of the SF-36, BPI pain today, BPI Pain Severity, BPI Pain Interference, and PCS) and depression (CES-D). High correlations ($r \geq 0.7$) amongst the predictors could indicate common underlying factor(s). Pearson r correlations are displayed in Table 3. Many scales were moderately correlated with each other. As expected, both Pain Severity and Pain Interference from the BPI were moderately correlated with the SF-36 Physical Components Summary. The BPI has been noted to have good convergent validity with other general measures of health-related quality of life, such the SF-36 (Keller, et al., 2004). Pain Interference and Severity were highly correlated with one another ($r = 0.78, p < .001$).

Intercorrelations of the predictor and criterion variables with age, sex, BMI, and depression were also computed prior to regression calculations, as displayed in Table 4. A modest negative correlation was found between age and pain today. Depression was significantly correlated with Pain Severity, Pain Interference, and Pain Catastrophizing. Lastly, age, BMI, and depression were negatively all correlated to the Physical Components Summary of the SF-36.

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Table 3

Correlations of Predictor and Criterion Variables

	Physical Components Summary	Pain Today	Pain Severity	Pain Interference	Pain Catastrophizing
Percent Change of Initial Body Weight	-.13	-.14*	.18*	.18*	.04
Physical Components Summary		.33**	-.52**	-.65**	-.34**
Pain Today			-.40**	-.33**	-.15**
Pain Severity				.78**	.38**
Pain Interference					.48**

** Correlation is significant at the 0.01 level (2-tailed).
* Correlation is significant at the 0.05 level (2-tailed).

Table 4

Correlations of Predictor and Criterion Variables with Demographic Indices, BMI at Randomization, and Depression

	Age	Race	Sex	BMI	Depression
Pain Today	-.12*	.06	.03	-.10	.04
Pain Severity	.01	.08	.02	.03	.26**
Pain Interference	.05	.01	.03	.11	.32**
Pain Catastrophizing Total	.07	.05	.05	.13	.42**
Percent Change of Initial Body Weight	-.02	.08	.07	-.02	.08
Physical Components Summary	-.24**	.05	-.11	-.28**	-.20**

** Correlation is significant at the 0.01 level (2-tailed).
* Correlation is significant at the 0.05 level (2-tailed).

Missing Data

Missing data were deleted listwise such that these regression analyses represent a completers analysis of all participants for whom weights were available at Year 1. A rigorous missing data statistical plan was proposed, with different mechanisms that led to missing data analyzed. Sensitivity of the different statistical methods (completers analysis, baseline observation carried forward analysis, last observation carried forward) were verified to be robust in the original LOSS dataset (Ryan, et al., 2010). Furthermore, the completers analysis was used without significantly reducing variance or power, which is a common concern for partially missing data (Tabachnick & Fidell, 2007). There were no differences in age, sex, BMI, or race observed in those individuals who completed the Year 1 measurement (N = 208; 53% of baseline sample) and those who did not.

Primary Aim

The primary aim of this study was to assess the extent to which self-reported pain at the outset of treatment may affect the success of the intensive medical weight loss treatment. The literature does not indicate an empirical basis for which the presence of pain, pain severity, or pain interference should predict overall weight loss. As reported in the main outcome results of the parent LOSS study, treatment condition was associated with significant differences at Year 1 (Ryan, et al., 2010). Specifically, at Year 1 among the 60% of attending IMI participants, the mean weight loss was 13.9%, yet only 0.9% for the 50% of attending UC participants. To control for the main effect of treatment condition, group assignment was entered into the regression equation first. Since the different components of the BPI were highly correlated, they were entered in a stepwise fashion in order to avoid problems of multi-collinearity in the regression equations. Hierarchical regression analyses were performed to further analyze the strength of the

associations between percentage change of IBW and self-reported pain. After control of treatment condition, the following variables from the BPI were entered into the regression equation: pain today, pain severity, and pain interference.

Results of the regression for the prediction of percent change of IBW are summarized in Table 5. As consistent with previous studies, the independent impact of depression was controlled for and entered into Step 1 of the analysis. It did not contribute significant variance into the model. As expected, R for regression was significantly different from zero for treatment condition [$F(2,206) = 27.71$, $p < .001$] in Step 2. Inspection of R^2 indicates that treatment condition predicted approximately 24% of the variability within treatment outcome ($R^2 = .24$, adjusted $R^2 = .24$). In Step 3, the pain indices were allowed to compete for entry in the next step of the analysis. Pain severity was the only variable to meet minimum criteria for entry [$F(3,205) = 20.50$, $p < .001$], such that increasing levels of pain severity were associated with poorer treatment outcome. The final model predicted 29% of the variance in treatment outcome, with pain severity accounting for an incremental 5% of the variance in percent change of IBW.

Table 5

Hierarchical Regression Analyses Predicting Percent Change of Initial Body Weight

	R^2	β	p
Step 1	.01		
Depression		.08	.29
Step 2	.24**		
Treatment Condition		-.49**	.00
Step 3	.29**		
Pain Today		.06	.37
Pain Severity		.15**	.01
Pain Interference		.05	.58

Secondary Aims

A secondary aim of this study was to determine how pain beliefs, specifically pain

catastrophizing, may affect treatment outcome. Participants who reported higher levels of pain

catastrophizing were expected to have the poorest response to the intensive medical intervention.

The authors of the Pain Catastrophizing Scale define a cut-off score for clinically relevant levels

of catastrophizing as a total PCS score of 30 (Sullivan, 2009). This score corresponds to the 75th

percentile of the distribution of PCS scores in clinic samples of chronic pain patients.

Unfortunately, the PCS was only administered to IMI participants at their randomization visit, so

limited data are available for this measure (N = 193). Among this group, levels of pain

catastrophizing were very low, with only five participants (2.5%) reporting PCS Total score $\geq$

30. Ninety-three percent of the group who completed the PCS had scores lower than 20 (50%ile).

Thus, no further analysis of pain beliefs was available.

Another aim of this study was to assess the functional interference of self-reported pain in

participants' quality of life. It was hypothesized that when compared to participants not

experiencing pain, greater levels of disability would be reported by participants experiencing

pain on a measure of health-related quality of life (SF-36). Self-reported pain for this research

question was operationalized using scores on the BPI at randomization to indicate if patients

reported pain on that day. BPI Pain Severity and Pain Interference scores were not used, as the

correlations with the dependent variable (i.e, Physical Component Summary score of the SF-36

at randomization) were too high and multi-collinearity was again a concern. Simultaneous

regression was used, with the sample subset of completers analyzed as in the regression for the

primary research aim. Age, BMI, and depression were controlled for in this model, as these

variables were reported above to have significant correlations with the dependent variable. *R* for

regression was significantly different from zero for the presence of pain [$F(4,204) = 14.61$, $p <$ .001]. Inspection of R^2 indicates that the self-reported presence of pain predicted approximately 11% of the variability within health-related quality of life ($R^2 = .11$, adjusted $R^2 = .11$). The effect size is large by Cohen's d standards ($r = .51$).

DISCUSSION

The present study was the first to assess the presence of pain, pain-related disability, and pain beliefs in the context of a pragmatic clinical trial to mimic medical practice patterns of care for the treatment of obesity. Taken as a whole, results indicate that pain and pain severity are significantly associated with weight loss treatment outcome after one year of intervention. Further, the presence of pain is independently associated with decrements in health-related quality of life, after controlling for age, BMI, and depression. The primary aim of the current study was to assess the extent to which self-reported pain would affect the success of the intensive medical weight loss treatment. Pain severity at the outset of treatment was significantly associated with greater percentage change in initial body weight, even after controlling for the impact of depression and treatment condition. The direction of the relationship was such that increasing levels of pain severity at the outset of treatment was significantly associated with poorer treatment outcome in both groups. The same relationship was not observed for pain interference or the mere presence of self-reported pain. The findings of the present study provide preliminary evidence that pain severity is an important indicator of treatment outcomes for extremely obese individuals trying to lose weight by nonsurgical methods.

In the present sample, pain was reported by over one-third of participants. This was operationalized as self-reported pain that was beyond 'everyday' pain on a single day. Participants provided a binary response. This assessment method was similar to that of The Gallup Organization (Stone & Broderick, 2012). Their survey of pain that occurred yesterday as a "yes" or "no" response was applied to reduce recall biases associated with the longer reporting periods (up to three months) often used in pain assessment (Wright, et al., 2010). However, this is also a consideration for the interpretation of these results, as there is a trade-off in minimizing

recall bias with the depth of measurement beyond one day. Pain reported in this manner may not capture the typical pain experience for an individual. The frequency of pain in this sample was also lower than expected. Previous research has reported frequencies of comorbid pain ranging from 56 to 68% in samples of weight treatment seeking, obese adults (Fontaine, et al, 2000; Fontaine & Barofsky, 2001; Peltonen, Lindroos, Torgeson, 2003). Although among non-treatment-seeking, but similarly obese counterparts, pain has been observed in approximately one-third of participants (Fontaine, et al., 2000; Wright, et al., 2010). One reason for this relatively low finding may be the highly controlled sample. Specifically, prior to randomization, careful attention was made to control for demographic variables and depression, known factors to be associated with higher levels of self-reported pain. This may be a more accurate and conservative frequency estimate of pain, as known correlates have not only been statistically controlled, but excluded from this sample.

The present sample was also highly controlled for BMI, in that the study focused on morbid, class III obesity. The estimates provided above are for treatment-seeking adults from a range of BMI classes. The present study would not suggest that morbidly obese individuals have less pain than with lesser degrees of excess weight. Instead, for individuals at such high BMI, duration and chronicity of pain and comorbid health conditions may provide contextual evidence for this reporting. Morbid obesity is typically associated with serious health complications and conditions, many of which are associated with and contribute to pain. Morbid obesity is also not a quickly resolving condition, thus individuals are experiencing both excess weight and related consequences for a number of years. Sullivan and colleagues (2002) reported that the number of years since the onset of pain (i.e., pain chronicity) may significantly affect individuals' reporting of pain, as well as moderate the relationship between cognitive appraisal of pain and pain-related

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disability. The longer an individual experiences pain conditions, the lower their expectancies for symptom resolution and treatment outcome. Chronic pain conditions are often characterized by repeated treatment failures and exhaustion of treatment outcomes, and the appraisal of 'common' pain changes (Sullivan, et al., 2002). The participants in this sample were specifically asked about pain that was in excess of "everyday" pain, which may have underestimated the degree of pain they were experiencing.

The contribution of pain beliefs, specifically catastrophizing, to treatment outcome was not understood in the present study. Participants who reported higher levels of pain catastrophizing were expected to have the poorest response to the intensive medical intervention. However, problems with questionnaire administration led to failure of the construct to be assessed in the entire sample. When analyzed for score ranges, levels of total pain catastrophizing were very low, so as to preclude further analysis of the impact of pain beliefs. This may again be a function of the control for depressive symptomatology in the sample. The construct of catastrophizing was used by Beck (1976) to describe a 'cognitive distortion' that could contribute to the development or exacerbation of depressive and anxious symptoms. Literature suggests that the essential features of catastrophizing in depression and anxiety may be similar to those of catastrophizing as discussed in the pain literature (Turner & Aaron, 2001). The lack of catastrophizing beliefs about pain in this sample likely suggests this hypothesis is true. Furthermore, as suggested by Sullivan and colleagues (2002), there is also evidence that the psychological correlates of pain-related disability change over time. They reported that the stage of pain chronicity also moderated the relationship between PCS subscales and pain-related disability, such that helplessness (but not magnification or rumination) was seen more in chronic

pain patients. With a more diverse sample of weight loss treatment seeking adults, this relationship could be explored further.

The presence of pain in the current sample was independently associated with decrements in health-related quality of life, after controlling for age, BMI, and depression. With large effect size, this finding is especially robust, considering that only one-third of the sample reported pain today on the BPI, below the prevalence suggested in the literature. Research has hypothesized that the comorbidity of physical pain and obesity may act in a reciprocal fashion to increase physical disability, and this further indicates that pain may independently decrease quality of life and increase disability for morbidly obese patients. This finding provides evidence for the importance of assessing pain in obese patients, particularly those seeking treatment for weight loss. For morbidly obese adults with multiple chronic health conditions, the assessment of both pain presence and comorbid pain-related conditions is important so that researchers may control for the contribution of comorbidities to both pain and quality of life.

One proposed mechanism of the reciprocal obesity-pain relationship on health-related quality of life is the negative impact of sedentary behavior on both conditions. That is, obesity may increase the risk for pain-related illness through increased load on the body. Yet, the sedentary lifestyle of overweight individuals may contribute to increased risk of pain conditions (Leboeuf-Yde, et al., 1999; Shiri, et al., 2010). While exercise is an important part of multidisciplinary treatment of both chronic pain and obesity, there is reduced exercise capacity in obese women (Hulens, Vansant, Lysens, Claessens, Muls, 2001), due to complaints of musculoskeletal pain. Therefore, alternative exercise programs in conjunction with weight reduction may be necessary for obese chronic pain patients.

Limitations and Future Directions

Interpretation of these data is limited by several factors. First, although there were no significant group differences found between individuals who did and did not complete the year 1 weight measurement, the retention rate was lower than anticipated in the parent study's power calculations. The issue of poor retention in weight loss studies is well known, and it takes special efforts to produce excellent retention rates such as in the Diabetes Prevention Program and the Look AHEAD trials (Pi-Sunyer, et al., 2007). Participant retention in both 1- and 2- year weight loss studies, especially medication-based programs, is approximately 50%, similar to that of the current study (Hauptman, et al., 2000; Pi-Sunyer, et al., 2007; Sjöström, et al.,1999). Ryan and colleagues (2010) suggested that it may be expected for primary care physicians to observe that only half of their patients seeking weight loss intervention may remain in the program. However, for those who continue treatment, weight loss is achieved. Although the retention in the current study may mimic real-world weight loss behavior, it was less than desired (Simmons-Morton, Obarzanek, & Cutler, 2006).

The generalizability of these findings is also affected by the limitations of the study design, particularly the use of self-report measures, the small homogeneous sample, and lack of comparison measures between treatment conditions at more than two time points. Pain, pain catastrophizing, and pain-related disability were measured with self-report questionnaires. Assessment by this method must be cautious given that self-reporting can be influenced by numerous factors including cognitive abilities, mood, sleep disturbance, and medication usage (Peter & Watt-Watson, 2002). In the current study, attempts were made to control for depressive symptoms, which were found not to contribute to the treatment outcome. Self-report methods are inherent to pain assessment (Greve, Bianchini, Ord, 2012); however, more objective observance

of pain-related behavior would add value to the understanding of pain in this population. Specifically, future studies should make provisions to more closely monitor medication usage over the course of the study. Thus, the use of self-report exclusively for these behaviors requires that the current findings be considered with caution.

The sample was homogeneous as it consisted only of morbidly obese adults seeking treatment for weight loss, from a recruitment pool of state employees and dependents who qualified for state medical insurance benefits. The sample was predominantly female and white. Given the restriction by participant BMI, generalizability of the current results are limited in that they cannot be applied across the larger spectrum of overweight and obesity. Extreme or morbid obesity is typically associated with more serious health complications than lesser degrees of excess weight. Although this sample has a high proportion of comorbid chronic illnesses, it was not accessed to which illnesses pain may be attributed. Recruitment was also limited to individuals who are state employees or their dependents. Results may have differed if participants from a broader cross-section of the state population were recruited. Yet, the present study had the distinct advantage of representation from multiple areas of the state.

The research generally accepts weight losses of 5% or more as associated with improved health outcomes (Sjöström, et al.,1999). The present study was not designed to test the effect of sustained modest weight loss in extreme obesity, particularly on its impact towards pain. Recommendations for some pain conditions (i.e., osteoarthritis) include weight loss in pain treatment. Even modest increases in BMI are related to the development and progression of pain disorders (Holmberg, Thelin, & Thelin, 2005); reciprocally, decreases in weight may impact pain. Thus, it would be expected that weight loss over the course of the medical intervention would impact participants' reported pain positively. Future studies of larger weight-loss-seeking

samples should focus assessment of pain, related pain conditions, and pain medication usage to understand better the impact of incremental weight loss on pain outcomes. Furthermore, the current study did not evaluate a causal relationship between pain and obesity. There was no identification of relationship between development of obesity before or after occurrence of pain symptoms to suggest if obesity occurred before or after, possibly as a consequence, of pain symptoms. If a causal relationship can be identified, then studies could be developed to evaluate the benefits of weight reduction as a treatment for pain, with measurements of depression, disability, and quality of life as outcome measures.

Conclusions

Despite these limitations, the findings of the present study provide preliminary evidence that pain severity is an important indicator of treatment outcomes for extremely obese individuals trying to lose weight by non-surgical methods. There is still much to learn about the co-occurrence of pain and obesity. Continued research is necessary, applying rigorous scientific methodology to investigate the causal sequence and interaction of these variables, and to expand understanding of the reciprocal relationship. Further examination of subsequent effects on health-related outcomes and medication usage would be beneficial. Explanatory models need to be developed and treatments tested to most effectively target this comorbidity and meet the needs of individuals struggling with both excess weight and pain.

REFERENCES

American College of Rheumatology Subcommittee on Osteoarthritis. Recommendations for the medical management of osteoarthritis of the hip and knee: 2000 update. *Arthritis & Rheumatism, 43*, 1905-1915.

Anderson, J.J. & Felson, D.T. (1988). Factors associated with osteoarthritis of the knee in the first national Health and Nutrition Examination Survey (HANES I): evidence for an association with overweight, race, and physical demands of work. *American Journal of Epidemiology, 128*, 179-189.

Barofsky, I., Fontaine, K.R., & Cheskin, L.J. (1997). Pain in the obese: impact on health related quality of life. *Annals of Behavioral Medicine, 19,* 408-410. doi: 10.1007/BF02895160.

Beck, A.T. (1976). *Cognitive therapy and the emotional disorders.* New York, NY: International Universities Press.

Berry P.H., & Dahl, J.L. (2000). Making pain assessment and management a healthcare system priority through the new JCAHO plan. *Journal of Pharmaceutical Care in Pain & Symptom Control, 8,* 5-20. doi: 10.1300/j088v08n02_02.

Board on Health Sciences Policy. (2011). Relieving pain in America: A blueprint for transforming prevention, care, education, and research. Accessed from the Institute of Medicine, http://books.nap.edu/openbook.php?record_id=13172

Bray, G.A. (2004). Medical consequences of obesity. *Journal of Clinical Endocrinology and Metabolism, 80,* 2583-2589. doi: 10.1210/jc.2004-0535.

Bray, G.A. (2007). The metabolic syndrome and obesity. Humana Press. doi: 10.1007/978-1-59745-431-5.

Bray, G.A. & Bellanger, T. (2006). Epidemiology, trends, and morbidities of obesity and the metabolic syndrome. *Endocrine, 29*, 109-117. doi: 10.1385/ENDO:29:1:109.

Brooks, P.M. (2006). The burden of musculoskeletal disease – a global perspective. *Clinical Rheumatology, 25*, 778-781. doi: 10.1007/s10067-006-0240-3.

Brown, W.J., Mishra, G., Kenardy, J., et al. (2000). Relationships between body mass index and well being in young Australian women. *International Journal Obesity Related Metabolic Disorders 24,*1360-1368. doi: 10.1038/sj.ijo.0801384.

Caro, J.F. & Considine, R.V. (2004). Leptin: from laboratory to clinic. In: Bray, G.A., Bouchard, C., & James, W.P. (Eds.) *Handbook of obesity: etiology and pathophysiology* (pp. 275-296). 2nd ed. New York, NY: Marcel Dekker.

Centers for Disease Control and Prevention. (2010). Vital signs: state-specific obesity prevalence among adults – United States, 2009. *Morbidity and Mortality Weekly Report, 59,* 951-955.

Cimolin, V., Vismara, L., Galli, M., Zaina, F., Negrini, S., & Capodaglio, P. (2011). Effects of obesity and chronic low back pain on gait. *Journal of NeuroEngineering and Rehabilitation, 8.* doi: 10.1186/1743-0003-8-55.

Cleeland, C.S. (1989). Measurement of pain by subject report. In C.R. Chapman & J.D. Loeser (Eds.): *Advances in pain research and management* (pp. 391-403). New York, NY: Raven Press.

Coggon, D., Reading, I., Croft, P., McLaren, M., Barrett, D., & Cooper, C. (2001). Knee osteoarthritis and obesity. *International Journal of Obesity and Related Metabolic Disorders, 25,* 622-627. doi: 10.1038/sj.ijo.0801585.

Crawford, A.G., Cote, C., Couto, J., Daskiran, M., Gunnarsson, C., Haas, K., ... Rob, S. (2010). Prevalence of obesity, type II diabetes mellitus, hyperlipidemia, and hypertension in the United States: findings from the GE Centricity electronic medical record database. *Population Health Management, 13,* 151-161. doi:10.1089/pop.2009.0039.

Deitel, M. (2003). Overweight and obesity worldwide now estimated to involve 1.7 billion people. *Obesity Surgery, 13,* 329-330. doi: 10.1381/096089203765887598.

Dixon, J.B., Dixon, M.E., & O'Brien, P.E. (2001a). Preoperative predictors of weight-loss at 1-year after lap band surgery. *Obesity Surgery, 11,* 200-207. doi: 10.1381/096089201321577884.

Dixon, J.B., Dixon, M.E., & O'Brien, P.E. (2001b). Quality of life after lap-band placement: influence of time, weight Loss, and comorbidities. *Obesity Research, 9,* 713-721. doi: 10.1038/oby.2001.96.

Doll, H.A., Petersen, S.E.K., & Stewart-Brown, S.L. (2000). Obesity and physical and emotional well-being: associations between body mass index, chronic illness, and the physical and mental components of the SF-36 questionnaire. *Obesity Research, 8,* 160-170. doi: 10.1038/oby.2000.17.

Ellis, A. (1962). *Reason and emotion in psychotherapy*. New York, NY: Lyle Stuart.

Fabris de Souza, S.A., Faintuch, J., Valezi, A.C., Sant'Anna, A.F., Gama-Rodrigues, J.J, De Batitsta Fonseca, I.C., & De Melo, R.D. (2005). Postural changes in morbidly obese patients. *Obesity Surgery, 15*, 1013-1016. doi: 10.1381/0960892054621224.

Felson, D.T. (1990). The epidemiology of knee osteoarthritis: results from the Framingham Osteoarthritis Study. *Seminars in Arthritis and Rheumatism, 20*, 42-50. doi: 10.1016/0049-0172(90)90046-I.

Flegal, K.M., Carroll, M.D., Ogden, C.L., & Curtin, L.R. (2010). Prevalence and trends in obesity among U.S. adults, 1999-2008. *JAMA, 303*, 235-241. doi: 10.1001/jama.2009.2014.

Fine, J.T., Colditz, G.A., Coakley, E.H., Moseley, G., Manson, J.E., Willett, W.C., & Kawachi, I. (1999) A prospective study of weight change and health related quality of life in women. *JAMA, 282,* 2136-2142. doi: 10.1001/jama.282.22.2136.

Fontaine, K.R. & Barofsky, I. (2001). Obesity and health related quality of life. *Obesity Reviews, 2,* 173-182. doi: 10.1046/j.1467-789x.2001.00032.x.

Fontaine, K.R., Bartlett, S.J., & Barofsky, I.J. (2000). Health related quality of life among obese persons seeking and not currently seeking treatment. *International Journal of Eating Disorders, 27,* 101-105. doi: 10.1002/(SICI)1098-108X(200001)27:1<101::AID-EAT12>3.0.CO;2-D.

Fransen, M., Woodward M., Norton, R., Coggan, C., Dawe, M., & Sheridan, N. (2002). Risk factors associated with the transition from acute to chronic occupational back pain. *Spine, 27,* 226-237. doi: 10.1097/00007632-200201010-00022.

Greve, K.W., Bianchini, K.J., & Ord, J.S. (2012). The psychological assessment of persons with chronic pain. In: G.J. Larrabee (Ed.), Forensice neuropsychology: a scientific approach, 2nd edition (pp. 302-335). New York, NY: Oxford University Press.

Haslam, D.W. & James, W.P. (2005). Obesity. *Lancet, 366*, 1197–1209. doi: 10.1016/S0140-6736(05)67483-1.

Hauptmann, J., Lucas, C., Boldrin, M.N., … Orlistat Primary Care Study Group. (2000). Orlistat in the long-term treatment of obesity in primary care settings. *Archives of Family Medicine, 9,* 160-167.

Heo, M., Allison, D.B., Faith, M.S., Zhu, S., & Fontaine, K.R. (2003). Obesity and quality of life: mediating effects of pain and comorbidities. *Obesity Research, 11,* 209-216. doi: 10.1038/oby.2003.33.

Heo, M., Pietrobelli, A., Wang, D., Heymsfield, S.B., & Faith, M.S. (2010). Obesity and functional impairment: influence of comorbidity, joint pain, and mental health. *Obesity, 18*, 2030-2038. doi: 10.1038/oby.2009.400.

Hitt, H.C., McMillen, R.C., Thornton-Neaves, T., Koch, K., & Cosby, A.G. (2007). Comorbidity of obesity and pain in the general population: results from the Southern Pain Prevalence study. *The Journal of Pain, 8*, 430-436. doi: 10.1016/j.jpain.2006.12.003.

Holmberg, S., Thelin, A., & Thelin, N. (2005). Knee osteoarthritis and body mass index: A population-based case-control study. *Scandinavian Journal of Rheumatology, 34*, 59-64. doi: 10.1080/03009740510017922.

Hoy, D., March, L., Brooks, P., Woolf, A., Blyth, F., Vos, T., & Buchbinder, R. (2010). Measuring the global burden of low back pain. *Best Practice & Research Clinical Rheumatology, 24*, 155-65.doi: 10.1016/j.berh.2009.11.002.

Hulens, M., Vansant, G., Lysens, R., Claessens, A.L., & Muls, E. (2001). Exercise capacity in lean versus obese women. *Scandinavian Journal of Medicine & Science Sports, 11,* 305-309. doi: 10.1034/j.1600-0838.2001.110509.x.

Institute of Medicine. (1995). Weighing the options: criteria for evaluating weight management programs. Washington, D.C.: Government Printing Office.

International Association for the Study of Pain Task Force on Taxonomy. (1994). Part III: Pain terms, a current list with definitions and notes on usage. In H. Merskey & N. Bogduk (Eds.) *Classification of Chronic Pain, Second Edition* (pp. 209-214). Seattle, WA: IASP Press.

International Association for the Study of Pain Task Force on Taxonomy. (2011). IASP Taxonomy. Accessed from the International Association for the Study of Pain, http://www.iasp-pain.org/AM/Template.cfm?Section=Pain_Defi...isplay.cfm&ContentID=1728

Janke, E.A., Collins, A., & Kozak, A.T. (2007). Overview of the relationship between pain and obesity: what do we know? Where do we go next? *Journal of Rehabilitation Research and Development, 44*, 245-262. doi: 10.1682/JRRD.2006.06.0060.

Keller, S., Bann, C.M., Dodd, S.L., Schien, J., Mendoza, T.R., & Cleeland, C.S. (2004). Validity of the Brief Pain Inventory for use in documenting the outcomes of patients with non-cancer pain. *Clinical Journal of Pain, 20,* 309-317. doi: 10.1097/00002508-200409000-00005.

Kolotkin, R.L., Norquist, J.M., Crosby, R.D., Suryawanshi, S., Teixeria, P.J., Heymsfield, S.B. … Nguyen, A.M. (2009). One-year health-related quality of life outcomes in weight loss trial participants: comparison of three measures. Health and Quality of Life Outcomes, 7.

Lake, JK; Power, C.; & Cole, TJ. (2000) Back pain and obesity in the 1958 British birth cohort: cause or effect? *Journal of Clinical Epidemiology, 53,* 245-250. doi: 10.1016/S0895-4356(99)00155-9.

Leboeuf-Yde, C., Kyvik, K.O., & Bruun, N.H. (1999). Low back pain and lifestyle: Part II - Obesity. Information from a population based sample of 29,424 twin subjects. *Spine, 24,* 1518-1525. doi: 10.1097/00007632-199904150-00009.

Losina, E., Walensky, R.P., Reichman, W.M., Holt, H.L., Gerlovin, H., Solomon, D.H., … Natz, J.N. (2011). Impact of obesity and knee osteoarthritis on morbidity and mortality in older Americans. *Annals of Internal Medicine, 154*, 217-226.

Marcus, D.A. (2004). Obesity and the impact of chronic pain. *Clinical Journal of Pain, 20,* 186-191. doi: 10.1097/00002508-200405000-00009.

Martens, M.P., Parker, J.C., Smarr, K.L., Hewett, J.E., Ge, B., Slaughter, J.R., &Walker, S.E. (2006). Development of a shortened Center for Epidemiological Studies Depression Scale for the assessment of depression in rheumatoid arthritis. Rehabiliation Psychology, 51, 135-139. doi: 10.1037/0090-5550.51.2.135.

Martins, C., Strømmen, M., Stayne, O.A., Nossum, R., Mårvik, R., Kulsend, B. (2011). Bariatric surgery versus lifestyle interventions for morbid obesity – changes in body weight, risk factors and comorbidities at 1 year. *Obesity Surgery, 21*, 841-849. doi: 10.1007/s11695-010-0131-1.

Melzak, R. & Katz, J. (1994). Pain measurement in persons in pain. In: P.D. Wall & R. Melzak (Eds.), *Textbook of Pain.* London: Churchhill Livingstone.

Miaskowski, C., Bair, M., Chou, R., D'Arcy, Y., Hartwick, C., Huffman, L., … Manwarren, R. (2008). Principles of Analgesic Use in the Treatment of Acute Pain and Cancer Pain. 6th ed. Glenview, IL: American Pain Society.

Mokdad, A.H., Marks, J.S., Stroup, D.F., & Gerberding, J.L. (2004). Actual causes of death in the United States, 2000. *JAMA, 291*, 1238 – 1245. doi: 10.1001/jama.291.10.1238.

National Center for Chronic Disease Prevention and Health Promotion (NCCDPHP). (2011). *Arthritis – At a Glance*. Accessed from the Centers for Disease Control and Prevention, http://www.cdc.gov/chronicdisease/resources/publications/aag/pdf/2011/Arthritis-AAG-2011-508.pdf

National Institutes of Health & National Heart, Lung, and Blood Institute. (2000). The practical guide: identification, evaluation, and treatment of overweight and obesity in adults. NIH Publication Number 00-4084.

Ogden, C.L., Carroll, M.D., Curtin, L.R., McDowell, M.A., Tabak, C.J., & Flegal, K.M. (2006). Prevalence of overweight and obesity in the United States, 1994-2004. *JAMA, 295*(13), 1549-1555. doi: 10.1001/jama.295.13.1549.

Okifugi, A., Donaldson, G.W., Barck, L., & Fine, P.G. (2010). Relationship between fibromyalgia and obesity in pain, function, mood, and sleep. *The Journal of Pain, 11*, 1329-1337. doi: 10.1016/j.jpain.2010.03.006.

Osman, A., Barrios, F.X., Kopper, B.A., Hauptmann, W., Jones, J., O'Neil, E. (1997). Factor structure, reliability, and validity of the Pain Catastrophizing Scale. *Journal of Behavioral Medicine, 20,* 589-605. doi: 10.1023/A:1025570508954.

Peltonen, M., Lindroos, A.K., & Torgeson, J.S. (2003). Muscoskeletal pain in the obese: a comparison with a general population and long term changes after conventional and surgical obesity treatment. *Pain, 104,* 549-557. doi: 10.1016/S0304-3959(03)00091-5.

Peter, E. & Watt-Watson, J. (2002). Unrelieved pain: an ethical and epistemological analysis of distrust in patients. *Canadian Journal of Research, 34*, 65-80.

Pi-Sunyer, F. X. (2002). The obesity epidemic: pathophysiology and consequences of obesity. *Obesity Research, 10*, 97S-104S. doi: 10.1038/oby.2002.202.

Pi-Sunyer, F.X., Blackburn, G., Brancati, F.L., … Look AHEAD Research Group. (2007). Reduction in weight and cardiovascular disease (CVD) risk factors in individuals with type 2 diabetes: one year results of Look AHEAD trial. *Diabetes Care, 30,* 1374-1383.

Radloff, L.S. (1977). The CES-D Scale: a self-report depression scale for research in the general population. Applied Psychological Measurement, 1, 385-401. doi: 10.1177/014662167700100306.

Ray, L., Lipton, R.B., Zimmerman, M.E., Katz, M.J., & Derby, C.A. (2011). Mechanisms of association between obesity and chronic pain in the elderly. *Pain, 152*, 53-59. doi: 10.1016/j.pain.2010.08.043.

Redinger, R.N. (2007). The pathophysiology of obesity and its clinical manifestations. *Gastroenterology & Hepatology, 3,* 856-863.

Rejeski, W.J., Ettinger, W.H., Jr., Martin, K., & Morgan, T. (1998). Treating disability in knee osteoarthritis with exercise therapy: a central role for self efficacy and pain. *Arthritis Care and Research, 11,* 94-101. doi: 10.1002/art.1790110205.

Rejeski, W.J., Focht, B.C., Meisser, S.P., Morgan, T., Pahor, M., & Pennix, B. (2002). Obese older adults with knee osteoarthritis: weight loss, exercise and quality of life. *Health Psychology, 21,* 419-426. doi: 10.1037/0278-6133.21.5.419.

Ryan, D.H., Johnson, W.D., Myers, V.H., Prather, T.L., McGlone, M.M., Rood, J., … Sjöström, L.V. (2010). Nonsurgical weight loss for extreme obesity in primary care settings. *Archives of Internal Medicine, 170*, 146-154. doi: 10.1001/archinternmed.2009.508.

Sandell, L.J. (2009). Obesity and osteoarthritis: is leptin the link? *Arthritis & Rheumatism, 60*, 2858-2860. doi: 10.1002/art.24862.

Sellinger, J.J., Clark, E.A., Shulman, M., Rosenberger, P.H., Heapy, A.A., & Kerns, R.D. (2010). The moderating effect of obesity on cognitive-behavioral pain treatment outcomes. *Pain Medicine, 11*, 1381-1390. doi: 10.1111/j.1526-4637.2010.00935.x.

Shiri, R., Karppinen, J., Leino-Arjas, P., Solovieva, S., & Viikari-Juntara, E. (2010). The association between obesity and low back pain: a meta-analysis. *American Journal of Epidemiology, 171*, 135-154. doi: 10.1093/aje/kwp356.

Simmons-Morton, D.G., Obarzanek, E., Cutler, J.A. (2006). Obesity research: limitations of methods, measurements, and medications. *JAMA, 295*, 826-828.

Sjöström, C.D., Lissner, L., Wedel, H., & Sjöström, L. (1999). Reduction in incidence of diabetes, hypertension, and lipid disturbances after intentional weight loss induced by bariatric surgery: the SOS intervention study. Obesity Research, 7, 477-484.

Sjöström, L. (2000). Surgical intervention as a strategy for treatment of obesity. *Endocrine, 13*, 213-230. doi: 10.1385/ENDO:13:2:213.

Stewart, A. L., Hays, R. D.,& Ware, J. E. (1992). Methods of validating health measures. In A. L. Stewart & J. E. Ware (Eds.), *Measuring functioning and well-being: The Medical Outcomes Study approach* (pp. 309-324), Durham, NC: Duke University Press.

Stewart, W.F., Ricci, J.A., Chee, E., Morganstein, D., & Lipton, R. (2003). Lost productive time and cost due to common pain conditions in the US workforce. *JAMA, 290*, 2443-2454. doi: 10.1001/jama.290.18.2443.

Stone, A.A. & Broderick, J.E. (2012). Obesity and pain are associated in the United States. *Obesity*. doi:10.1038/oby.2011.397.

Sullivan, M.J.L, Bishop, S., & Pivik, J. (1995). The Pain Catastrophizing Scale: development and validation. *Psychological Assessment, 7,* 524-532. doi: 10.1037/1040-3590.7.4.524.

Sullivan, M.J.L., Sullivan, M.E., & Adams, H.E. (2002). Stage of chronicity and cognitive correlates of pain-related disability. *Cognitive Behaviour Therapy, 31*, 111-118. doi: 10.1080/165060702320337988.

Sullivan, M.J.L, Thorn, B., Haythornthwaite, J., Keefe, F., Martin, M., Bradley, L.A., & Lefebvre, J.C. (2001). Theoretical perspectives on the relation between catastrophizing and pain. *Clinical Journal of Pain, 17*, 52-64. doi: 10.1097/00002508-200103000-00008.

Tan, G., Jensen, M.P., Thornby, J.I., & Shanti, B.F. (2004). Validation of the Brief Pain Inventory for chronic nonmalignant pain. *Journal of Pain, 5,* 133-137. doi: 10.1016/j.jpain.2003.12.005.

Teitel, A.D. & Zieve, D. (2011). Osteoarthritis. Accessed from the National Institutes of Health, http://www.ncbi.nlm.nih.gov/pubmedhealth/PMH0001460/

Tessier, D.J. & Eagon, J.C. (2008). Surgical management of morbid obesity. *Current Problems in Surgery, 45*, 68-137. doi: 10.1067/j.cpsurg.2007.12.003.

Tunis, S.R., Stryer, D.B., & Clancy, C.M. (2003). Practical clinical trials: increasing the value of clinical research for decision making in clinical and health policy. *JAMA, 290*, 1624-1632. doi: 10.1001/jama.290.12.1624.

Turner, J.A. & Aaron, L.A. (2001). Pain-related catastrophizing: what is it? *The Clinical Journal of Pain, 17*, 65-71. doi: 10.1097/00002508-200103000-00009.

United Stated Department of Health and Human Services Center for Drug Evaluation and Research. (2007). Guidance for industry: Developing products for weight management. Accessed from the Food and Drug Administration, http://www.fda.gov/downloads/Drugs/GuidanceComplianceRegulatoryInformation/Guidances/ucm071612.pdf

Vismara, L., Menegoni, F., Zaina, F., Galli, M., Negrini, S., Capodaglio, P. (2010). Effect of obesity and low back pain on spinal mobility: a cross sectional study in women. *Journal of NeuroEngineering and Rehabilitation, 7*. doi: 10.1186/1743-0003-7-3.

Wadden, T.A., Berkowitz, R.I., Sarwer, D.B., Prus-Wisneiwski, R., & Steinberg, C. (2001). Benefits of lifestyle modification in the pharmacologic treatment of obesity. *Archives of Internal Medicine, 161*, 218-225. doi: 10.1001/archinte.161.2.218.

Wadden, T.A., Brownell, K.D., Foster, G.D. (2002). Obesity: responding to the global epidemic. *Journal of Consulting and Clinical Psychology, 70*, 510-525. doi: 10.1037/0022-006X.70.3.510.

Wadden, T.A., Butryn, M.L., Wilson, C. (2007). Lifestyle modification for the management of obesity. Gastroenterology, 132, 2226-2238. doi: 10.1053/j.gastro.2007.03.051.

Wadden, T.A. & Phelan, S. (2003). Behavioral assessment of the obese patient. In T.A. Wadden & A.J. Stunkard (Eds.), *Handbook of Obesity Treatment* (pp. 186-228), New York, NY: The Guilford Press.

Wadden, T.A. & Osei, S. (2003). The Treatment of Obesity: An Overview. In T.A. Wadden & A.J. Stunkard (eds.), Handbook of Obesity Treatment(pp. 229-248), New York, NY: The Guilford Press.

Wadden, T.A., Webb, V.L., Moran, C.H., & Bailer, B.A. (2012). Lifestyle modification for obesity: new developments in diet, physical activity, and behavior therapy. *Circulation, 125,* 1157-1170. doi: 10.1161/CIRCULATIONAHA.111.039453.

Wang, Y. & Beydoun, M.A. (2007). The obesity epidemic in the United States – gender, age, socioeconomic, racial/ethnic, and geographic characteristics: a systematic review and meta-regression analysis. *Epidemiologic Reviews, 29*, 6-28. doi: 10.1093/epirev/mxm007.

Ware, J.E. (2000). SF-36 health survey update. Spine, 25, 3130-3139. doi:10.1097/00007632-200012150-00008.

Ware, J., Kosinski, M., & Keller, S.D. (1994). SF-36 physical and mental health summary scales: A user's manual. Boston, MA: The Health Institute.

Ware, J.E., Snow, K.K., Kosinski, M., Gandek, B. (1993). SF-36 Health Survey: Manual and interpretation guide. Boston, MA: New England Medical Center.

Ware, J.E., & Kosinski, M. (2001). Interpreting SF-36 summary health measures: A response. Quality of Life Research, 10, 405-413. doi: 10.1023/A:1012588218728.

Webb, R., Brammah, T., Lunt, M., Urwin, M., Allison, T., Symmons, D. (2003). Prevalence and predictors of intense, chronic, and disabling neck and back pain in the UK general population. *Spine, 28*, 1195-1202. doi: 10.1097/01.BRS.0000067430.49169.01.

Wing, R.R. & Hill, J.O. (2001). Successful weight loss maintenance. *Annual Review on Nutrition, 21*, 323-341. doi: 10.1146/annurev.nutr.21.1.323.

Wing, R.R., & Phelan, S. (2005). Long term weight maintenance. *American Journal of Clinical Nutrition, 82,* 222S-225S.

Wright, E.A., Katz, J.N., Cisternas, M.G., Kessler, C.L., Wagenseller, A., & Losina, E. (2010). Impact of knee osteoarthritis on health care resource utilization in a US population-based national sample. Medical Care, 48, 785-791. doi: 10.1097/MLR.0b013e3181e419b1.

Wright, L.J., Schur, E., Noonan, C., Ahumada, S., Buchwald, D., & Afari, N. (2010). Chronic pain, overweight, and obesity: findings from a community0based twin registry. *The Journal of Pain, 11*, 628-635. doi: 10.1016/j.jpain.2009.10.004.

World Health Organization. (2011). Fact Sheet No. 311: Obesity and overweight. Retrieved from the World Health Organization, http://www.who.int/mediacentre/factsheets/fs311/en/

Yamakawa, K., Tsai, C.K., Haig, A.J., Miner, J.A., & Harris, M.J. (2004). Relationship between ambulation and obesity in older persons with and without back pain. *International Journal of Obesity and Related Disorders, 28*, 137-143. doi: 10.1038/sj.ijo.0802478.

Yancy, W.S., Olsen, M.K., Westman, E.C., Bosworth, H.B., & Edelman, D. (2002). Relationship between obesity and health related quality of life in men. *Obesity Research, 10,* 1057-1064. doi: 10.1038/oby.2002.143.

Zhao, Y. & Encinosa, W. (2007). Healthcare Cost and Utilization Project (HCUP). Statistical Brief #23: Bariatric surgery utilization and outcomes in 1998 and 2004. Retrieved from the Agency for Healthcare Research and Quality, http://www.hcup-us.ahrq.gov/reports/statbriefs/sb23.pdf

VITA

Brooke Louise Barbera was born and raised in Napoleonville, Louisiana. She earned a Bachelor of Arts degree from Newcomb College of Tulane University in New Orleans, Louisiana. Her undergraduate experience was a liberal arts education with an emphasis in psychology and classical studies. While at Newcomb College, she completed senior honors theses in both disciplines, and graduated *magna cum laude* in May 2003. During and after her tenure at Tulane University, Brooke worked as research assistant at Pennington Biomedical Research Center in Baton Rouge, Louisiana. She entered the doctoral program in psychology at Louisiana State University in 2005, under the supervision of Phillip Brantley, Ph.D. Her clinical emphasis at Louisiana State University was behavioral medicine, with clinical practica completed at Earl K. Long Medical Center and the Louisiana State University Psychological Services Center. Her research efforts continued at Pennington Biomedical Research Center and at Earl K. Long Medical Center, studying weight maintenance, surgical treatment for obesity, and diabetes self-management. She received her Master of Arts degree in clinical psychology from Louisiana State University in 2008. In 2010, Brooke completed an APA-accredited pre-doctoral internship, with emphases in behavioral medicine and neuropsychology, at Warren Alpert Medical School of Brown University in Providence, Rhode Island. While at Brown, she worked under the research mentorship of Bess Marcus, Ph.D., studying the promotion of physical activity in primarily Latina populations. Clinical specialties included consultation-liaison psychology, pre-surgical assessment of bariatric surgery candidates, assessment of pain in outpatient populations, and the neuropsychological assessment of psychiatric hospital patients. Brooke is currently working toward post-doctoral training and licensure at The NeuroMedical Center in Baton Rouge, Louisiana.