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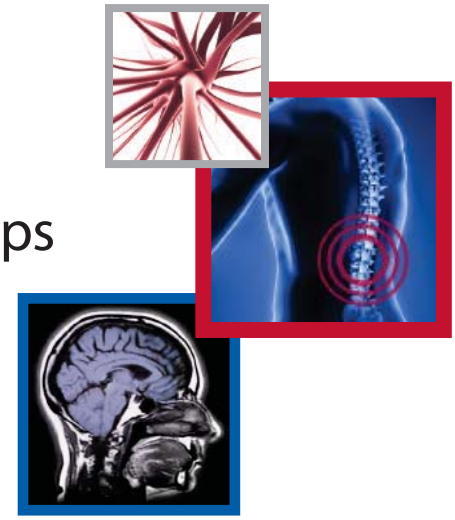

Obesity and low back pain: relationships and treatment

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Practice Points

- Obesity is related to low back pain with increased risk for spine degeneration due to mechanical and inflammatory factors.
- Irrespective of the strategy used, weight loss can reduce the severity and prevalence of low back pain symptoms, most notably in patients who begin at the morbidly obese (BMI >40 kg/m^2) level.
- Exercise interventions for the treatment of low back pain may be effective from several aspects including weight loss, muscle strengthening and reduction of other obesity-related diseases with minimal risk of injury to the patient.

SUMMARY Low back pain (LBP) and obesity (defined as BMI ≥30 kg/m^2) are common problems in the general population. The treatment of LBP is not clearly defined and has variable success in different populations and time-frames. When a person presents with LBP who also meets the criteria for obesity, many questions are raised as to the value of weight-loss treatments and the success and/or safety of usual treatments of LBP, especially exercise. Based on the current medical literature for the treatment of LBP in patients who are obese: there should be attempts at weight loss (nonsurgical or surgical), especially if the person is in the morbidly obese (BMI >40 kg/m^2) range; all usual forms of exercise and physical therapy treatments are possible and can be beneficial without increased concerns for injury; and, if necessary, invasive treatments such as spinal injections or surgery are potentially beneficial and have similar complication rates to the nonobese population. These options should be offered when medically necessary and appropriate.

Epidemiology of obesity in patients with spine conditions

Low back pain (LBP) is a common musculoskeletal issue, with approximately 17% of individuals ≥18 years of age reporting LBP in a 2007 study [1]. In recent large European and American cohort studies, the incidence of chronic LBP ranges from 21 to 26.3% in men and women [2]. A recent estimate of the annual costs of LBP-related complications is US$85.9 billion in 2005 in the USA, representing a 65% increase in costs since 1997 [3]. Recent estimates of the prevalence of obesity among persons with LBP range from 49.4% to 68.1% in men and women, respectively [2].

Obesity, defined as a BMI ≥30 kg/m^2, has been identified as a culprit contributing to chronic LBP [4] and higher severity levels of back pain

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symptoms [5]. In children with Prader–Willi syndrome, those with high BMI values have a higher risk of kyphotic deformity in conjunction with scoliosis [6]. Excessive body weight is directly related to several conditions of the spine, including the number of lumbar discs with degeneration [7], progression of disc disease [7], increased likelihood of radicular pain and neurologic symptoms [8] and a higher prevalence of lumbar facet osteoarthritis [9]. Being overweight, defined as BMI >25 kg/m^2, at a younger age leads to an increased risk [7] and severity [10] of disc degeneration. If left untreated, these spine conditions can progress and adversely affect functional ability, mobility and participation in activities in the home and community [10–14]. From the quality of life perspective, this clinical population faces the increased likelihood of negative affect and low mental outlook, especially if there are other comorbidities present.

The causal effect between obesity and LBP is likely multifactorial and involves differences in mechanical load to the spine, the effects of systemic chronic inflammation and decreased disc nutrition [15]. There are known postural changes with obesity that are likely contributing factors to an increase in mechanical load to the spine that include:

- A more flexed trunk posture leading to a decrease in flexion motion of the thoracic and thoracolumbar segments [16];
- An increased lordosis and decreased lateral bending of the lumbar spine [16];
- An increased hip joint movement [17];
- An increased anterior pelvic tilt [18];
- An increased hip-to-bench ratio for table-top work activities [17] (a person has to stand further back from the table due to the size of the abdomen and reach farther forward).

This limited spinal mobility has a negative impact on disc nutrition by limiting fluid movement and rehydration [15]. The discs and z-joints are also affected directly by increased inflammatory responses generated by the adipose cells in the form of cytokines and acute-phase reactants [15,19–21]. Disc nutrition is also reduced by a decrease in blood supply to the disc due to atherosclerotic changes in the blood vessels to the disc [15]. This is common in persons with obesity due to dyslipidemia (elevated triglycerides and low-density lipoprotein cholesterol with low high-density lipoprotein cholesterol) and hypertension, and the limited blood supply leads to malnutrition of disc cells.

Despite these proposed and known theories of the effects of obesity on the spine, the role of obesity in directly causing LBP is still being confirmed. A review by Leboeuf-Yde of 56 research studies between 1965 and 1997 led to a conclusion that increased body weight may be a possible weak risk factor for LBP, but there were insufficient data to link it as a clear causative factor [22]. In 2009, Shiri *et al.* identified 33 studies to include in a meta-analysis on the issue [15]. The conclusions they obtained were that obesity was associated with chronic LBP, a higher prevalence of LBP in the past 12 months and a higher incidence of seeking care for LBP. Overweight persons had a higher prevalence than nonoverweight people, but lower than the obese population. In the most recent 10-year population study published from Norway of over 60,000 persons in the HUNT study, high BMI was significantly associated with an increased prevalence of LBP [2]. The effect was stronger in women than men, and was not significantly impacted by covariables such as smoking, education, employment status, activity at work or leisure-time physical activity.

The current consensus on treatment for subacute or chronic LBP involves some form of exercise [23–25] or physical activity, along with the use of medications, physical modalities, psychological/behavioral treatment, injections or surgery. Patients with LBP who are overweight or obese are often recommended to undergo a weight-loss program with or without the exercise/activity program. These patients want to know: whether their weight/size is contributing to their LBP; whether a weight reduction program by itself will reduce or eliminate their LBP; whether an exercise/physical activity treatment program for their LBP will be safe and have the same benefit as it would in a smaller/thinner person; whether their size/obesity will limit the effect of the other forms of nonoperative treatment for LBP such as medications, modalities, behavioral treatment or spinal injections; and whether the patient should ever consider surgical options for a disc herniation, degenerative lumbar stenosis or other degenerative problems.

Weight reduction & treatment of LBP

The medical literature for weight reduction as a treatment for LBP has very good evidence that weight reduction is an effective measure in obesity surgery, but less evidence for nonsurgical programs. Melissas *et al.* initially reported on a group of 50 morbidly obese (BMI >40 kg/m^2) patients

undergoing vertical-banded gastroplasty with mean presurgical BMI of 47 kg/m^2 and weight of 132 kg [26]. The prevalence of LBP prior to surgery was 58% (29 of 50) compared with 24% in a lean control group. Two years after vertical banded gastroplasty surgery, 19 of the 29 obese patients with LBP had complete resolution of symptoms, and the other ten had decreased frequency of symptoms and use of medications. The mean postsurgical BMI had decreased to 32.9 kg/m^2 and functional status had improved on both the Roland–Morris Disability Index and the Oswestry Disability Index (ODI) [26]. In a separate patient population, Khoueir *et al.* later reported on their bariatric surgery group that had 38 of 58 (65%) patients with axial LBP who had a significant mean 44% decrease in visual analog scale (VAS; preoperative mean: 5.2; postoperative mean: 2.9) and a significant mean 24% decrease in ODI (preoperative mean: 26.75; postoperative mean: 20.35) [27]. At the 12-month follow-up, there was also a significant 58% improvement in physical health as assessed by Short Form-36. This markedly obese group decreased BMI from 52.3 to 38.3 kg/m^2. Even with a shift only from morbidly obese to obese status, LBP decreased and functional measures improved. Based on these and other studies, there is a clear reduction in LBP with surgical interventions for obesity.

By contrast, the literature regarding successful reduction of LBP with nonoperative treatment of obesity is less conclusive but starting to show signs of success. Early studies demonstrated only possible beneficial effects of weight loss on LBP, but were much less specific in design to answer this question. A lifestyle-modification study of hypertensive persons in a general working population demonstrated a decrease in weight and BMI with an increase in physical activity, but no effect on LBP compared with a control group [28]. The mean BMI in this study intervention group was 29.4 kg/m^2, which is much lower than the starting point for the surgical or other nonsurgical studies. In a separate trial of 35 females with musculoskeletal pain enrolled in a 12-week weight-loss program, the subjects showed a decrease in pain for only four body regions, but did show a decrease in overall pain [29].

The most recent study of nonsurgical weight loss in 46 adults with LBP and a mean BMI of 44.7 kg/m^2 used a 52-week multidisciplinary program [30]. This program included liquid meal replacements for 12 weeks (phase 1) followed by supervised caloric restriction from weeks 13–52 (phase 2). Study participants went to weekly educational meetings and group therapy sessions for the first 26 weeks, and monthly group meetings for the second 26 weeks. During the second 26 weeks, they were to also participate in 60–90 min of physical activity. Over the yearlong study, mean BMI was reduced to 39.6 kg/m^2 (mean decrease of 5.1 kg/m^2 [11.4%]) and ODI improved significantly from 31.9 to 27.1, and Numerical Pain Score (NPS) trended towards improvement (3.3 at baseline; 2.6 at 1 year). Weight, BMI, NPS and ODI all showed the most improvement during the initial 13 weeks of liquid meal replacements, and as the mean weight and BMI decreased, the NPS and ODI scores also decreased. The higher the BMI at baseline and the greater the percentage of reduction in BMI at week 14 and week 53, the more positive was the association with improvements in disability. There were some subjects who experienced weight loss in both phase 1 and phase 2, and these were the most likely subjects to experience a decrease in NPS. This is the most well-designed and definitive study carried out to date that indicates that nonoperative weight loss in an obese population with LBP can be successful in reducing symptoms and increasing function.

Exercise & obesity

There have been many concerns about the safety and/or benefit of initiating exercise in obese persons. Janney and Jakicic randomized 397 sedentary adults with BMI between 25 and 40 kg/m^2 to exercise intervention or control groups [31]. Over the 18-month study, 32% of the subjects (in either the exercise or control group) experienced at least one injury that they attributed to exercise, but only 7% were attributed to exercise alone. Overall, the overweight and obese subjects in the exercise group did not have any higher incidence of musculoskeletal injury than the control group subjects. Interestingly, in both the exercise and control groups, the higher the BMI at baseline, the higher the risk of injury, and there was a direct relationship between this and the time to first injury. Any weight reduction during the study was related with a decrease in the relative risk of injury as time went on.

Exercise therapy regimens to treat LBP can be administered without significant risk of injury in trials of effectiveness. Mangwani *et al.* demonstrated in 140 patients with LBP categorized as normal (BMI <25 kg/m^2), overweight

(BMI 25–29.9 kg/m^2) and obese (BMI >30 kg/m^2) that BMI does not limit recovery from LBP with the use of physical therapy for up to 12 weeks [32]. All three groups showed a significant improvement in pain intensity, physical impairment index and range of motion of the lumbar spine. Similarly, Snow *et al.* randomized 54 obese patients (mean BMI 42.6 kg/m^2) with musculoskeletal pain as measured by VAS (0–10 points) >5 to a physical therapy treatment program versus control group prior to a weight-loss program [33]. The group receiving physical therapy experienced a significant reduction in VAS to 2.3 with the weight-loss program. Both the treatment and control groups then showed a significant decrease in BMI and body fat composition after the 6-month intervention. The physical therapy treatment group, however, did not show any ability to lose more weight than the control group. Even though the physical therapy group's pain level was lower and their physical activity level was higher, they did not have any further reduction in pain levels based on weight loss.

Other types of exercise have been tested in subjects with obesity that did not have LBP including aerobic training programs, resistance exercise programs and aquatic-based programs. Obese persons with osteoarthritis (mostly hip and knee, but may have included LBP) have been shown to tolerate and significantly benefit from a 20-week community-based aquatic exercise program [34]. The exercise protocol included range of motion, strengthening and endurance exercises for 45–60 min per session. This study did not specifically assess the effects of aquatic exercise on weight loss. Studies have shown equal weight-loss success with water walking [35] and underwater treadmill programs [36] compared with land-based training if there is equal intensity, frequency and duration to the protocols. It has also been shown that weight loss is clearly associated with a reduced loading pattern to the knee joint [37].

Cardiovascular and resistance training programs may also be an option for obese persons, as Schjerve *et al.* showed by randomizing participants with BMI >30 kg/m^2 to three different treatment groups [38]. The groups were a high-intensity interval aerobic training group, a continuous moderate-intensity aerobic training group and a maximal strength training group doing resistance training along with abdominal and back exercises. All groups showed improvements in maximal oxygen uptake, aerobic work capacity and endothelial function without significant injuries. Both aerobic training groups showed a decrease in body weight, while the resistance training group did not. This study did not include subjects with LBP, and although these are not the primary types of exercise performed for the treatment of LBP, as the literature will show below, any type of exercise may provide benefit for LBP.

Other treatments for LBP

Persons with axial LBP have many options for treatment, and the inconclusive nature of the medical literature about the best treatment practices leads to a lot of scrutiny and debate. A recent comprehensive review of treatment for nonspecific LBP evaluated physical, behavioral, and modality practices for their best medical evidence [39]. Following Cochrane Database review techniques, there were 83 randomized controlled trials (RCTs) identified in adults with greater than 12 weeks of LBP that achieved adequate scientific merit. Most of these were for exercise therapy (n = 37), behavioral treatment (n = 21), multidisciplinary treatment (n = 6) and back school (n = 5), with very few appropriate trials on modalities and other commonly used treatments such as transcutaneous electronic nerve stimulation (n = 6), low-level laser therapy (n = 3), traction (n = 1), massage (n = 0), back supports (n = 0) and heat/cold therapy (n = 0). The main conclusions were that: moderate evidence exists for multidisciplinary treatment programs to reduce pain intensity and disability in the short-term compared with no treatment or waitlisted controls; low evidence exists regarding behavioral treatment to reduce pain intensity in the short-term compared with no treatment or waitlisted controls; low evidence exists for exercise therapy to improve short-term pain intensity and disability, but clearly does improve long-term function; and there are insufficient data to determine the effects of any of the other treatments. There was no subgroup analysis for the relationships between any of the treatment options compared with BMI or obesity.

A further analysis of the exercise RCTs by van Middlekop's group confirmed their effectiveness in reducing pain and improving function, while emphasizing that no specific exercise regimen is more effective than any other [23]. Hayden *et al.* also completed a meta-analysis of exercise treatment for LBP and utilized 61 RCTs encompassing 6390 participants [24]. They reported similar

findings for improvements in pain and function with persons experiencing chronic LBP. The effects are much less clear for subacute LBP, as they concluded that exercise therapy is equally as effective as no treatment and other conservative treatments, except for mild evidence to support graded-activity exercises in occupational settings [25]. Generally, maintaining usual physical activity in acute LBP is the recommended treatment regimen; however, this is questionable in obese persons who may not have regular activity, exercise or a very active lifestyle. As noted in the exercise section above, the use of exercise in obese patients with LBP is as safe and efficacious as it is in the nonobese population.

Although cognitive–behavioral therapy (CBT) is the most well-established treatment option for persons with chronic LBP, it is most often a component of a more comprehensive program including exercise. The multidisciplinary programs may contribute benefit to obese patients by the weight-loss effects of exercise, and the beneficial effects of the exercise are known to be equal in the obese and nonobese populations [32]. Recently, it has been noted that an obese patient may have a harder time getting started with the exercise due to increased fear of movement based on higher scores on the Tampa Scale of Kinesiophobia and decreased mental subscores on the Short Form-8 [40]. The use of CBT without the exercise component has shown less effect in an obese population compared with a nonobese population on measures of disability, physical aspects of quality of life and emotional functioning [41]. These are the first studies to begin to stratify the evidence from the general population with LBP to the relationship of baseline emotional indicators and obesity levels.

Medications are commonly used for the treatment of acute [42] and chronic LBP [43]. These include NSAIDs [44], skeletal muscle relaxants, antidepressants and opioids. Except for postoperative pain [45,46], these medications do not necessarily cure any of the sources of LBP but should be used to assist with a person's ability to maintain usual physical activity levels and/or perform an exercise regimen. The effects of these medications may be altered by the patient's size and obesity. NSAIDs may be more necessary and essential in an obese patient because of the inflammatory cascade related to adipose cells [19,20]. Some of the skeletal muscle relaxants are affected by obesity-induced increases in CYP2E1 activity in the liver, which alters metabolism of chlorzoxazone and other lipophilic drugs [47,48]. The effectiveness and safety of opioids are effected by the volume of distribution, ideal body weight and degree of lipophilic properties [49–51].

Both spinal injection procedures and surgical procedures are reported to be more technically difficult and experience more complications in obese patients; however, there are few data to support this position. The most common spinal injection procedures utilized for the treatment of chronic LBP are epidural steroid injections, z-joint (facet) injections and radiofrequency denervation of the z-joint.

Recent publications regarding the success or failure of lumbar facet radiofrequency denervation procedures did not show obesity to be a variable in the outcome, and obese patients did as well as nonobese patients [52]. As the diagnostic medial branch blocks that are performed to determine who is a candidate for denervation are transitioned from fluoroscopic or computed tomography-guided procedures to ultrasound-guided procedures, there may be some limitations with the use of ultrasound [53] in obese patients, which will require the continued use of fluoroscopy in obese patients. However, in obese older patients with LBP from degenerative lumbar stenosis, the obese patients experienced a more significant improvement in pain scores than did the nonobese patients over a 3-month follow-up [54]. To have a better outcome due to obesity could not be explained by the authors, but would certainly emphasize the fact that spinal injections have the same potential benefit in obese patients as in nonobese patients. There were no data found for epidurals in acute radiculopathy in obese versus nonobese patients.

In the surgical population for LBP, studies have reported variable effects of obesity on obese patients undergoing fusions and other procedures. Djurasovic *et al.* reported that outcomes for lumbar fusion were similar for obese and nonobese patients, but wound complications were more common in obese patients [55]. Patel *et al.* published a similar increase in complications for 84 patients undergoing elective thoracic or lumbar fusions, and showed an increasing complication rate from 14% with BMI <25 kg/m^2 to 20% with BMI between 25 and 30 kg/m^2, up to 36% with BMI >40 kg/m^2 [56]. In recent publications from the SPORT, which tried to randomize patients for operative versus nonoperative treatment of disc herniation [57], degenerative lumbar stenosis [58] and degenerative lumbar

spondylolisthesis [59], obese patients did not have the same rate of success as nonobese patients with either treatment, but obesity did not turn out to be an independent variable for determining whether a patient should have operative or nonoperative treatment.

By contrast, Yadla *et al.* demonstrated no relationship between BMI and the incidence of minor or major complications in elective thoracolumbar procedures for degenerative problems, which included 69% who had a thoracic and/or lumbar fusion and 31% with decompression only [60]. This population had a mean BMI of 31.3 kg/m^2, with 40.8% meeting the definition of obese and 11.5% being morbidly obese. There was an increased risk related to age, hypertension and performance of a fusion, but not to BMI. Gepstein *et al.* have published similar findings in an obese, elderly population [61]. In a study of anterior lumbar surgery for fusion, Peng *et al.* showed no difference between obese and nonobese patients on complications and perioperative outcomes such as blood loss, analgesic use, time to ambulation or length of hospitalization [62].

With the more recent interest in performing minimally invasive surgeries and fusions, Singh *et al.* demonstrated a high success rate in obese patients undergoing posterior lumbar interbody fusion by a less invasive technique [63]. The technique was technically more demanding but had less blood loss and complications than more open techniques. Rodgers *et al.* report no greater risk of complications with obesity when doing minimally invasive extreme lateral interbody fusion [64]. Tomasino *et al.* showed equal results in obese patients with tubular discectomy, and they had reduced incision length and length of stay in addition to the reduced blood loss and operative times [65]. Minimally invasive lumbar discectomy shows a lower complication and adverse event rate than minimally invasive fusion surgery, but there were no differences in either group related to obesity [66]. For these types of procedures, a high BMI should not be considered as a reason not to do surgery.

Obesity may lead to the presence of more severe radiculopathy based on electromyographic evaluations [67], and may increase the risk of recurrent herniated nucleus pulposis after lumbar discectomy [68]. Meredith *et al.* report that an obese patient is 12-times more likely to have a recurrent disc herniation and is 30-times more likely to require reoperation than a non-obese patient [68]. By contrast, Kim *et al.* report recurrent herniation is affected by disc height and segmental motion and is not related to obesity [69].

Conclusion & future perspective

An obese person with LBP now knows that:

- There is a direct relationship between their level of obesity and the likelihood they will get LBP;
- They should undergo a weight-loss program, either surgical or nonsurgical, to help reduce or eliminate their LBP. This is going to be most effective if the patient is in the morbidly obese category (BMI >40 kg/m^2) and is more effective nonsurgically when combined with an exercise regimen that will help increase physical function and decrease disability;
- It is safe for an obese person to do many different forms of exercise for their LBP, and they can enjoy the same improvement as other patients when doing physical therapy for LBP;
- Medications for their LBP may have to be adjusted due to their size, but there are no data to suggest that there should be any differences in treatment compared with a nonobese person with LBP when considering the use of modalities or spinal injections;
- The use of CBT by itself may not be as effective in an obese person, but will be more effective as part of a multidisciplinary program;
- Most surgical procedures for discectomies, decompressions and lumbar and/or thoracolumbar fusions have the same complications and outcomes regardless of patient size, and this is becoming more evident with minimally invasive procedures. One of the few concerns in surgery is whether there is a significant risk of recurrent disc herniation in the obese population.

Therefore, based on the current medical literature regarding obesity and the treatment of LBP, the only change in treatment for an obese patient is to add a weight-loss program to the usual pathway. All future studies on the nonsurgical and surgical treatment of LBP need to more rigorously quantify the level of exercise and weight lost during treatment and perform subgroup analyses of the effects in patients with acute versus subacute versus chronic LBP and who are overweight (BMI 25–30 kg/m^2) versus obese (BMI 30–40 kg/m^2) versus morbidly obese (BMI >40 kg/m^2).

Financial & competing interests disclosure

The authors have no relevant affiliations or financial involvement with any organization or entity with a financial interest in or financial conflict with the subject matter or materials discussed in the manuscript. This includes employment, consultancies, honoraria, stock ownership or options, expert testimony, grants or patents received or pending, or royalties.

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