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ERGONOMY (N TEASDALE, SECTION EDITOR)

Obesity and Low Back Pain: Is There a Weight of Evidence to Support a Positive Relationship?

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Abstract Obesity and low back pain (LBP) are responsible for significant morbidities and financial expenditure. Numerous studies have demonstrated a positive relationship between obesity and LBP, but a concurrent investigation of causality is often omitted. Spinal clinicians routinely prescribe exercise and weight loss for obese patients with LBP, despite a paucity of literature evaluating why obesity might cause LBP or how exercise and weight loss might be suitable treatments for LBP. Etiologies have tended to focus on the biomechanical effects of obesity that lead to excessive loading and degeneration of the lumbar spine. However, recent evidence suggests that systemic inflammation associated with obesity may also be an important contributor to LBP. In this article, the latest evidence investigating the relationship between obesity and LBP is reviewed, an overview of the impact of exercise and weight loss on LBP is provided, and proposed mechanisms connecting obesity, systemic inflammation, and LBP are outlined.

Keywords Obesity · Low back pain · Exercise · Weight loss · Lifestyle modification · Bariatric surgery · Non-surgical · Health care utilization · Body mass index · Inflammation · C-reactive protein · Vitamin C · Morbidity · Etiology

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Introduction

Obesity is one of the most detrimental medical conditions afflicting modern society, not only in terms of morbidity and mortality but also with regards to out-of-pocket, insurance and government costs. As population levels of obesity have continued to rise, so too have the number of individuals with low back pain (LBP). LBP is a common musculoskeletal disorder in both developed and developing countries. Because of the resultant discomfort and disability, LBP is associated with high levels of health care utilization and work absenteeism. Although many suspected risk factors have been studied, the etiology of LBP remains equivocal. Given the increasing number of obese individuals coinciding with the escalating prevalence of LBP, there is a growing interest in establishing the impact of obesity on LBP and determining the physiological causality.

Obesity

Obesity is defined as abnormal or excessive fat accumulation that presents a risk to health and wellbeing. Originally, obesity was considered the end-product of an imbalanced equation in which the body consumes more calories than it

burns. Although over-eating and under-exercising are significant contributors, a myriad of genetic, environmental, behavioral, and social factors are now recognized as playing a major role [1, 2]. Obesity, categorized at a population level as body mass index (BMI) greater than 30 kg/m^2, is a risk factor for cardiovascular disease and cancer. Fat deposition in the abdominal region has been linked with diabetes. Obesity has also been linked to systemic inflammation which contributes to the pathogenesis of metabolic dysfunction [3].

The latest statistics indicate obesity rates have reached epidemic proportions in North America: 24.1 % of adult Canadians and 35.7 % of adult Americans are currently classified as obese [4, 5]. The economic impact of obesity and concomitant diseases assume a significant portion of health care expenditure. If current obesity trends continue to escalate, by 2030 all 50 states of the United States of America (USA) could have adult obesity rates of at least 44 % and treatment costs for obesity-related diseases could add $48-$66 billion to current estimates of $147-$210 billion per year [6, 7].

Low Back Pain

LBP corresponds with spinal and paraspinal symptoms in the lumbosacral region. There are numerous potential sources of LBP including but not limited to intervertebral discs, facet joints, vertebrae, neural structures, muscles, ligaments, and fascia. Often LBP can be difficult to diagnose due to varying presentations, multiple potential etiologies, lack of a definitive diagnostic test, and the complex nature of neuropathic and nociceptive pain [8–10]. As a result, symptoms may become exacerbated, requiring an increase in the complexity and number of treatments.

Around 26 % of North American adults will experience LBP at some point every year [11]; the lifetime prevalence of LBP is estimated at 84 % [12]. With the increasing number of LBP patients has come an exponential rise in the cost of its treatment. Drug prescriptions, in-patient treatments, and emergency services have all been the subject of a significant increase in per-user expenditure [13]. Total direct costs associated with treatment for spine pain (primarily LBP) exceed $85 billion per year in the USA [14]. The financial impact would be doubled to almost $170 billion per year if disability payments and lost taxation revenue attributable to spinal disorders were also considered [15].

Relationship between Obesity and Low Back Pain

Obesity has long been suspected as a contributing factor in the presentation of musculoskeletal pain in the hips, knees and feet. Weight accumulation, particularly in the upper body, places a greater mechanical load on weight-bearing joints and structures, potentially increasing the rate of degeneration through excessive wear-and-tear. Patients with increased BMI are more susceptible to distal lower extremity injuries than those within normal bodyweight limits [16]. Based on our experience, spinal clinicians also believe obesity contributes to LBP in much the same manner. Consequently, obese patients are often advised to lose weight upon spinal consultation for two proposed reasons: 1) to decrease the mechanical load on the affected weight-bearing spinal structures and; 2) to moderate the obesity-induced lordotic curvature of the lumbar spine.

Previous reviews and meta-analyses have identified possible connections between obesity and LBP [17•, 18]. Opportunely, a raft of recent studies has added much needed evidence to support a positive relationship between obesity and LBP [19••, 20–23, 24••, 25–33] (Table 1). In addition, results from the Spine Patient Outcomes Research Trial (SPORT) indicate that obesity can influence lumbar spine surgery treatment outcomes. Patients with degenerative spondylolisthesis with BMI greater than 30 kg/m^2 had a higher postoperative infection rate, twice the reoperation rate, and less improvement in physical function at 4-years follow-up than those with BMI less than 30 kg/m^2 [34]. Likewise, obese patients with lumbar disc herniations experienced significantly fewer improvements in primary outcome measures (i.e., physical function, bodily pain, disability) than non-obese patients in both operative and non-operative treatment groups [35•].

The majority of studies to date focus more on establishing a relationship between obesity and LBP rather than examining any physiological causality. Obesity-specific mechanisms that have been mentioned in relation to LBP include: 1) a disproportion of fat versus muscle leading to detrimental compensatory actions; 2) mechanical stressors causing degeneration of load-bearing structures and; 3) chronic whole-body systemic inflammation. High-quality prospective data that supports the general belief of a biomechanical relationship between obesity and LBP is lacking. Moreover, because anecdotal findings and a cohort study [36••] suggest that even mild weight loss results in improvements in LBP, there are likely non-biomechanical pathways involved which have not yet been fully explored.

Bariatric Surgery, Weight Loss, and Low Back Pain

Obese patients are commonly advised to lose weight to help alleviate their LBP symptomatology. While lifestyle modification provides the foundation for weight loss, bariatric surgery is the only remaining option for some patients due to musculoskeletal difficulties that prevent the completion of daily exercises [37]. Bariatric surgery consistently results in sustained weight loss and decreases in the rate of concomitant diseases and mortality above and beyond that which is possible

Table 1 Recent studies indicating positive relationships between obesity and low back pain

Reference	Year	Country	Population	Age	Summary of findings
[19••]	2013	Norway	LBP >3 months in previous year (LBP: n=3,254; Total: n=25,450)	30-69 years	LBP with BMI ≥30 kg/m^2: OR=1.34 (men); OR=1.22 (women)
[20]	2012	Kuwait	LBP±sciatica referred for MRI (LBP: n=138; Total: n=214)	16-29 years	54 % of LBP patients were obese ‡ DDD with BMI ≥30 kg/m^2: OR=16.73
[21]	2012	Iran	LBP in previous 30 days (LBP: n=7,415; Total: n=25,307)	20-65 years	17 % of LBP patients were obese ‡ LBP with BMI ≥30 kg/m^2: OR=1.15
[22]	2012	UK	LBP >1 day in previous month (LBP: n=553; Total: n=3,376)	17 years	LBP with BMI ≥30 kg/m^2: OR=1.05 (boys); OR=1.39 (girls)
[23]	2012	Turkey	LBP >2 months (LBP: n=45; Total: n=60)	NR	BMI higher in LBP patients who had metabolic syndrome (P=0.03)
[24••]	2012	Finland	No LBP (≤7 days in previous year) (LBP: n=1,224; Total: n=1,224)	24-39 years	Radiating LBP with abdominal obesity: OR=1.7
[25]	2012	Australia, NZ, UK	LBP reported between 2006–2008 (LBP: n=319; Total: n=928)	21-67 years	31.5 % of LBP patients were obese ‡ LBP with BMI ≥30 kg/m^2: OR=1.1 (day); OR=1.34 (shift)
[26]	2011	Spain	Data collection June 2006–2007 (LBP: n=6,515; Total: n=29,478)	≥16 years	LBP with BMI <30 kg/m^2: 18.3 %; BMI ≥30 kg/m^2: 26.1 %
[27]	2011	Norway	LBP ≥3 months in previous year (LBP: n=3,314; Total: n=32,417)	≥20 years	9 % of LBP patients were obese ‡ LBP with BMI ≥30 kg/m^2: RR=1.21 (men); RR=1.21 (women)
[28]	2011	Finland	LBP or sciatica between 1981–2008 (LBP: n=373; Total: n=9,016)	14-42 years	2nd hospitalization with surgery for sciatica with BMI ≥25 kg/m^2: HR=0.5 (men); HR=7.1 (women)
[29]	2011	China	Juvenile DDD (LBP: n=29; Total: n=83)	13-20 years	Overweight/obesity had significant association with juvenile DDD (OR=14.19; P=0.023)
[30]	2011	USA	LBP-based absence of ≥4 days (LBP: n=381; Total: n=5,691)	NR	76 % of LBP patients were overweight/obese † LBP with BMI ≥30 kg/m^2: OR=2.15
[31]	2011	Australia	Recruited for obesity+MSK study (LBP: n=135; Total: n=135)	25-62 years	BMI and fat mass positively associated with low back disability (P<0.0001)
[32]	2011	Netherlands	Persistent LBP over 10 years (LBP: n=4,738; Total: n=5,706)	20-60 years	18 % of LBP patients were obese ‡ LBP with BMI ≥30 kg/m^2: OR=1.62
[33]	2011	USA	LBP >3 months (LBP: n=192; Total: n=192)	NR	48 % of LBP patients were obese ‡ ODI scores were significantly higher in obese patients (P<0.001)

† Overweight/Obese: BMI ≥25 kg/m^2, ‡ Obese: BMI ≥30 kg/m^2

LBP: low back pain, BMI: body mass index, OR: odds ratio, MRI: magnetic resonance imaging, DDD: degenerative disc degeneration, UK: United Kingdom, NZ: New Zealand, RR: risk ratio, HR: hazard ratio, USA: United States of America, MSK: musculoskeletal, ODI: Oswestry Disability Index

with exercise and dietary changes and pharmacological treatments [38]. As such, indications for bariatric surgery have been relaxed: what was originally considered a procedure for only the morbidly obese (i.e., BMI ≥40 kg/m^2, or BMI ≥35 kg/m^2 with serious co-existing medical conditions) is now being performed in patients with BMI 30–35 kg/m^2.

Recent studies illustrate the presence of a relationship between weight loss following bariatric surgery and reduced LBP. A prospective cohort study in 30 morbidly obese patients showed that an average reduction in body weight of 37 kg 12-months after surgery significantly decreased axial and radicular LBP [39]. In a prospective comparative study of 25 patients, an average weight loss of 22 kg three months after surgery led to a 54 % decrease in the severity of LBP and a significant improvement in quality of life [40•]. A similar prospective study in 20 patients indicated that the average weight loss 3-months after surgery was 24 kg, and that the Numeric Pain Rating Scale demonstrated a significant decrease in LBP [41]. Another prospective assessment of 38 patients showed an average decrease in mean weight of 39 kg at 12-months post-operation, with a statistically significant mean 44 % decrease in axial LBP on the Visual Analogue Scale [42].

Unfortunately, adverse events and complications have long-been associated with bariatric surgery. Gastric bypass presents the most risk but offers the most benefit, gastric banding has the lowest risk and is the least effective, while sleeve gastrectomy is positioned in-between in terms of morbidity and effectiveness [43]. Of 5364 patients who underwent bariatric surgery, the 1-year mortality rate was 0.06 % and the frequency of complications was 5.4 % for gastric banding, 6.5 % for sleeve gastrectomy, and 9.7 % for gastric bypass [44]. Medical complications tend to focus on malabsorption, gastric dumping syndrome and nutritional deficiencies [45]. In addition, patients undergoing bariatric surgery use more in-patient and non-primary out-patient care during follow-up [46]. Bariatric surgery is clinically-effective and cost-effective for obese patients with BMI 30–40 kg/m^2 and diabetes, but is less likely to be cost-effective for patients with BMI 30–35 kg/m^2 and no diabetes [47].

Exercise, Weight Loss, and Low Back Pain

For the obese patient, completion of a regular exercise regimen (with or without caloric restriction) ideally leads to weight loss and improvements in risk factors for diabetes and cardiovascular disease. Even with negligible weight loss, obese patients with a good level of cardiorespiratory fitness are at reduced risk for cardiovascular mortality compared to lean but unfit individuals [48]. Recent evidence suggests combination exercise (i.e. aerobic+resistance) gives greater benefits for weight loss, fat loss and cardiorespiratory fitness than aerobic or resistance training alone [49]. Regardless of modality, regular exercise is also vitally important for long-term maintenance of weight loss [50]. For these reasons, exercise is a predominant component in lifestyle modifications commonly prescribed by spine clinicians for obese patients suffering from LBP.

Previously, researchers have sought to determine whether low-intensity aerobic exercise, moderate-to-high intensity aerobic exercise, stretching or resistance training programs are better for LBP patients. In 52 sedentary patients with LBP, 6-weeks of moderate intensity walking were as effective as 6-weeks of muscle strengthening exercises in improving LBP outcomes [51]. Similar improvements in pain intensity, disability, anxiety and depression have been reported in a study of 50 patients with LBP who completed a 12-week high intensity aerobic exercise program compared to a passive modalities group who performed only moderate intensity exercise and general strengthening activities [52]. A comprehensive study of 286 patients with LBP who were randomized to a 12-week program comprising high and low intensity exercise, occupational therapy, and education or a 12-week program comprising back muscle strengthening exercises reported significant improvements in both groups with regard to pain, disability, and most quality of life dimensions over a 24-month follow-up period [53]. An individualized 6-week clinical Pilates program in 60 patients with LBP also produced similar beneficial effects on self-reported disability, pain, function and health-related quality of life as a general exercise program [54].

Currently, the literature is bereft of high-quality studies that elucidate the extent to which exercise and weight loss can reduce LBP for obese patients. Furthermore, although the evidence is clear that exercise is beneficial for LBP, it is not clear which modality of exercise is most suitable. There appears only one pertinent study addressing the interrelationships between exercise, weight loss and LBP. A multidisciplinary prospective cohort study of 46 obese patients with LBP showed that a 12-month medically supervised, non-surgical weight loss program involving meal replacement, caloric restriction, education, exercise, and group therapy could result in significant weight loss and improvements in LBP, disability and function [36••]. This same study also showed a small correlation between weight loss and LBP reduction. Admittedly, whether or not exercise and weight loss were the source of LBP improvements - as opposed to other specific components of the program - remains an open question. Nonetheless, the outcomes of the comprehensive program are promising.

Obesity, Systemic Inflammation, and Low Back Pain

Studies that investigate the role of obesity in initiating or exacerbating LBP are becoming increasingly prevalent. Recently, associations between obesity and inflammation, inflammation and pain signaling, and LBP and inflammation have

been postulated. Many pro- and anti-inflammatory mediators that stem from, interact with, and adversely affect adipose tissue and skeletal muscle have been studied in this context. Much of the focus has been placed upon C-reactive protein (CRP), tumor necrosis factor alpha (TNF-α), interleukin-6 (IL-6), and adiponectin due to the profusion of evidence and their connection to deleterious musculoskeletal conditions.

Adipose tissue accumulation, particularly in visceral compartments, leads to inflammation through an increase in pro-inflammatory cytokines and a decrease in anti-inflammatory adipokines [55]. Initiation of the inflammatory process comes from cellular stress at the level of the adipocyte [56]. Adipocytes undergo hyperplasia and hypertrophy, and together with circulating free fatty acids and their by-products, initiate an inflammatory signaling cascade. Inflamed adipocytes recruit circulating monocytes, leading to infiltration of the inflamed adipose tissue and differentiation into local macrophages. Accumulated macrophages contribute to inflammation by releasing pro-inflammatory cytokines. The degree of macrophage infiltration is correlated with the extent of adiposity [57].

CRP, a common marker of inflammation, is consistently found to be elevated in obese patients [58]. BMI and waist circumference are highly correlated to CRP serum levels [59•]. Increased plasma concentrations of CRP are attributable to the pathogenesis of atherosclerosis and diabetes [57, 60]. CRP is mainly produced in the liver in response to various pro-inflammatory cytokines, such as IL-6. Generally thought of as a marker of inflammation, CRP has also been shown to mediate inflammation by up-regulating pro-inflammatory cytokines and down-regulating adiponectin [61].

IL-6 is a cytokine that is released by adipose tissue and in skeletal muscle. IL-6 is primarily modulated by the transcription factor NF-κB, and is positively correlated with levels of obesity [62]. IL-6 has typically been associated with cardiovascular diseases, morbidity, and physical frailty. The idea that IL-6 is a pro-inflammatory cytokine has been perpetuated by the fact that it up-regulates CRP production and is present at high concentrations in the acute inflammatory state [63]. In addition, IL-6 may be involved in initiating a transition from acute inflammation to a chronic inflammatory state [64].

TNF-α is a pro-inflammatory cytokine that is pivotal to inflammation and insulin resistance in obesity. Its release is dependent on increased levels of circulating free fatty acids [65]. TNF-α is released by macrophages that have infiltrated adipose tissue. Specifically, TNF-α is produced as a result of activation of free fatty acid-mediated TLR4 and NF-κB in the macrophages [66]. TNF-α also reduces the expression of anti-inflammatory adipokines such as adiponectin [67], whereas adiponectin acts to inhibit NF-κB and TNF-α [68].

Adiponectin is a unique adipokine that exerts its anti-inflammatory effects in adipose tissue, in macrophages, and in skeletal muscle. Circulating adiponectin levels decrease with obesity and are present in higher concentrations in lean subjects [69]. Low levels of adiponectin are associated with increased morbidity and high levels of CRP and IL-6 [70].

In the obese state, macrophages accumulate in skeletal muscle. Macrophages and lipids that accumulate in skeletal muscle can cause the release of pro-inflammatory cytokines, which then contribute to systemic inflammation. There is evidence that pro-inflammatory cytokines released in skeletal muscle can lead to peripheral insulin resistance and impaired glucose control [71], which can then further exacerbate inflammation and lead to hyperalgesia [72, 73].

Noxious or even normally benign stimuli acting in inflamed tissue can cause or amplify pain due to decreased neural excitation thresholds [74]. It appears that pro- and anti-inflammatory cytokines play an important role in this pain modulation. Obese Zucker rats were shown to have an increased sensitivity to pain, while expression of adiponectin receptor mRNA and upregulated TNF-α mRNA were also identified in their spinal cords [75]. Other studies have further demonstrated that TNF-α and IL-6 are involved in pain signaling and hyperalgesia [76, 77].

Given these findings, LBP may be mediated or exacerbated by alterations to pain modulation through systemic inflammation. Previous studies have indicated elevated inflammatory mediators are found in herniated lumbar discs [78] and vertebral end-plate changes [79]. Only one study though has examined the interrelations among obesity, systemic inflammation, and LBP. Analysis of a population-based sample (n=15,322) drawn from the 1999 to 2004 National Health and Nutrition Examination Survey found that normal weight participants with elevated CRP had 1.74 greater odds of reporting LBP than participants with non-elevated CRP, while obese participants with elevated CRP had 2.87 greater odds of reporting LBP than obese participants with non-elevated CRP [80••]. This study also showed that participants with a greater waist circumference were more likely to report LBP. Given these findings, further investigation is warranted into effective treatment methodologies and modalities to negate the impact of systemic inflammation on LBP in obese patients.

Exercise, Weight Loss, Decreased Systemic Inflammation and Reduced LBP

Since obesity and LBP both have an association with systemic inflammation, the question arises as to whether a specific intervention exists that can potentially alleviate both chronic conditions simultaneously. The proposition put forward in this review is that exercise and weight loss can improve LBP in obese patients, in part, through the beneficial effects that regular exercise and reduced adiposity have on the systemic inflammatory state associated with obesity.

Exercise and weight loss can lead to decreased subcutaneous adipose tissue, and more importantly in the context of inflammation, decreased visceral adiposity. This is thought to occur via a decrease in adipocyte size, although the number of adipocytes in adipose tissue may also be altered [81]. Exercise programs that invoke weight loss have also been shown to reduce macrophage infiltration in adipose tissue [82]. Together, these changes within adipose tissue decrease expression of pro-inflammatory mediators and markers while increasing the expression of anti-inflammatory adipokines.

Exercise positively alters the level of pro- and anti-inflammatory mediators and markers in obese [83••] and sedentary [84] individuals, with those who engage in exercise generally having lower levels of CRP [85]. Weight loss can result in decreased CRP; larger reductions in weight can affect CRP levels more markedly [86]. Programs combining aerobic exercise, resistance training and weight loss have been particularly effective in down-regulating CRP [83••, 87]. Specifically, CRP was correlated with both aerobic capacity and weight loss independently [87]. In contrast, some exercise interventions without significant weight loss have shown that circulating CRP levels were not affected [88].

Studies examining the effect of exercise and weight loss on TNF-α have produced ambiguous results. TNF-α levels can be markedly reduced after a program of exercise and weight loss [70, 83••, 89]. However, some studies have indicated that weight loss alone can decrease TNF-α [90, 91], while others have shown that exercise and not weight loss can decrease TNF-α expression [92].

The effect of exercise on IL-6 is controversial because IL-6 may have pro- or anti-inflammatory functions depending on the target tissue and stimuli involved. Exercise studies have produced conflicting results because IL-6 increases during acute exercise [93], but decreases with regular exercise [70]. Weight loss effects on IL-6 have not produced clear results [94].

As with the pro-inflammatory cytokines and markers, studies of adiponectin, exercise, and weight loss in obese patients have produced variable results. Aerobic and resistance training produced more consistent increases in adiponectin if these programs also resulted in significant fat loss [69]. One study demonstrated that exercise with or without diet-induced weight loss increased adiponectin receptor expression, but only diet-induced weight loss produced an actual increase in serum adiponectin levels [95].

Regular exercise can improve local and systemic inflammation by reducing intramuscular adipose content in skeletal muscles [96•]. Additional benefits include glucose uptake, increased insulin sensitivity and down regulation of pro-inflammatory cytokine receptors [92]. Enhanced glucose control is relevant to improved pain modulation because hyperglycemia and insulin resistance have been shown to contribute to systemic inflammation [55] and hyperalgesia [73].

By reducing the degree of inflammation through exercise and weight loss, obese patients can potentially control the progression of their LBP. Despite the presence of conflicting studies about the importance of weight loss in comparison to exercise interventions, and whether a combination of the two is necessary in order to obtain an anti-inflammatory effect, there is sufficient evidence such that a simplified model has been put together to propose that exercise and weight loss can relieve LBP through changes to the pro-inflammatory environment (Fig. 1). Specifically, reduction in adipose tissue volume and conditioning of skeletal muscle can ultimately reduce the level of circulating pain mediating cytokines (i.e., Il-6 and TNF-α) and markers of inflammation (i.e., CRP), and increase the level of circulating anti-inflammatory adipokines (i.e., adiponectin)]. This reduction in systemic inflammation can subsequently decrease the extent of inflammatory insult on neurons and improve pain thresholds and pain modulation, therefore allowing for a more appropriate response to mechanical strain or benign stimuli.

Future Concepts and Theories to Explore

The presence of pain can negatively affect the success of weight loss programs [97]. In addition, interventional studies are often hampered by the non-specific nature of LBP. Given the multiple potential etiologies of LBP, there is likely significant variability in individual responses to any targeted treatments, reducing the power of any study and ultimately lessening the effectiveness of the treatment if applied too

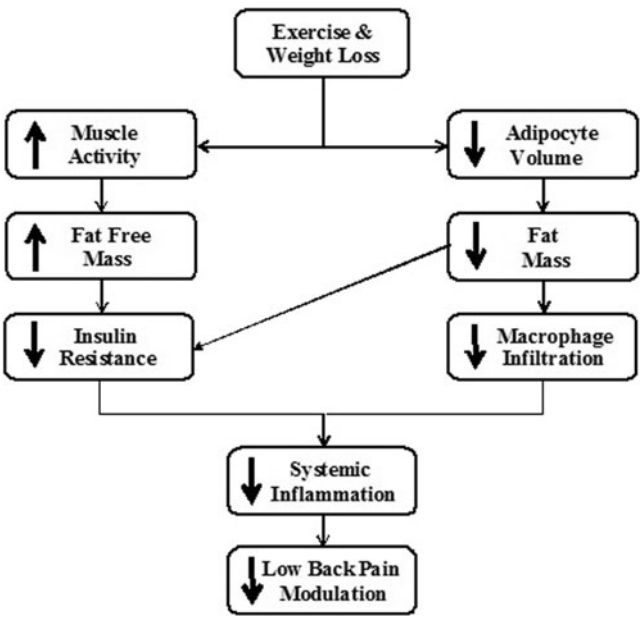

Fig. 1 A simplified model proposing how exercise and weight loss might reduce low back pain through physiological changes to the pro-inflammatory environment

broadly. Hence, the ability to accurately identify the cause of the LBP would greatly improve the chance of success for any future applications of treatment. A recent study suggests that an interrelationship might exist between obesity, inflammation, and LBP [80••]. In this review, the theory put forward is that obesity causes increased systemic inflammation, increased inflammation might contribute to increased LBP, and that exercise and weight loss may lead to decreased inflammation and reduced LBP. Looking further into the concept of obesity, inflammation and LBP has significant merit because trying to reduce inflammatory levels in obese individuals with LBP may be the link in the cycle between their pain and obesity. As discussed, one potential treatment plan to reduce systematic inflammation and LBP in obese patients is through the prescription of aerobic exercise (e.g., walking) or combination exercise (i.e., aerobic+resistance) and weight loss. Another method could be through pharmacological manipulation, such as vitamin-C supplementation. To date, research on inflammation and vitamin-C supplementation in obese individuals has been sparse [98, 99], and only one study has outlined a theory connecting vitamin-C and degenerative spinal conditions [100].

Conclusion

It is without question that obesity and LBP are responsible for numerous comorbidities that pose a significant challenge to health care systems worldwide. The literature suggests a positive relationship exists between obesity and LBP. However, there is a shortage of studies discussing the possible pathways in which obesity leads to LBP, or whether interventions that include exercise and weight loss are a viable treatment for LBP. Given the complexities of these medical conditions, and the myriad of possible mechanisms involved, some spinal clinicians postulate that this relationship may be purely coincidental. Recent evidence suggests that systemic inflammation associated with obesity may provide a link with LBP. Despite the clinical equipoise, it is a promising area in which to identify a cohort of patients that would benefit from exercise-based weight loss interventions to address this theory. Overall, despite the current lack of evidence in regards to exercise, weight loss and reduction in LBP in obese patients, it is important to remember that regular exercise and weight loss is of benefit to other aspects of health and well-being aside from a potential reduction in LBP.

Compliance with Ethics Guidelines

Conflict of Interest Darren M. Roffey is a consultant for Palladian Health.

Adele Budiansky declares that she has no conflict of interest.

Matthew J. Coyle declares that he has no conflict of interest.

Eugene K. Wai declares that he has no conflict of interest.

Human and Animal Rights and Informed Consent This article does not contain any studies with human or animal subjects performed by any of the authors.

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- • Of importance
- •• Of major importance

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