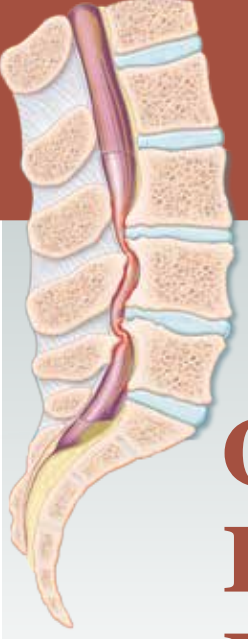

Obesity, Weight Loss, and Low Back Pain: An Overview for Primary Care Providers—Part 1

ABSTRACT

Recognizing that the increasing incidence of obesity coincides with the rising prevalence of LBP, there is growing interest in establishing the relationship between over-weight and back pain. It is likely that any association is multi-factorial and that the connection is not as mechanistically simple as previously believed. Systemic inflammation associated with obesity may be an important contributor. Proposed treatment options vary from cognitive behavioural therapy to bariatric surgery with none yet fully proven. Despite the ambiguity, it appears prudent for primary care providers treating obese patients with LBP to recommend weight loss and exercise.

KEYWORDS: Obesity, low back pain, inflammation, intervertebral disc, multi-factorial, causality, association

CME Pre-test Quiz

Editor's comment:

Both obesity and low back pain are significant health risks and impose substantial financial burdens on society. The next two issues of *Back Health* are devoted to exploring the relationship between being overweight and having back pain. Dr. Roffey and his co-authors have produced a comprehensive review of the subject covering the putative associations and mechanisms and research on the relevance of weight loss in managing back pain. In Part 2 they turn their attention to evidence-based treatment recommendations.

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Overview of the Relationship between Obesity and LBP

In the "Global Burden of Disease 2010" study by the Institute for Health Metrics and Evaluation, low back pain (LBP) was found to cause more disability than any other health condition, affecting nearly 10% of the global population at any given time and being responsible for over 10% of all life years lived with disability.[1,2] Further cause for concern for LBP is its increasingly acknowledged association with obesity. Ten percent of the global population was considered obese (i.e. body mass index (BMI) >30 kg/m^2) in 2005 and 23% were considered overweight (i.e. BMI 25-30 kg/m^2), and the number of obese and overweight individuals is expected to increase 45% by 2030.[3]

Although the etiology of LBP is not fully understood and is likely multi-factorial, involving an interaction between physical, occupational, psychosocial, and economic risk factors, it is believed that obesity may play a role in its onset and continual degradation. A systematic review in 2000 identified 65 studies on obesity and LBP and found that 32% reported a statistically significant association.[4] However, no evidence was found to support criteria required for causation, including temporality, reversibility, and consistency, suggesting that obesity was a potential risk factor for LBP, but could not be considered a cause.[4] In 2009 a meta-analysis of 33 studies on obesity and LBP reported statistically significant odds ratios between obesity and the incidence of LBP, the 12-month prevalence of LBP, care seeking for LBP, and chronic LBP.[5] Authors concluded that while the relationship between obesity and LBP may be causal, it could also be bi-directional (i.e. obesity can cause LBP; or LBP can cause inactivity, leading to obesity). Furthermore, statistically significant odds ratios have been reported between obesity and sciatica, hospitalization for sciatica, and surgery for sciatica, suggesting that obesity impacts health services utilization for LBP.[6]

Numerous potential mechanisms have been offered to explain the relationship between obesity and LBP. These include: increased body mass creating higher compressive or shearing forces on intervertebral discs leading to accelerated intervertebral disc degeneration or vertebral endplate changes; obese individuals having an increased overall risk of traumatic injury; intervertebral disc degeneration attributable to cumulative or repetitive loading; and inadequate intervertebral disc nutrition due to insufficient movement or atherosclerosis reducing intervertebral disc blood supply.[5] Other as yet unproven hypotheses include ideas that obesity and LBP

are linked via systemic inflammation (i.e. mediators secreted by adipocytes) or metabolic changes attributable to the amount of body adipose tissue.[3]

MORE RECENT STUDIES USING ADVANCED IMAGING, SUCH AS MRI SCANS, HAVE DEMONSTRATED ACCELERATED DISC DEGENERATION IN OBESE AND OVERWEIGHT INDIVIDUALS.

One recent study attempted to elucidate the interaction between obesity, LBP, and physical activity using data from the National Health and Nutrition Examination Survey (NHANES), on a nationally representative sample of adults in the United States.[7] The study found that while the risk of reporting LBP was markedly higher with increasing BMI (e.g. LBP prevalence was 2.9% with BMI <25 kg/m^2 vs. 11.6% for BMI >35 kg/m^2), this risk appeared to be mitigated by the amount of time spent engaging in moderate physical activity (e.g. brisk walking, dancing, gardening).[7] Authors concluded that while both obesity and reduced physical activity appeared to be independent risk factors for LBP, overweight and obese individuals could markedly reduce their risk of LBP by engaging in additional physical activity, even if not accompanied by weight loss.

The purpose of this two part article is to help primary care physicians understand the various pathways through which obesity may impact LBP, review studies assessing the efficacy of weight loss to manage symptoms of LBP, summarize evidence-based recommendations to manage obesity through weight loss and exercise, and discuss the impact of obesity on outcomes when spine surgery is performed for LBP.

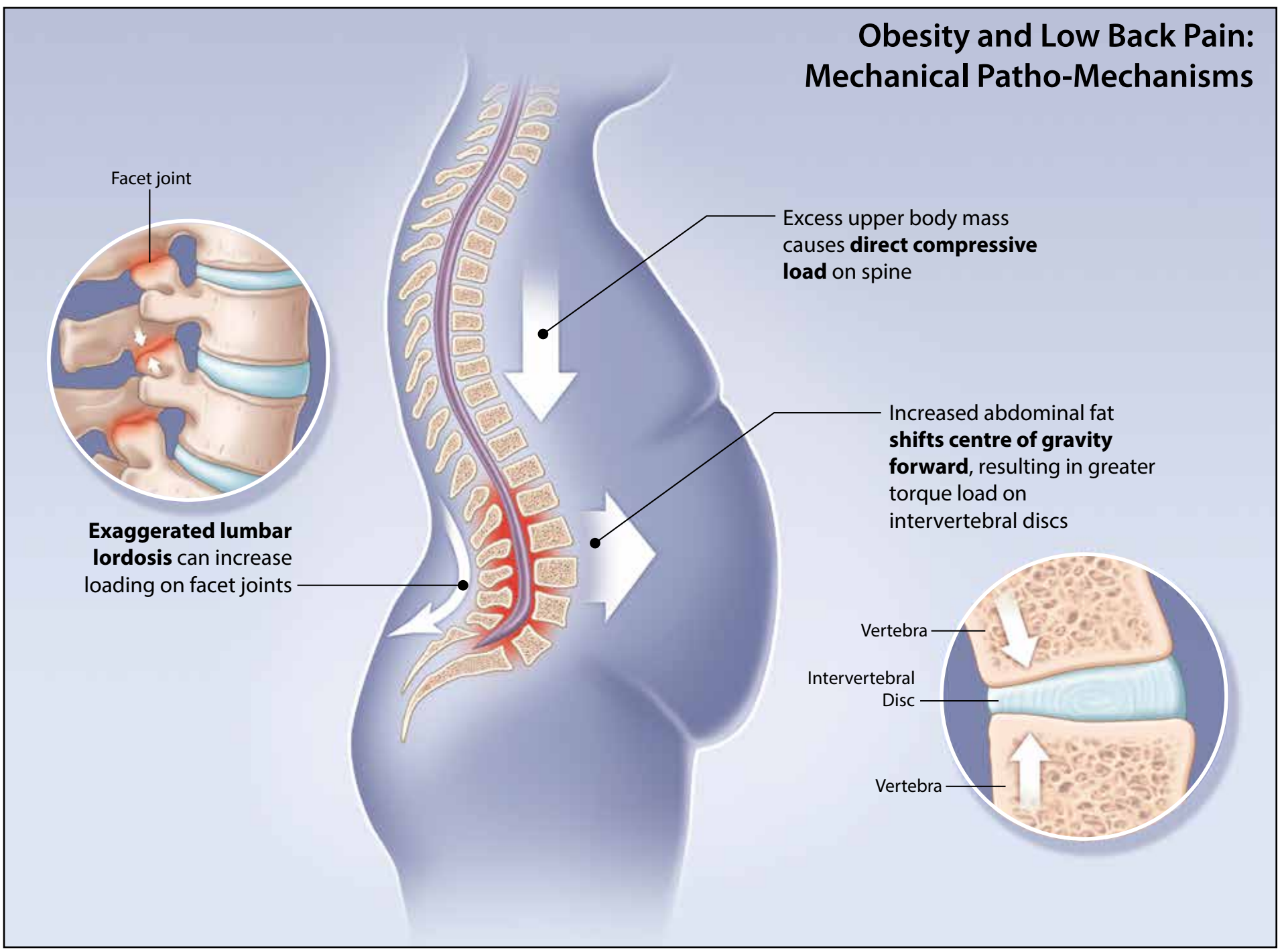
Patho-Mechanisms of Obesity and LBP

Authors to date have focused on trying to demonstrate a relationship between obesity and LBP.[4-6] The multi-factorial nature of both obesity and LBP likely explains some of the conflicting results. A better understanding of the causal mechanisms of back pain may help treatment options; however, high-quality studies that explore any causal effects between obesity and LBP are lacking.[8] A review of the literature has identified a number of studies evaluating possible patho-mechanisms,[9] which can be divided into two broad categories: 1) mechanical, and 2) inflammatory.

Mechanical

There is a general consensus among many spine clinicians that

increased body weight leads to an increased strain on the spinal column, resulting in higher muscular strain and accelerated spinal column degeneration (e.g. intervertebral discs disease). Besides the excess upper body mass causing a direct compressive load on the spine, other biomechanical factors may further exacerbate the load on the lumbar spine. Increased abdominal fat shifts the centre of gravity of the body in a forward direction, resulting in greater torque load on the intervertebral discs. As a consequence, an obese individual may compensate by exaggerating their lumbar lordosis, which in-turn increases the loading on the facet joints. Furthermore, obese individuals tend to have disproportionate muscle mass and with an associated increased core strength and endurance relative to their higher weight.[10,11] Over time, this will lead to altered kinematics during activities, which may have detrimental compensatory actions and increase the strain on the vertebral structures.

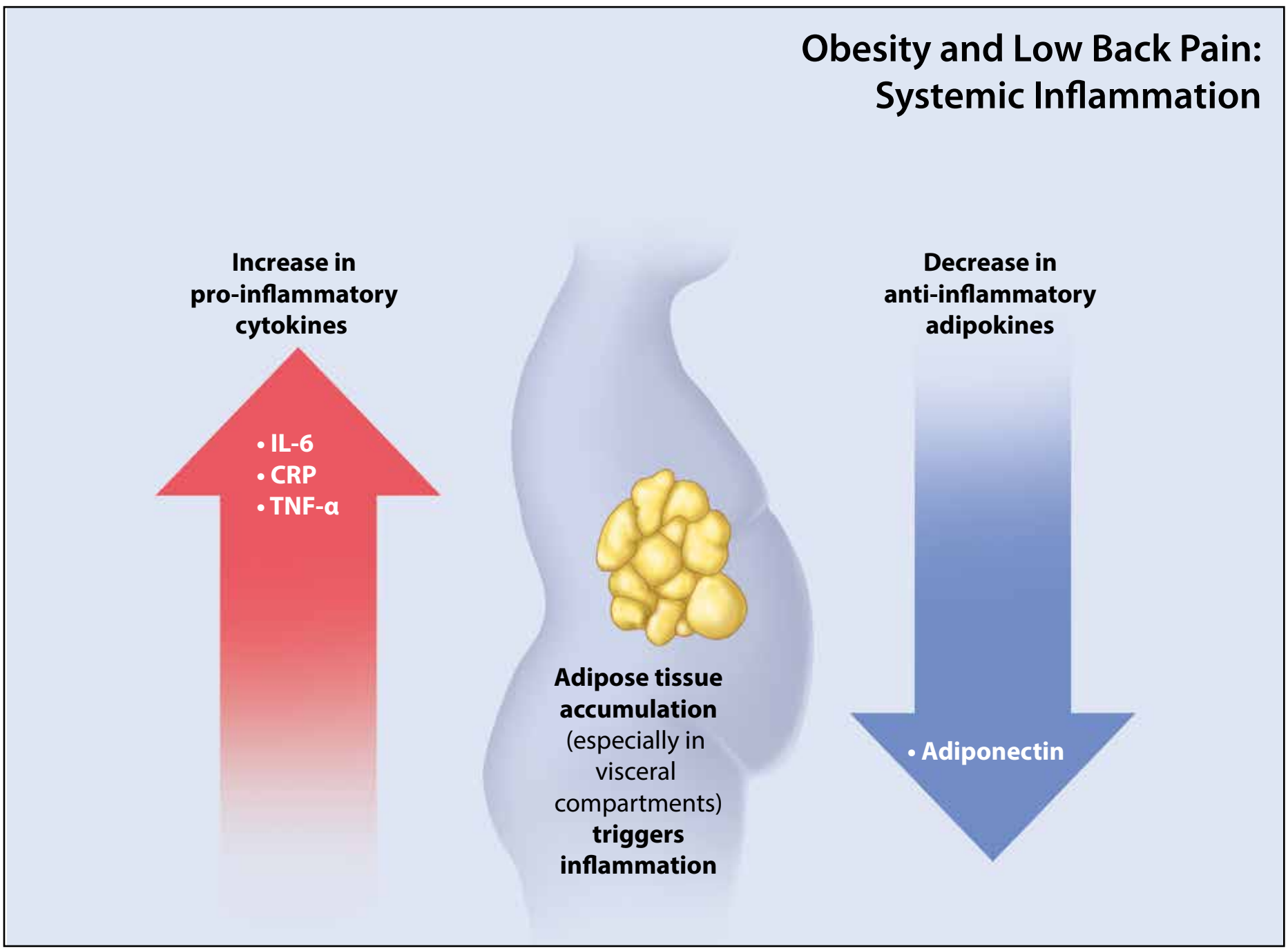


Studies correlating LBP with X-rays have generally had conflicting results. However, more recent studies using advanced imaging, such as MRI scans, have demonstrated accelerated disc degeneration in obese and overweight individuals.[12-15] Facet joint arthritis was also associated with increased abdominal fat[16] and obesity.[17]

Systemic Inflammation

Recently, associations between obesity and inflammation, inflammation and pain signalling, and LBP and inflammation have been postulated.[3,9] In this context many pro- and anti-inflammatory mediators that either stem from or interact with adipose tissue and adversely affect both body fat and skeletal muscle have been studied.

Adipose tissue accumulation, particularly in visceral compartments, has been demonstrated to trigger inflammation through an increase in proinflammatory cytokines and a decrease in anti-inflammatory adipokines.[18] Initiation of the inflammatory process comes from cellular stress at the



level of the adipocyte.[19] As a result the adipocytes undergo hyperplasia and hypertrophy, and together with circulating free fatty acids and their by-products, initiate an inflammatory signalling cascade.

IL-6 is a cytokine that is released by adipose tissue and skeletal muscle, and influences production of C reactive protein (CRP). TNF-α is a pro-inflammatory cytokine that is pivotal to inflammation and insulin resistance in obesity. As such, it is not surprising that CRP, IL-6 and TNF-α have been shown to be consistently elevated in obese patients[20,21] and are highly correlated to BMI and waist circumference.[22] 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.[23]

There is evidence that pro-inflammatory cytokines released in skeletal muscle can lead to peripheral insulin resistance and impaired glucose control,[24] which can then further exacerbate inflammation and lead to hyperalgesia.[25,26] Noxious or even normally benign stimuli acting in inflamed tissue can cause or amplify pain due to decreased neural excitation thresholds.[27] Other studies have further demonstrated that TNF-α and IL-6 are involved in pain signalling and hyperalgesia.[28,29] It appears that pro- and anti-inflammatory cytokines play an important role in this pain modulation.

Specific to LBP, previous studies have identified elevated inflammatory mediators in herniated lumbar discs[30] and vertebral endplate degenerative changes.[31] Analysis of NHANES data (n=15,322) 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.[32]

The inter-relationship between adipose cells, skeletal muscle and pro- and anti-inflammatory mediators may help explain the beneficial effects of exercise on LBP in the setting of obesity.

Evidence for Weight Loss Interventions in Treating Low Back Pain

Lifestyle modification is an integral component for non-surgical management of LBP in almost every population. To that end, obese individuals presenting with LBP at primary and tertiary care clinics are commonly advised to lose weight in order to help alleviate their symptomatology. Despite this, a 2008 systematic review failed to uncover any studies to support the efficacy of non-surgical weight loss programs to improve LBP.[33] There is a paucity of studies on this topic in the literature; only a few studies

SUMMARY OF KEY POINTS

Epidemiological studies have noted a significant association between obesity and low back pain.

Besides the increased mechanical strain, excessive adipose tissues can promote systematic inflammation which may be an important contributor to low back pain.

The relationship between obesity and low back pain likely involves an interaction between physical, occupational, psychosocial and economic risk factors.

Only a handful of studies have evaluated weight loss as an intervention to treat low back pain. Further research is required.

have investigated the relationship between a change in weight or BMI and improvement of LBP and disability after non-surgical or surgical intervention.

Non-Surgical Weight Loss

A pilot prospective cohort of 46 patients with an average BMI of 44.7 kg/m^2 underwent a medically supervised multidisciplinary approach towards the nonsurgical lifestyle modification management of weight loss and its effect on LBP.[34] The weight loss program entailed meal replacement, caloric restriction education, exercise, and group therapy. A total of 61% of the patients completed the 52-week program, and the results revealed that a reduction in BMI was significantly associated with clinically important improvements in the ODI.[34] In contrast, a previous randomized control trial which was designed to assess the effects of a lifestyle-modification intervention for hypertension (when applied to patients with LBP) demonstrated a decrease in weight and BMI without a reduction in the prevalence of LBP.[35] Furthermore, a retrospective multicentre study involving 128 patients with chronic LBP was designed to investigate the relationship between BMI changes in pain and disability after an exercise-based chronic LBP treatment regime.[36] Authors concluded that there was no significant relationship that existed between BMI changes post non-surgical intervention and pain and disability as measured by the VAS and ODI respectively. It is important to note that methodological issues, such as limited power or sensitivity of instruments, may explain some of the conflicting results. As such, further research in this area is still warranted.

Surgical Weight Loss

Bariatric surgery is a treatment option that consistently results in sustained weight loss. It may be the last remaining option for patients with musculoskeletal limitations

that prevent them from participating in a daily exercise program.[37] Despite conflicting evidence with respect to non-surgical weight loss and LBP, an increasing body of evidence has illustrated the positive association 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 up to 12-months after surgery significantly decreased axial and radicular LBP.[38] A prospective comparative study of 25 patients demonstrated that an average weight loss of 22 kg up to three months after surgery led to a 54% decrease in the severity of LBP and a significant improvement in quality of life.[39] Similarly, a prospective study involving 20 patients revealed an average weight loss of 24 kg up to 3-months after surgery, and that the Numeric Pain Rating Scale demonstrated a significant decrease in LBP.[40] Another prospective assessment of 38 patients showed an average decrease in mean weight of 39 kg up to 12-months post-operation, with a statistically significant mean 44% decrease in axial LBP on the VAS.[41] Combined, these studies show promise in surgical weight loss as a treatment for LBP. The decision to undergo bariatric surgery requires extensive consultation and careful patient selection to ensure that those with the greatest predicted benefit undergo the operation (Table 1).

Table 1: Summary of Evidence for/against Bariatric Surgery

	Bariatric Surgery		
Indications[19,34]	– BMI ≥30 kg/m^2 with serious co-existing medical conditions – BMI ≥35 kg/m^2 with concomitant obesity-related conditions – BMI ≥40 kg/m^2		
Types of Complications[35,36]	– Malabsorption – Gastric dumping syndrome – Nutritional deficiencies – High reoperation rates		
Financial Concerns[34]	– More inpatient hospital resource use – More non-primary out-patient care during follow-up		
Types of Procedures	**Gastric Bypass**	**Sleeve Gastrectomy**	**Gastric Banding**
Frequency of Complications[37]	9.7 %	6.5 %	5.4 %
Harms: Benefits[38]	High risk: Most benefit	Moderate risk: Moderate effectiveness	Low risk: Least effective

CLINICAL PEARLS

Patients presenting with low back pain are often deconditioned and carrying excessive weight. Education on the possible relationship between obesity and low back pain may help motivate towards achieving a healthier lifestyle.

Dramatic physiological changes are not necessarily required; even mild to moderate weight loss may improve low back pain symptomatology.

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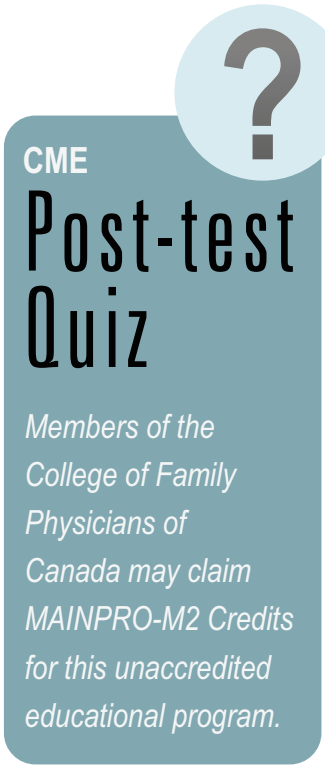

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