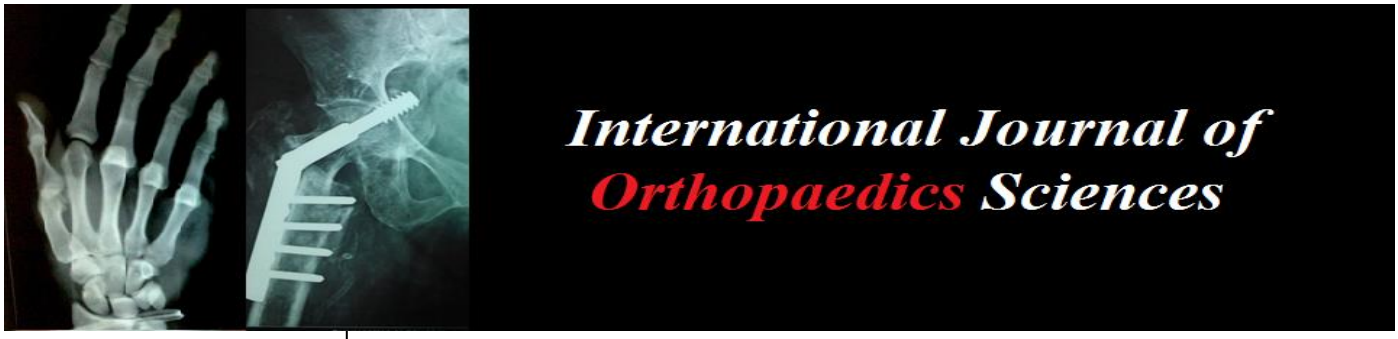

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The association of lumbar intervertebral disc degeneration and lumbar spinal stenosis on magnetic resonance imaging with body mass index in overweight and obese adults in Indian population

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Abstract
Aims: To investigate the association of being overweight or obese with the presence, extent, and severity of lumbar disc degeneration and lumbar spinal stenosis on magnetic resonance imaging (MRI) in adults in the Indian population.
Study Design: Level 4, Cross-sectional study
Place and duration of study: Department of Orthopaedics, SVP Hospital, Ahmedabad between February 2019 and October 2019.
Methodology: A population-based cross-sectional study of 500 Indian volunteers was conducted. Subjects underwent radiographic and clinical assessment, and weight and height were measured. T2-weighted MRIs of the lumbar spine were obtained. The presence, extent, and severity of disc degeneration and additional radiographic and clinical parameters were assessed. Asian-modified body mass index (BMI) (kg/m^2) categories were used.
Results: The study included 305 men and 195 women (mean age 50.59 years). Disc degeneration was noted in 450 (90%) subjects. BMI was significantly higher in subjects with disc degeneration (mean 29.18 kg/m2) than in subjects without degeneration (mean 23.66 kg/m2). A significant increase in the number of degenerated levels, global severity of disc degeneration, and end-stage disc degeneration with lumbar spinal stenosis was noted with elevated BMI, in particular in overweight and obese subjects. End-stage disc degeneration with disc space narrowing was significantly more pronounced in obese subject.
Conclusion: Our findings, to systematically assess lumbar disc degeneration and lumbar spinal stenosis on MRI, indicated a significant association between the presence, extent, and global severity of disc degeneration and lumbar spinal stenosis with BMI (>23 kg/m^2) in overweight and obese adults.

Keywords: BMI, lumbar intervertebral disc degeneration, magnetic resonance imaging, cross-sectional study

Introduction
Low back pain is a common debilitating condition worldwide with severe socioeconomic and health care consequences [1, 2]. Low back pain can cause functional impairment, diminished quality of life, loss of working ability, potential psychological distress, and increased health care costs [1, 3]. Although various factors have been implicated in low back pain, intervertebral disc degeneration that is evident radiologically or on advanced imaging (i.e., magnetic resonance imaging [MRI]) is a known cause of low back pain [4, 13]. For many years, a strong emphasis has been placed on identifying risk factors associated with disc degeneration and lumbar spinal stenosis in the lumbar spine in adults. Age, abnormal physical loading [15], and environmental [16, 17], hormonal [18], systemic [19, 21] and genetic influences [16, 22, 25] have been suggested to contribute to the development of disc degeneration and lumbar spinal canal stenosis. Some studies have suggested that elevated body mass index (BMI), in particular being overweight or obese, may be related to disc degeneration [17, 26, 27] and LSS, but such an association remains largely a matter of speculation to date, with several studies providing strong evidence against such a link [6, 28, 29].

In a recent systematic review of MRI studies assessing disc degeneration and low back pain, Chou *et al.* [5] found that disc degeneration is a significant risk factor for the development of such pain. In fact, previous studies have also shown that an increase in the global severity of disc degeneration or the presence of end-stage disc degeneration with lumbar canal stenosis is significantly associated with an increased risk of having low back pain [6, 10, 14]. These inconsistencies can be attributed primarily to the lack of large epidemiologic studies and proper study design, patient-based studies, insufficient statistical analyses, the mode of radiographic/imaging assessment used in defining the phenotype of disc degeneration, and/or conjecture arising from limited radiographic interpretation of additional spinal findings (e.g., Schmorl's nodes) that may contribute to the degenerative process [6, 17, 26, 28, 30]. Increasing obesity is a serious public health problem worldwide. Furthermore, the associations between being overweight or obese and the extent of disc degeneration (i.e., the number of lumbar levels with disc degeneration) and the severity of disc degeneration of the lumbar spine remain unknown because previous studies have failed to quantitatively assess such parameters on advanced imaging. So far, the mechanisms underlying the association between obesity and LBP are not fully known. Obesity has been shown to increase the risk of cardiovascular disease and diabetes [31, 33], osteoarthritis [34, 35], and spine diseases [36, 37]. It has been shown that the adverse effects of excess weight tend to be delayed, sometimes for a decade or longer [38]. Obesity increases the risk of

LBP, for example, because of lumbar disc disorders [16, 18], through mechanical load. It has been suggested that mechanical load is the principal factor initiating the degenerative process in the lumbar spine [19]. Furthermore, besides direct biomechanical effect on cartilage and skeleton, indirect effects by changes in body mass can be mediated by mechanoreceptors, cytokines, and growth factors. These factors have the potential to alter the properties of bone matrix, ligamentum flavum, synovium, and cartilage, all of which could promote the development of osteoarthritis, hypertrophy of the ligamentum flavum, and disc degeneration. Decreased muscle mass is also associated with insulin resistance, which further weakens the skeletal muscles and promotes systemic inflammation. In addition to mechanical load, obesity may cause LBP via low-grade systemic inflammation [16, 18, 20, 21]. It is well known that adipose tissue is metabolically active and produces adipokines (i.e. adiponectin, resistin and leptin), macrophage-derived factors (i.e., interleukin-1 [IL-1]), or pro-inflammatory cytokines and chemokines (i.e. CRP, tumor necrosis factor and IL-6). Another potential mechanism may be vascular insufficiency to the vertebrae and subsequently to the disc, brought on by atherosclerosis or high serum lipid levels that can affect nutrient and metabolite transport into the disc. Other possible mechanisms could involve a metabolic disorder or gene-environment interaction effects. Leptin, in addition to affecting energy balance, stimulates the synthesis of pro-inflammatory cytokines and nitric oxide; that is, it is directly linked to pain modulation. An association between C-reactive protein-a marker of systemic inflammation and sciatica has been shown in a few case-control studies [24], whereas studies on the association between C-reactive protein and LBP are sparser [25]. It would be particularly valuable to address the association between obesity and LBP in relation to inflammatory factors in a population-based study. Clinical and experimental studies have shown not only mechanical but also metabolic obesity-specific pathways for the development of facet joint osteoarthritis, disc degeneration and hypertrophy of spinal ligaments [39, 45]. These spondylotic changes narrow the spinal canal, which can progress to lumbar spinal stenosis (LSS), a condition associated with a health burden and impaired quality of life compared with stroke, cardiovascular diseases, and diabetes. Common symptoms include leg pain, especially during walking, associated with numbness and paresthesia and sometimes loss of motor control and bladder disturbances.

The numbers of overweight and obese individuals are of global concern and the prevalence continues to rise in many populations [46, 47]. Overall 39% and 13% of the world's adult population are overweight and obese, respectively [47], and it is expected that such rates will increase exponentially by the year 2030 if left unabated. In fact, based on epidemiologic trends seen in the past (e.g., bubonic plague, pneumonia and infections, cardiovascular disease and cancer, etc), society has entered the obesity phase [46]. In India, it has been estimated that 1 of 3 children are obese, and the likelihood that excess body weight will remain and even lead to more severe obesity in adulthood is high [48]. Several European countries (e.g., the UK, Germany, and Croatia) have reported that 60% of their population is overweight. Although it was previously believed that the numbers of overweight and obese individuals did not represent a public health concern among Asian countries because of low prevalence, India, the world's second-most populous nation and one of the largest economy, has seen an increase in the numbers of overweight and obese residents due to its financial affluence, the rise of the fast-food culture, and the adoption of more Westernized lifestyles, which has affected both urban and rural communities[34]. According to ICMR-INDIAN study 2015, the prevalence rate of obesity and central obesity varies from 11.8% to 31.3% and 16.9% to 36.3% respectively. Although body weight is associated with the development of cardiovascular disease, diabetes, and malignancies among other conditions, its effects on disc degeneration and lumbar canal diameter, as mentioned above, have remained elusive. Since being overweight, and particularly, obese, have been associated with low back pain[50] and since disc degeneration on MRI is a factor related to low back pain [8, 10, 11], it would appear reasonable to hypothesize that elevated BMI may be instrumental in the development of disc degeneration and increased risk of lumbar canal stenosis. Since MRI is widely regarded as the gold standard of imaging modalities to assess disc degeneration and lumbar canal diameter and because obese individuals are at increased risk to develop osteoarthritis in both loaded and unloaded joints, we hypothesized that an elevated body mass index (BMI) that indicates being overweight or obese will also increase the rate of clinically manifested LSS in the presence, extent, and severity of lumbar disc degeneration in Indian adults.

Anatomy and Physiology of the Intervertebral Disk

The intervertebral disks separate the vertebral bodies to facilitate load transmission and multiaxial flexibility while playing the role of "shock absorber" in response to dynamic spinal compression. Moreover, the disk acts as a "spacer" by providing height to the spinal column, allowing passage of nerves through the intervertebral foramen and facilitates biomechanical synergy with the posterior facet joints. The intervertebral disk consists of an inner gelatinous core (nucleus pulposus) and a thick outer ring of fibrous cartilage (Annulus fibrosus). The nucleus pulposus is mainly composed

of a proteoglycan and type II collagen in a ratio of 20:1. The annulus fibrosus serves as an intervertebral ligament composed of up to 25 concentric collagen lamellae that provide bending and shear stiffness/strength. The nucleus is separated from the adjacent vertebra by the endplate, which is a thin bilayer of cartilage and porous subchondral bone. Early degenerative processes include enzymatic degradation of nucleus proteoglycans that leads to decreased swelling and reduced disk water. These changes are brought upon by age progression and excessive physical loading, which adversely affect disk biomechanics leading to altered tissue stress distributions and biological activities that in turn cause nuclear fibrosis and disorganization of the annular architecture. Biochemical changes of the extracellular matrix and damage accumulation at the disk periphery trigger inflammatory cellular responses that promote a cascade of further structural modifications that progressively compromise disk biomechanics. Furthermore, the progression of disk degeneration may also be associated with an endplate and subchondral bone edema (e.g., Modic changes) that can further contribute to the development of pain. In fact, overweight/obesity has also been implicated with endplate and Modic changes in the lumbar spine.

2. Material and Methods

2.1 Study population. A population-based cohort study was initiated to assess the phenotype of disc degeneration (4, 11, 23, 25, 51-54). Western Indian subjects were recruited between February 2019 - October 2019 who visited our OPD in Department of Orthopaedics, SVP Hospital, and Ahmedabad. The invitation to participate did not discriminate with regard to social or economic demographics. Participants were not recruited based on the presence or absence of low back pain. A cross-sectional design was used to assess individuals who were of range 20–85 years years of age. The participants did not have infections, inflammation, neoplasms, deformities, or previous surgery of the lumbar spine. None of the participants were related.

2.2 Assessment of radiographs. Each participant underwent T2-weighted MRI of the lumbar spine. The technical parameters of the imaging protocol have been described previously. The presence and severity of disc degeneration were assessed using the criteria of Schneiderman *et al.* [54] (Table 1), in which higher scores indicate increased severity.

Table 1: MRI criteria for Disc Degeneration

Grade	Description
Normal	No signal changes
1	Slight decrease in signal intensity of the nucleus pulposus
2	Hypointense nucleus pulposus with normal disc height
3	Hypointense nucleus pulposus with disc space narrowing

*MRI criteria for disc degeneration based on T2-weighted sagittal MRI of the lumbar spine as described by Schneiderman et al.[48].

Disc degeneration of the lumbar spine was considered to be present if any level exhibited a degenerative disc disease score of 1. The presence of and the number of levels with end-stage disc degeneration with disc space narrowing (i.e., a degenerative disc disease score of 3 at any lumbar level) was noted. The extent of disc degeneration was defined as the number of lumbar segments affected and multilevel involvement (i.e., 2 levels). The presence and severity of disc herniation (i.e., bulge and extrusion), lumbar disc diameter, Schmorl's nodes (i.e., focal morphologic abnormality or defect of the vertebral endplate), high intensity zones of the disc, and vertebral marrow changes (i.e., high signal intensity of the vertebral body) were noted.

Clinical assessment. Age, sex, physical activity level (exercising <2 days per week versus >2 days per week), history of lumbar injury (i.e., an event that led to low back pain), history of smoking, occupation, weight (kg), and height (meters) were obtained for all participants. Objective weight (digital recording) and height data were obtained and recorded at a single institute consistently for each participant. Subjects removed shoes, excess bulky clothing (such as a jacket), and objects of weight (such as a purse, wallet, or metallic objects) before weight and height measurements were obtained. Height and weight measurements were assessed to the nearest hundredth decimal place and were noted at the time the subject underwent MRI. BMI (kg/m^2) categories modified for Asian populations based on World Health Organization guidelines were used [56]. Individuals with a BMI of 18.5 kg/m^2 were classified as underweight, those with a BMI of 18.5–23.0 kg/m^2 as normal, those with a BMI of 23.0–27.5 kg/m^2 as overweight, and those with a BMI of 27.5 kg/m^2 as obese. This classification scheme was used instead of the standard scheme used in the US or Europe (i.e., 18.5 kg/m^2 as underweight, 18.5–25 kg/m^2 as normal, 25–30 kg/m^2 as overweight, and 30 kg/m^2 as obese) to account for ethnic variations in body fat distribution and disease risk, which differ between Asians and Caucasians.

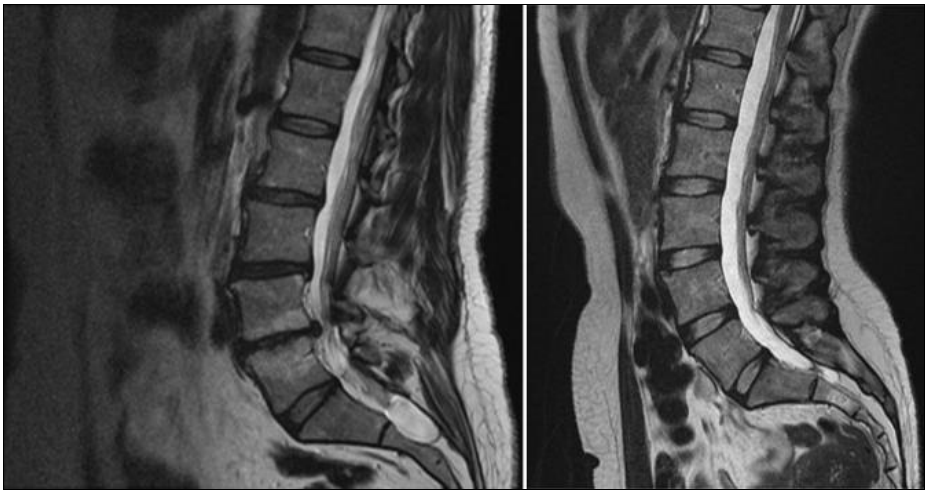

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Fig 1: Sagittal T2-weighted magnetic resonance imaging of the lumbar spine. (A) Obese subject with disk degeneration and bulging of L2–L5, with disk space narrowing, endplate irregularities, and Modic changes at L4–L5. Also, note the presence of a sacral cyst. (B) A normal-weight individual with non-degenerated lumbar disks.

3. Results and Discussion

The study included 305 men and 195 women, with a mean age of 50.50 years (range 20–85 years). Disc degeneration was present in 450 (90%) of the subjects. Disc degeneration was seen at L1–L2 in 29 (5.8%) of the subjects, at L2–L3 in 67 (13.4%) of the subjects, at L3–L4 in 188 (37.6%) of the subjects, at L4–L5 in 358 (71.6%) of the subjects, and at L5–S1 in 212 (42.4%) of the subjects. Women had a significantly higher prevalence of disc degeneration than men (82.56% in men versus 94.75% in women). As expected, the prevalence of disc degeneration was found to increase with older age. Of the study subjects, 0.2% were underweight, 7.4% were of normal weight, 38.2% were overweight, and 54.2% were obese. The mean BMI was 28.68 kg/m^2 (range 17.5– 55.56 kg/m^2). BMI was significantly higher in subjects with disc degeneration (mean 29.18 kg/m^2 [range 19.4–55.56 kg/m^2]) than in subjects without disc degeneration (mean 23.66 kg/m^2 [range 17.5–27.18 kg/m^2]). Multilevel disc degeneration was noted in 0%, 52.6%, 58.5%, and 63.83% of underweight, normal weight, overweight, and obese subjects, respectively. Individuals who were overweight and those who were obese had an increased likelihood of multilevel disc degeneration involvement. We also observed an almost linear positive association between BMI and Average Lumbar Spinal Canal Diameter (Figure 2), that is, the lowest rates of LSS were found in lean individuals and the highest in obese individuals.

Table 2: Association between Body Mass Index (BMI) and Average Lumbar Spinal Canal Diameter

Body Mass Index (kg/m^2)	Average Lumbar Spinal Canal Diameter (mm)					Average Lumbar Spinal Canal Diameter(mm)
	L1-L2	L2-L3	L3-L4	L4-L5	L5-S1	
Underweight (<18.5)	18	17	16	15	14	16
Normal (18.5-23)	17.6	16.4	14.8	13.6	11.7	14.8
Overweight (23-27.5)	17.2	15.7	14.4	12.8	11.3	14.3
Obese (>27.5)	15.2	14.1	12.4	11.7	11.0	12.9

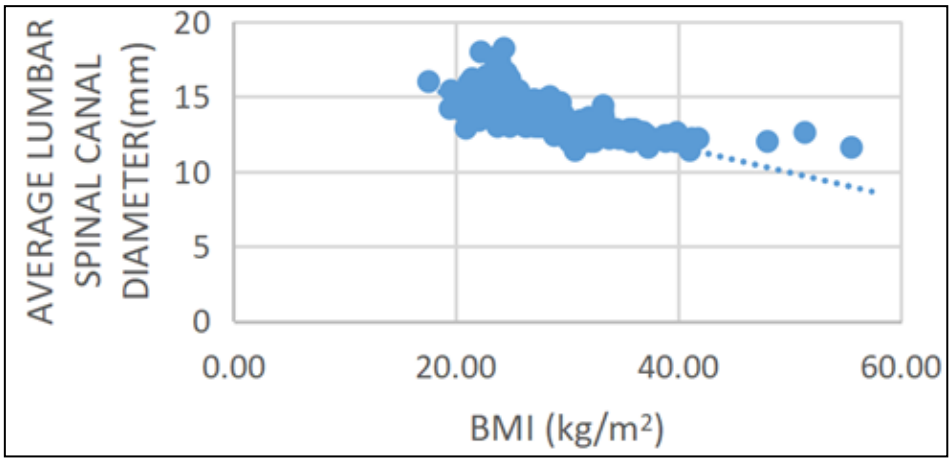

Fig 2: The association between body Mass index (BMI) and Average Spinal Lumbar Canal Diameter displayed as a scatter diagram with the trendline.

End-stage disc degeneration and disc space narrowing (Schneiderman grade 3) was noted in 371 (74.2%) of the subjects and at L1-L2 level in 20 (4%) subjects, at L2-L3 level in 45 (9%) subjects, at L3-L4 level in 126 (25.2%) subjects, at L4-L5 level in 194 (38.8%) subjects, at L5-S1 level in 136 (27.2%) subjects.

Table 3: Disc Degeneration Grading (ACC to Schneiderman *et al.*) at various Lumbar Disc Levels

Disc Degeneration Grade (Schneiderman *et al.*)	Number Of Patients				
	L1-L2 Disc Level	L2-L3 Disc Level	L3-L4 Disc Level	L4-L5 Disc Level	L5-S1 Disc Level
Normal	**50**				
1	1 (0.2%)	7 (1.4%)	18 (3.6%)	64 (12.8%)	27 (5.4%)
2	8 (1.6%)	15 (3%)	44 (8.8%)	100 (20%)	49 (9.8%)
3	20 (4%)	45 (9%)	126 (25.2%)	194 (38.8%)	136 (27.2%)
Total No.	29	67	188	358	212

A significant increase in the number of degenerated levels, global severity of disc degeneration, and end-stage disc degeneration (Schneiderman Grade 3) was also noted with elevated BMI, in particular in overweight and obese subjects. End-stage disc degeneration was significantly more pronounced in obese subjects.

Table 4: Association between Body Mass Index (BMI) and Disc Degeneration Grading (ACC to Schneiderman *et al.*)

Body Mass Index (kg/m2)	Number Of Patients			
	Normal	Grade 1	Grade 2	Grade 3
Underweight (<18.5)	1	-	-	-
Normal (18.5-23)	17 (3.4%)	40 (8%)	40 (8%)	60 (12%)
Overweight (23-27.5)	30 (6%)	37 (7.4%)	66 (13.2%)	140 (28%)
Obese (>27.5)	2 (0.4%)	40 (8%)	110 (22%)	321 (64.2%)

To our knowledge, this is the first cross-sectional study on the relation between BMI and Lumbar Intervertebral Disc Degeneration and LSS in an Indian Population. Our major finding is that higher BMI increases the risk of Lumbar Intervertebral Disc Degeneration (most commonly being L4-L5 level disc) and LSS. Since disc degeneration is related to low back pain and since being overweight or obese is also associated with low back pain, it is reasonable to assume that disc degeneration and low back pain may have increased body weight in common as a risk factor. However, to date, the relationship between BMI and disc degeneration has been a subject of controversy. In a study of 270 elderly Japanese subjects, Hangai *et al.* [17] showed that high BMI values were a risk factor for developing disc degeneration as evident on MRI. In their study of 129 Finnish men, Liuke *et al.* [26] showed that obesity was associated with the development of disc degeneration. Conversely, in the Rotterdam Study, which is a population-based study of Dutch subjects, a cross-sectional analysis of 2,819 individuals who underwent radiography did not show any association between elevated BMI and disc space narrowing [6]. In the Chingford Study, a prospective longitudinal assessment of 1,003 elderly women from the UK, a potential trend between elevated BMI values and the development of disc space narrowing was observed on plain radiographs; however, the association was not statistically significant [29]. In a cross-sectional analysis of 187 North American subjects from the Framingham Study who underwent computed tomography (CT) to assess spinal degenerative changes, Kalichman *et al.* [30] found a higher prevalence of facet joint osteoarthritis but not disc space narrowing in obese individuals. The analysis included 187 participants, of whom 13 had radiological findings of LSS. Another recent cross-sectional study of participants (n=938), in whom 78% were considered to have more than moderate radiographical central spinal stenosis, also displayed a positive association between radiological LSS and BMI [65]. More recently, based on their assessment of 44 pairs of male monozygotic twins in the Finnish Twin Cohort who had a difference in body weight of 8 kg, Videman *et al.* [28] concluded that being overweight or obese was not associated with disc degeneration, based on a quantitative signal variation on MRI. In fact, those authors concluded that greater body mass is "not harmful to the discs" and that it may "delay" disc degeneration. However, that study was limited by its small sample size and by the use of the criteria of an 8 kg difference in weight, which may have created subgroups that were not comparable. Our study, which included western Indian subjects ranging in age from 20 to 85 years, demonstrated a significant association between elevated BMI values and increased risk of LSS and the presence of disc degeneration at a greater number of lumbar levels, the global severity of disc degeneration, and end-stage disc degeneration with disc space narrowing on MRI.

End-stage disc degeneration with disc space narrowing is often coupled with degenerative changes throughout the vertebral motion segment (e.g., canal and neuro foraminal stenosis, ligamentous thickening) and altered lumbar kinematics that increase the risk of low back pain [6]. This severity of disc degeneration may help explain the increased prevalence of prolonged and chronic low back pain in overweight and obese individuals discussed in a recent systematic review by Shiri *et al.* [35]. Although previous studies did not show a significant association between elevated BMI and disc degeneration and lumbar canal stenosis, this is likely due to the lack of the use of advanced imaging (i.e., MRI) to assess the subtle disc changes that may otherwise be missed on plain radiographs or CT and the manner in which degenerative changes were assessed throughout the lumbar spine (i.e., assessing disc degeneration at each lumbar level or in specific regions). Therefore, it is very plausible that if the method of assessment of disc degeneration was consistent throughout studies, the effects of being overweight or obese on degenerative changes would be found to be more pronounced. Using such a large sample further facilitated controlling for various confounding variables associated with disc degeneration, providing the ability to arrive at robust conclusions in the assessment of the relationship of being overweight or obese to disc degeneration and its various dimensions. Being overweight and, in particular, obese are influential factors related to disc degeneration, but the exact mechanism of this association remains unknown. For numerous years, it has been postulated that being overweight or obese contributes to the compressive loading of the disc, leading to disc degeneration. However, recent studies have shown a linear association between hand osteoarthritis and atherosclerosis in elderly women [59]. Such findings are independent of increased loading effects that could be caused by body weight and perhaps may contradict the notion that being overweight or obese may contribute to arthritic changes due to altered biomechanics (e.g., of the hip or knee). Some authors contend that body weight may be a surrogate for physical loading and that musculoskeletal structures adapt to such forces [60, 61]. However, in our study, we showed that being underweight was associated with a significantly lower likelihood of having disc degeneration and

lumbar canal stenosis. These findings suggest that physical loading on the disc in the form of elevated BMI may biomechanically affect the disc or that it may be synonymous with an alternative mechanism that is not yet well understood. Nonetheless, our study has further broadened the understanding of factors related to lumbar disc degeneration and lumbar canal stenosis in adults. Although our study provides further insight into disc degeneration, there are certain potential limitations to consider. First, this study was conducted in a western Indian population, and our findings may not be generalizable to other populations. Moreover, focusing on western Indian subjects minimizes genetic variation that may confound the assessment of disc degeneration, which can occur in mixed population studies. Alternatively, variations exist between populations that may affect the impact that being overweight or obese has on disc degeneration; thus, factors related to disc degeneration may be population or ethnicity dependent and should be investigated further. A second limitation of this study was the cross-sectional design, which presents a challenge in assessing the causal pathway leading to disc degeneration. The third limitation was that although BMI is an established measure of overweight and obesity in both the clinic and research, the measure has admittedly a major limitation in its inability to differentiate adipose tissue from lean mass. Therefore, we are hesitant to conclude that being overweight or obese leads to the development of disc degeneration without more prospective analyses. However, due to the nature of the degenerative process, it would not seem plausible that being overweight or obese is entirely a result of disc degeneration. Future clinical studies assessing risk factors for disc degeneration should be cognizant of elevated BMI values, in particular, BMI values that indicate being overweight or obese and their effects on disease. In addition, future research efforts should be directed toward gaining a deeper understanding of the mechanisms that may contribute to disc degeneration in overweight and obese individuals, with the goal of developing preventative therapeutic interventions.

Conclusion

This study, one of the largest population-based cross-sectional study conducted to assess the phenotype of disc degeneration on MRI, is to our knowledge, the first to show that BMI values are significantly higher in individuals with disc degeneration (most commonly at L4-L5 disc level) than in individuals with non-degenerated discs. Elevated BMI values, in particular, BMI values indicating being overweight or obese, were associated with a greater extent and increased global severity of disc degeneration and Lumbar Spinal Stenosis. Furthermore, since this study was population-based, it is of tremendous public health importance. Since there is abundant evidence in the literature demonstrating the strong association of disc degeneration on MRI with low back pain [2, 4, 5, 7-13], and since the present study clearly illustrates that being overweight or obese is a strong determinant related to disc degeneration of the lumbar spine and Lumbar Spinal Stenosis, the public should be well informed that weight control is as important for preventing low back pain as it is for other conditions, such as heart disease and diabetes. Prevention and treatment of being overweight or obese must be a public health priority. If successful, such outcomes may lead to the prevention or minimization of the extent and severity of disc degeneration, which in turn may also decrease the risk of developing low back pain and the subsequent need for medical or surgical management, resulting in a more productive and healthier society. Whether weight loss reduces symptoms and progression of disc degeneration and LSS remains to be established.

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