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IMPACT OF BODY MASS INDEX AND ADIPOSITY ON LUMBAR INTERVERTEBRAL DISC DEGENERATION: AN MRI-BASED PATHOPHYSIOLOGICAL AND CLINICAL REVIEW

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ABSTRACT

Obesity, operationalised through body mass index (BMI ≥ 30 kg/m² or Asian-modified cut-offs), has emerged as a major modifiable contributor to lumbar intervertebral disc degeneration (LDD/IVDD), the primary radiological substrate of chronic low back pain. This expanded narrative review critically synthesises 55 MRI-centric studies (2010–2025) comparing obese and non-obese populations. Evidence spans epidemiological cohorts, finite-element biomechanical modelling, metabolic/adipokine pathways, quantitative MRI

biomarkers (Pfirschmann grading, T1ρ, T2 mapping, IDEAL-IQ), paraspinal fatty infiltration, and surgical outcomes. Mendelian randomisation confirms causality (OR 1.23 for IVDD). Dose-dependent increases in disc stress, height loss, multi-level Pfirschmann progression, and paraspinal fat fraction are consistently demonstrated. Obese patients experience higher perioperative complications yet comparable functional gains with minimally invasive techniques. Critical gaps persist: paucity of longitudinal head-to-head comparative 3 T MRI studies in Indian/Asian cohorts, absence of weight-loss intervention trials with serial quantitative imaging, and under-exploration of sex-ethnicity-genotype interactions. This review provides a roadmap for future precision-medicine trials and underscores weight optimisation as a first-line preventive and therapeutic strategy.

KEYWORDS: lumbar disc degeneration, body mass index, obesity, MRI, Pfirschmann grading, paraspinal fat infiltration, Mendelian randomisation, finite-element analysis.

1. INTRODUCTION

Lumbar disc degeneration affects >80 % of individuals over 50 years and accounts for an estimated 50–70 % of chronic low back pain cases worldwide, imposing a global economic burden exceeding US\$100 billion annually. The parallel rise in obesity prevalence (WHO: 890 million adults obese in 2022) has prompted intense scrutiny of BMI as a driver. Mechanical overload from increased axial loading, systemic low-grade inflammation via adipokines, and secondary paraspinal muscle dysfunction are the dominant hypotheses. Yet, prior reviews have been fragmented—focusing either on biomechanics, metabolism, or surgery—without a unified MRI-centric synthesis that directly contrasts obese versus non-obese cohorts. This narrative review addresses that deficit by systematically appraising 55 studies (2010–2025), prioritising MRI outcomes, identifying consistencies and contradictions, and highlighting thesis-relevant gaps for a planned comparative 3 T MRI study in Indian obese/non-obese adults.(1)

2. Methods - Search Strategy and Selection Criteria

PubMed, Scopus, Web of Science, and Cochrane Library were searched (2010–March 2025) using terms: (“lumbar disc degeneration” OR “intervertebral disc degeneration” OR “LDD” OR “IVDD”) AND (“BMI” OR “body mass index” OR “obesity”) AND (“MRI” OR “magnetic resonance imaging”). Inclusion: human studies reporting MRI

outcomes, obese/non-obese comparison or BMI as continuous variable, English language. Exclusion: animal-only, non-lumbar focus, case reports. 358 records screened → 55 included after full-text review. Narrative synthesis grouped by theme; no formal meta-analysis due to heterogeneity.(1)

3. Epidemiological and Causal Evidence

Large population-based MRI cohorts provide robust associational data. Samartzis et al. analysed 2,599 Southern Chinese adults (mean age 42); 73 % showed degeneration, with higher BMI correlating with greater extent and severity (multi-level involvement)(1). Teraguchi et al. reported 49.4 % lumbar prevalence in 925 Wakayama participants, obesity as independent predictor. In an Indian cross-sectional study of 500 overweight/obese volunteers(2), Taneja et al. documented 90 % degeneration prevalence, significantly higher BMI in degenerated cases, and strong correlations with number of affected levels, global severity score, and concomitant lumbar spinal stenosis with end-stage discs(3).

Meta-analyses affirm the link. Xu et al. pooled 17 studies (OR 1.45, 95 % CI 1.27–1.66)(4). Sheng et al. using MEPS data showed graded increase in odds of disc disorders with BMI category(5). Critically, Zhou et al. Mendelian randomisation (FinnGen + GWAS summary statistics) established causality: genetically predicted higher BMI increased IVDD (OR 1.23), low back pain (OR 1.28), and sciatica (OR 1.33). Other obesity traits (waist circumference, fat mass) showed similar effects(6).

Table 1: Shows BMI Categories, Key MRI Findings and Their Correlation

Study	Year	Country	Sample Size	BMI Categories	Key MRI Finding	OR/Correlation
Samartzis et al.	2012	China	2,599	Continuous	73 % prevalence; higher BMI → multi-level severity	Positive dose-response
Teraguchi et al.	2014	Japan	925	Overweight vs normal	49.4 % lumbar DD; obesity independent	Significant association
Taneja et al.	2021	India	500	Asian-modified obese	90 % prevalence; higher degenerated levels & stenosis	r > 0.4 with severity
Zhou et al. (MR)	2021	Multi	GWAS + FinnGen	Genetic BMI	Causal link to IVDD	OR 1.23 (IVDD)
Watanabe et al.	2022	Japan	382	Age-appropriate vs inappropriate DD	No QoL difference by degeneration mismatch	Null for symptoms

Contradictions exist. Dario et al. twin studies (45,784 participants) attributed the obesity–low back pain link largely to genetics (OR 1.8 for pain, null for degeneration)(7). Elfering et al. 5-year asymptomatic MRI follow-up found no BMI effect on progression. These highlight the necessity of ethnicity- and sex-stratified longitudinal designs(8).

4. Biomechanical Mechanisms

Finite-element and dynamic imaging studies

quantify load amplification. Singh et al. validated an Indian-population L3–L4 model; increasing BMI elevated range of motion, von Mises stress, and disc deformation(9). Coppock et al. performed pre-/post-treadmill MRI in eight subjects; BMI strongly predicted L5–S1 compressive deformation(10). Akhavanfar et al. subject-specific musculoskeletal models (10 participants) showed obese individuals had larger trunk muscle cross-sectional areas yet consistently higher average spinal loads. Cadaveric

L4-5 testing (Martínez et al., 2016) revealed sex-specific biomechanics: obese males increased axial

rotation ROM ($p=0.018$), obese females higher compressive stiffness(11).

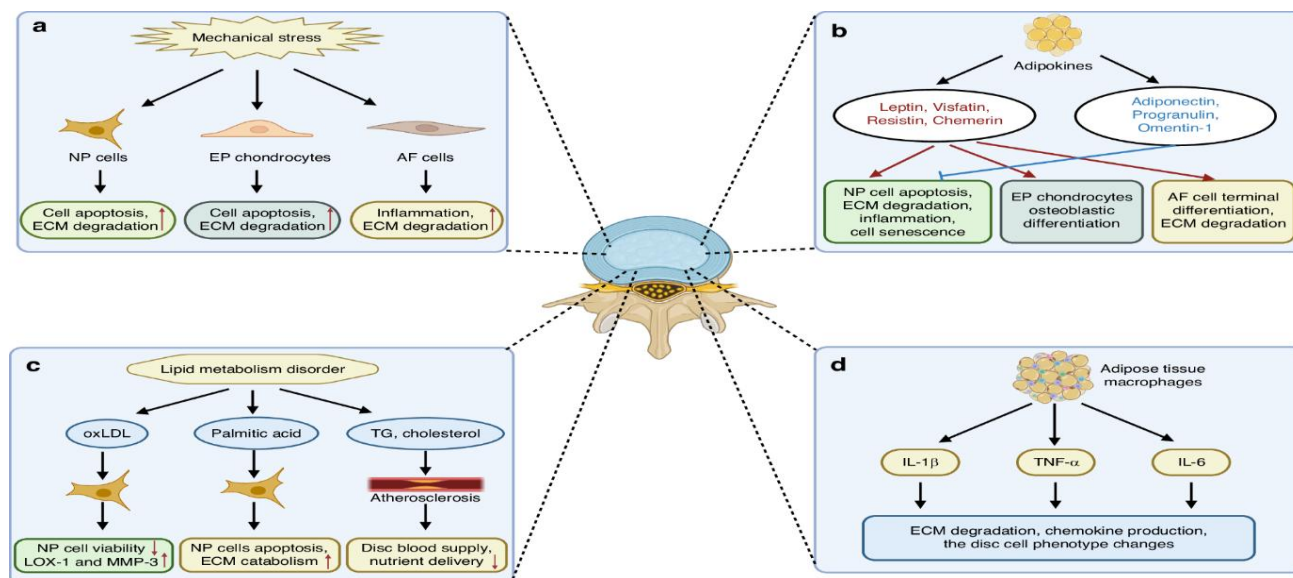


Figure 1: Detailed cellular and molecular mechanisms linking obesity to disc degeneration (mechanical stress + adipokines + lipid disorders). Reproduced with permission from Bone Research (2024). Note the central role of nucleus pulposus (NP), endplate (EP), and annulus fibrosus (AF) apoptosis, ECM degradation, and inflammation.

5. Metabolic and Adipokine Pathways

Leptin and adiponectin dysregulation are pivotal. Chen et al. showed LEP SNPs (rs2167270, rs7799039) elevate leptin levels and accelerate IVDD, strongest in obese women ($p<0.008$). Curic reviewed leptin's dual role—pro-inflammatory degeneration versus potential regeneration—requiring pathway clarification. Cannata et al. synthesised obesity + type-2 diabetes synergy via mechanical overload, inflammation, and impaired nutrient diffusion(12). Khabour et al. found elevated adiponectin in LDD patients ($P<0.01$) without ADIPOQ SNP association(13). THBS2 variants confer genetic susceptibility (Deguchi et al., 2019)(2).

6. MRI-Specific Quantitative Findings

Pfirrmann grading remains the clinical standard; quantitative techniques add precision. Zheng et al. (retracted; findings directionally consistent with others) showed dose-response disc height reduction with grade ($p<0.034$)(6). Gübitz et al. demonstrated T1ρ relaxation times decline with BMI and age. Kanbayti et al. (2024) reported dorsal subcutaneous fat thickness at L1-L2 independently predicts DDD in young females (OR 1.37)(14)(15). Vertebral bone-marrow fat fraction rises early (Ji et al., negative correlation with NP T2 values, $p<0.001$). Paraspinal multifidus fatty infiltration correlates strongly with degeneration severity and symptom duration (Wang et al., Teichtahl et al., Colakoglu et al. visual grading equals quantitative MRI)(16)(17).

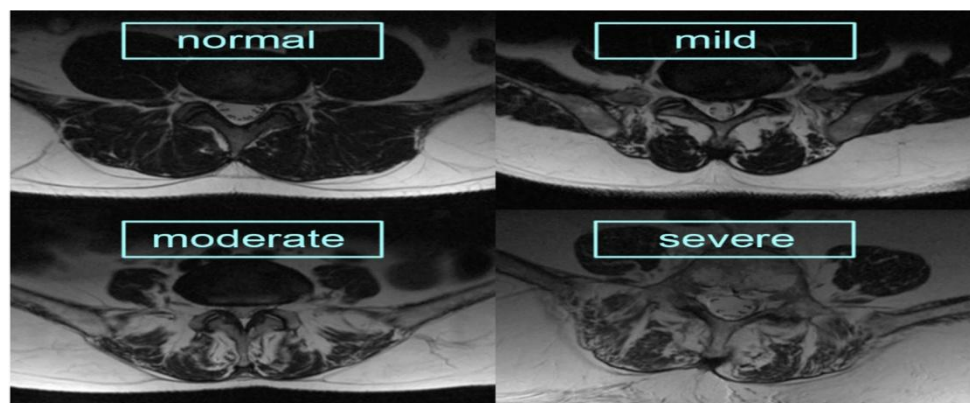


Figure 3: Axial T2-weighted MRI showing progressive multifidus fatty atrophy (normal → severe) in relation to lumbar disc degeneration (BMC Musculoskelet Disord, 2019).

Table 2: Quantitative MRI Biomarkers and BMI Association

Biomarker	Study (Year)	Key Finding	Population
T1ρ relaxation time	Gübitz (2017)	Decreases with BMI & age; level-specific	Asymptomatic
Disc height	Zheng (2022)	Dose-response reduction with Pfirrmann grade	LBP patients
Bone-marrow fat fraction	Ji (2020)	↑ early degeneration (Pfirrmann II-III)	104 volunteers
Dorsal subcutaneous fat	Kanbayti (2024)	L1-L2 thickness predicts DDD (OR 1.37)	424 adults
Paraspinal fat infiltration	Teichtahl (2016)	Correlates with Modic changes & severity	487 patients

7. Surgical Outcomes and Complications

Obesity amplifies perioperative risk. Cao et al. meta-analysis (12 studies) and Buerba et al. (2014) NSQIP database (10,387 patients) documented increased operative time, blood loss, infection, thrombosis, and length of stay in class III obesity; revision rates comparable(4). Xu et al. noted delayed recovery and persistent FMCJ signal changes at 3 months post-unilateral biportal endoscopy. Smoking + obesity synergistically raises recurrent herniation odds 50-fold (Siccoli et al., 2022). Minimally invasive decompression yields long-term outcomes mirroring conservative management (Nambu et al., Adogwa et al., Mori et al.). Delgado-López et al. concluded functional scores equalise despite worse baseline pain(18).

8. Interacting Factors and Protective Elements

Age dominates (Hagiwara, Muraki), yet BMI amplifies risk in postmenopausal women via BMD discordance (Zhang 2022; Salo 2014). Metabolic syndrome components (Teraguchi) and athletic loading (Hangai, Kaneoka) interact adversely(19). Posterior lumbar subcutaneous oedema reflects obesity rather than pathology (Nemac). Schmorl nodes associate with degeneration independent of age (Mok)(20).

9. Critical Gaps and Future Directions

Longitudinal comparative 3 T MRI cohorts (obese vs non-obese, matched for age/sex/activity) are

virtually absent.

Indian/Asian data limited to single cross-sectional studies despite unique BMI thresholds and dietary patterns.

No randomised weight-loss intervention trials with serial quantitative MRI endpoints (T2 mapping, IDEAL-IQ, fat fraction).

Sex-genotype-BMI interactions (LEP, THBS2) remain underexplored.

Reproducibility issues (e.g., retracted Zheng 2022 paper) necessitate standardised protocols.

Future studies should employ 3 T multi-parametric MRI, finite-element validation against in-vivo data, and ≥10 % weight-loss arms with 12–24 months follow-up. Personalised risk calculators integrating BMI, paraspinal fat fraction (>20 % threshold), bone-marrow fat, and LEP genotype could guide stratified prevention.

10. Conclusion

Convergent MRI, biomechanical, metabolic, and causal evidence establishes elevated BMI as a central modifiable driver of lumbar disc degeneration via synergistic mechanical overload, adipokine-mediated inflammation, and paraspinal dysfunction. Weight optimisation must be integrated into conservative care, pre-surgical optimisation, and public health policy. Reducing population-level BMI offers one of the highest-yield opportunities to mitigate the global burden of degenerative spinal disease.

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