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Correlation of Dorsal Subcutaneous Fat Thickness, Paraspinal Muscle Fat Infiltration, Modic Changes, and Demographic Factors with Spinal Canal Stenosis, with Lumbar Disc Degeneration as a Mediating Variable, Using Magnetic Resonance Imaging

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ABSTRACT

Background: Lumbar spinal stenosis is a common degenerative condition, but the joint relationships among paraspinal muscle fat infiltration, endplate changes, regional adiposity, disc degeneration, and MRI-defined canal narrowing are incompletely described. This study examined the level-specific associations of these MRI features with lumbar disc degeneration and MRI-defined central canal stenosis in a selected MRI-referral cohort, with disc degeneration examined as a hypothesized intermediate variable.

Methods: This retrospective cross-sectional study reviewed archived lumbar MRI examinations of 100 consecutive adults with degenerative findings at Royal Prima Hospital, Medan (acquired September–November 2025). At each level from L1-L2 to L5-S1, the dural sac cross-sectional area (graded 0–3), dorsal subcutaneous fat thickness, paraspinal muscle fat infiltration (Goutallier classification), Modic changes, and disc degeneration (modified Pfirrmann grading) were assessed. Level-specific Spearman correlation coefficients (ρ) with 95% confidence intervals were calculated, and the Benjamini–Hochberg false discovery rate (FDR) was applied across all 65 tests.

Results: Mean age was 48.87 ± 15.24 years, and 68% of participants were women. Age correlated with disc degeneration at all levels (ρ = 0.444–0.747), as did the Modic score (ρ = 0.275–0.541); multifidus fat infiltration correlated with disc degeneration from L1-L2 to L4-L5 (ρ = 0.280–0.632). Correlations with stenosis grade were weaker and level-specific: age at L3-L4 and L4-L5 (ρ = 0.361 and 0.405), Modic score from L2-L3 to L4-L5 (ρ = 0.254–0.490), and psoas and erector spinae fat infiltration at selected mid-lumbar levels (ρ = 0.256–0.392). Disc degeneration correlated with stenosis from L1-L2 to L4-L5 (ρ = 0.242–0.464) but not at L5-S1 (ρ = −0.035). Dorsal subcutaneous fat thickness was not significantly correlated with either outcome at any level.

Conclusion: In this MRI-referral cohort, age, Modic changes, and paraspinal muscle fat infiltration were associated with disc degeneration and, less consistently, with central canal stenosis. This pattern is consistent with, but does not demonstrate, an intermediate role of disc degeneration; longitudinal, multilevel studies with formal mediation analysis are needed.

Keywords: lumbar spinal stenosis; intervertebral disc degeneration; paraspinal muscles; Modic changes; magnetic resonance imaging

Introduction

Lumbar spinal stenosis is a common degenerative condition in older adults, and its burden is expected to rise as populations age (Hunter et al., 2025). Morphological (radiological) narrowing of the lumbar canal must be distinguished from the clinical syndrome, which requires compatible symptoms such as neurogenic claudication. In a systematic review, the pooled prevalence of clinically diagnosed lumbar spinal stenosis was 11% in the general population, 25% in primary care, and 29% in secondary care, whereas radiological stenosis was found in 11% of asymptomatic individuals (Jensen et al., 2020), indicating that imaging stenosis often occurs without symptoms. Lumbar spinal stenosis is one of the most common indications for spinal surgery in older adults in many health systems (Kwon et al., 2022; Ulrich et al., 2022), and it imposes substantial direct and indirect costs (Adogwa et al., 2012; Munakomi et al., 2023; Zileli

et al., 2020). Data from Indonesia remain limited. The processes that lead to canal narrowing are multifactorial and remain incompletely understood (Hennemann & de Abreu, 2021).

Degenerative stenosis results from interacting changes in the intervertebral disc, facet joints, ligamentum flavum, and spinal alignment, including degenerative spondylolisthesis (Gong et al., 2026; Iorio et al., 2016). Disc degeneration, characterized by dehydration of the nucleus pulposus and loss of disc height, alters load distribution and is one important contributor (Gradisnik et al., 2025; Scarcia et al., 2022); it acts together with ligamentum flavum hypertrophy, facet arthropathy, and osteophyte formation, which encroach on the central canal, lateral recesses, and neural foramina (Kushchayev et al., 2018; Kwon et al., 2022; A.-M. Wu et al., 2017). Magnetic resonance imaging (MRI) is the reference noninvasive modality for depicting these changes, although the precision of MRI measurements depends on acquisition parameters, measurement methods, and reader reliability (Liang et al., 2024; Scarcia et al., 2022).

Paraspinal muscle quality and regional adiposity have also been linked to spinal degeneration (Yang et al., 2022). Body mass index does not capture regional fat distribution, and MRI-based measures of subcutaneous fat and muscle fat infiltration have therefore been proposed (Özcan-Ekşi et al., 2021; Wang et al., 2022). Dorsal subcutaneous fat thickness, a simple linear MRI measure of regional subcutaneous adiposity, has shown inconsistent associations with canal stenosis (Atalay et al., 2022; Kushchayev et al., 2018). Paraspinal muscle fat infiltration, particularly of the multifidus and erector spinae, has been associated with low back pain, disability, and structural abnormalities (Chang et al., 2015; Teichtahl et al., 2015), and with adjacent segment disease after decompression (Ikeda et al., 2026; Teichtahl et al., 2015). The psoas, by contrast, is anatomically and functionally distinct from the posterior stabilizers, and its fat infiltration may reflect systemic sarcopenic or metabolic status rather than local posterior muscle function.

Vertebral endplate signal changes (Modic changes) are another component of the degenerative process. Modic type I changes are associated with bone marrow edema and inflammation, type II with fatty marrow replacement, and type III with sclerosis; these types represent distinct MRI phenotypes rather than successive severity grades (Liu et al., 2026; Zhu et al., 2025). Modic changes are more prevalent with advancing disc degeneration (Cao et al., 2025), and the two processes may develop in parallel or influence each other rather than follow a fixed sequence. Age and metabolic factors such as diabetes and dyslipidemia may influence all of these processes (Amroodi et al., 2025; R. Wu et al., 2025).

Previous studies have largely evaluated paraspinal muscle fat infiltration, dorsal subcutaneous fat thickness, and Modic changes separately (Jiang et al., 2024; Manju Bhashini Manoharan et al., 2026; Özcan-Ekşi et al., 2019; Teichtahl et al., 2015). Their joint, level-specific relationships with disc degeneration and MRI-defined canal stenosis in the same patients remain incompletely described, particularly in Indonesian patients.

Therefore, this study aimed to describe the level-specific associations of age, paraspinal muscle fat infiltration (multifidus, erector spinae, and psoas), Modic changes, and dorsal subcutaneous fat thickness with lumbar disc degeneration and with MRI-defined central canal stenosis from L1-L2 to L5-S1, and to explore whether the pattern of these associations is consistent with disc degeneration acting as an intermediate variable. We hypothesized that age, muscle fat infiltration, Modic changes, and dorsal subcutaneous fat thickness would be positively correlated with disc degeneration and stenosis severity, and that disc degeneration would be positively correlated with stenosis severity. Because the design is cross-sectional and correlational, these hypotheses concern associations rather than causal or predictive effects. No primary lumbar level was prespecified; all five levels were analyzed.

Methods

Study Design and Setting

This was a retrospective, observational, cross-sectional study of archived lumbar MRI examinations at Royal Prima Hospital, Medan. The MRI examinations were acquired during routine clinical care between September and November 2025, and the images and medical records were retrospectively reviewed and analyzed between May and August 2026. The study is reported in accordance with the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) statement (von Elm et al., 2007).

Participants and Sampling

The study population comprised adult patients (≥18 years) referred for lumbar MRI in routine clinical practice whose examinations showed degenerative findings. Eligible examinations acquired between September and November 2025 were identified consecutively from the PACS archive in chronological order (non-probability consecutive sampling). Inclusion required image quality sufficient to assess all study variables at all five levels. Patients were excluded if they had incomplete clinical or radiological data, inadequate image quality, or a history of congenital spinal abnormality, spinal infection, tumor, or lumbar trauma. Other conditions that may alter canal dimensions or muscle quality, such as previous lumbar surgery, spondylolisthesis, or scoliosis, were not specified as exclusion criteria and were not separately recorded. Because inclusion required degenerative findings, the sample is a selected MRI-referral cohort and does not represent patients with normal MRI or the general population; specific symptoms and clinical indications were not systematically recorded.

The minimum sample size was calculated as 62 subjects using the correlation formula (Sastroasmoro & Ismael, 2011), assuming a two-sided 5% significance level, 80% power, and a minimum correlation coefficient of 0.349, chosen as the smallest correlation of at least weak-to-moderate magnitude considered clinically meaningful. Allowing 10% for unusable data, a minimum of 69 subjects was required. A total of 100 consecutive examinations met all criteria and were included. With this sample, each individual correlation test had 80% power to detect $|\rho| \geq 0.28$ at $\alpha = 0.05$; the study was not powered for multiplicity-adjusted, multilevel, or mediation analyses.

Variables

The level-specific imaging exposures were paraspinal muscle fat infiltration (multifidus, erector spinae, and psoas), Modic changes, and dorsal subcutaneous fat thickness; age was a patient-level exposure. The outcome was the severity of MRI-defined central canal stenosis at each level, and disc degeneration was examined as a hypothesized intermediate variable. Sex was recorded for descriptive purposes only. The unit of analysis was the intervertebral level: every variable was assessed at each of the five levels (L1-L2 to L5-S1), and analyses were performed separately for each level. Age was calculated from the date of birth to the date of the MRI examination, in years. Central canal stenosis was assessed using the dural sac cross-sectional area (DSCSA) on axial T2-weighted images at each intervertebral disc level and graded as 0 = none (>100 mm^2), 1 = mild (75–100 mm^2), 2 = moderate (50–75 mm^2), or 3 = severe (<50 mm^2), based on the commonly used 100 mm^2 and 75 mm^2 thresholds for relative and absolute stenosis (Kuittinen et al., 2014), with an additional 50 mm^2 threshold to identify severe narrowing. This measure captures central canal narrowing only; lateral recess and foraminal stenosis were not assessed. DSCSA was categorized to provide clinically interpretable grades, and the continuous values are reported descriptively.

Dorsal subcutaneous fat thickness was measured on the midsagittal T2-weighted image (the slice showing the spinous processes and spinal canal in the midline) as the perpendicular distance from the skin surface to the thoracolumbar fascia at each disc level, in millimeters. It is a single linear proxy of regional subcutaneous adiposity and was not normalized for body size because body mass index was not available. Paraspinal muscle fat infiltration of the multifidus, erector

spinae, and psoas was graded on axial T2-weighted images at each disc level using the Goutallier classification (Goutallier et al., 1994), which was originally developed for rotator cuff muscles and has been widely applied to lumbar paraspinal muscles on MRI (Jiang et al., 2024): grade 0 = no fat, grade 1 = some fatty streaks, grade 2 = fat less than muscle, grade 3 = fat equal to muscle, and grade 4 = fat greater than muscle. Goutallier grading is a visual semiquantitative surrogate rather than a quantitative measurement of intramuscular fat fraction.
Modic changes were classified per disc level according to Modic et al. (1988): type I (hypointense on T1-weighted and hyperintense on T2-weighted images), type II (hyperintense on both), and type III (hypointense on both). The more extensive change of the two adjacent endplates was recorded, and mixed lesions were classified according to the predominant type. Disc degeneration was graded using the modified Pfirrmann system (Griffith et al., 2007), from grade 1 (homogeneous, bright disc with normal height) to grade 8 (homogeneous, dark disc with height reduced by >60% or collapsed), on an ordinal scale.

Data Collection

This study used secondary data, namely archived lumbar MRI examinations and medical records. All examinations were acquired with the hospital's routine clinical lumbar MRI protocol, including sagittal T1-weighted and T2-weighted and axial T2-weighted sequences, and were reviewed on the PACS workstation using its built-in measurement tools (electronic calipers for thickness and freehand region-of-interest tracing for DSCSA). Measurements and gradings were performed by the research team using the predefined criteria described above. Readers were not blinded to the other imaging variables or to patient age, and inter- and intrarater reliability were not formally assessed in this retrospective study; these issues are acknowledged as limitations.

Statistical Analysis

Data were checked for completeness, and records with errors or insufficient information were excluded. Sex was coded as 1 = male and 2 = female, and Modic type as 0 = none, 1 = type I, 2 = type II, and 3 = type III.
Continuous variables are presented as mean ± SD and categorical variables as frequencies and percentages; ordinal imaging grades (modified Pfirrmann, Goutallier, Modic score, and stenosis grade) are summarized as mean ± SD to allow comparison across levels. Because the imaging variables were ordinal or not normally distributed, associations were assessed using Spearman's rank correlation coefficient (ρ), separately at each intervertebral level (n = 100 per level). Age, a patient-level variable, was correlated with the level-specific measurements at each level. Modic type was entered as an ordinal score; because Modic types are distinct phenotypes rather than a validated severity continuum, correlations involving this score are interpreted as associations with the presence and type of endplate change. A 95% confidence interval (CI) was calculated for each coefficient using Fisher's z-transformation. Because 65 correlations were tested (Tables 3–5), the Benjamini–Hochberg false discovery rate (FDR) procedure was applied across all tests, and associations with $q < 0.05$ were considered robust (Benjamini & Hochberg, 1995). Correlation strength was described as weak (0.10–0.39), moderate (0.40–0.69), or strong (0.70–0.89) (Schober et al., 2018). Disc degeneration was examined as a hypothesized intermediate variable by assessing exposure–disc and disc–stenosis correlations. No multivariable, multilevel, or formal mediation analysis was performed; these patterns therefore describe associations and cannot establish mediation or causal ordering.

Ethical Considerations

The study protocol, including a waiver of informed consent for the use of anonymized retrospective imaging and medical-record data, was approved by the Health Research Ethics Committee, Faculty of Medicine, Dentistry, and Health Sciences, Universitas Prima Indonesia, and permission was obtained from Royal Prima Hospital, Medan. Data were coded, stored securely, and reported only in aggregate without individual identification.

Result

Demographic Characteristics of Study Subjects

This study included 100 patients, 68 women (68%) and 32 men (32%), with a mean age of 48.87 ± 15.24 years (Table 1). This relatively young mean age indicates that the cohort represents a broader population referred for lumbar MRI with degenerative findings rather than a classic older-adult population with symptomatic lumbar spinal stenosis.

Table 1. Demographic Characteristics of Study Subjects (n = 100)

Characteristic	n = 100
Age (Years), Mean ± SD	48.87 ± 15.24
Sex, n (%)	
Male	32 (32%)
Female	68 (68%)

Radiological Characteristics of Study Subjects

Table 2 presents the distribution of radiological characteristics at each intervertebral level from L1-L2 to L5-S1. Mean dorsal subcutaneous fat thickness increased from proximal to distal levels, from 16.73 ± 9.81 mm at L1-L2 to 25.99 ± 13.90 mm at L5-S1. The mean modified Pfirrmann grade was highest at L4-L5 (4.39 ± 1.65). The mean Modic score was highest at L5-S1 (0.60 ± 1.04); because Modic types are distinct phenotypes, this score summarizes the presence and type of endplate change rather than a severity scale.

Paraspinal muscle fat infiltration (Goutallier grade) differed among muscle groups. The multifidus and erector spinae had the highest mean grades at L5-S1 (2.34 ± 0.73 and 2.18 ± 0.66, respectively), whereas the psoas had the highest mean grade at L4-L5 (1.23 ± 0.66).

Table 2. Distribution of Radiological Characteristics (MRI) per Intervertebral Disc Level (n = 100)

MRI Parameter	L1-L2	L2-L3	L3-L4	L4-L5	L5-S1
Dorsal subcutaneous fat thickness (mm), mean ± SD	16.73 ± 9.81	15.57 ± 9.41	18.80 ± 10.72	24.05 ± 13.49	25.99 ± 13.90
Disc degeneration (mod. Pfirrmann 1-8), mean ± SD	3.59 ± 1.48	3.84 ± 1.61	3.97 ± 1.63	4.39 ± 1.65	4.21 ± 1.67
Modic score (0 = none; 1–3 = type I–III), mean ± SD	0.37 ± 0.87	0.40 ± 0.88	0.42 ± 0.85	0.48 ± 0.94	0.60 ± 1.04
Fat infiltration, Goutallier grade (0-4), mean ± SD					
- Multifidus muscle	1.86 ± 0.59	1.88 ± 0.59	2.08 ± 0.66	2.27 ± 0.76	2.34 ± 0.73
- Erector spinae muscle	1.78 ± [SD]	1.85 ± [SD]	1.89 ± [SD]	2.18 ± [SD]	2.18 ± 0.66
- Psoas muscle	0.98 ± 0.53	1.09 ± 0.59	1.19 ± 0.56	1.23 ± 0.66	0.93 ± 0.70
Dural sac cross-sectional area (mm^2), mean ± SD	183.13 ± 42.64	158.21 ± 46.96	138.05 ± 46.40	120.77 ± 53.70	114.99 ± 43.14
Stenosis grade based on DSCSA (0-3), mean ± SD	0.05 ± 0.26	0.15 ± 0.50	0.36 ± 0.75	0.69 ± 0.98	0.62 ± 0.80

Ordinal grades are summarized as mean ± SD to allow comparison across levels. Modic score: 0 = none, 1 = type I, 2 = type II, 3 = type III. Stenosis grade: 0 = DSCSA >100 mm^2, 1 = 75–100 mm^2, 2 = 50–75 mm^2, 3 = <50 mm^2. DSCSA, dural sac cross-sectional area.

Mean DSCSA decreased from L1-L2 (183.13 ± 42.64 mm^2) to L5-S1 (114.99 ± 43.14 mm^2), whereas the mean stenosis grade was highest at L4-L5 (0.69 ± 0.98), followed by L5-S1 (0.62 ± 0.80). This apparent discrepancy reflects the categorization of DSCSA: a lower mean area does

not necessarily produce more segments below the 75 mm^2 and 50 mm^2 thresholds, and the distribution of DSCSA was wider at L4-L5 (SD 53.70 mm^2) than at L5-S1 (SD 43.14 mm^2).

Correlation Analysis

Spearman correlation (ρ) was used to assess the associations at each lumbar level (n = 100 per level). Tables 3–5 report ρ, its 95% CI, and the p-value; associations that remained significant after FDR correction across all 65 tests are marked with †.

Table 3. Spearman Correlations of Age and MRI Features with Lumbar Disc Degeneration (n = 100 per level)

Independent Variable	Correlation Parameter	L1-L2	L2-L3	L3-L4	L4-L5	L5-S1
Age	Spearman ρ	0.658	0.746	0.747	0.705	0.444
	95% CI	0.52 to 0.77	0.63 to 0.83	0.63 to 0.83	0.57 to 0.80	0.26 to 0.60
	p-value	<0.001*†	<0.001*†	<0.001*†	<0.001*†	<0.001*†
Dorsal subcutaneous fat thickness (mm)	Spearman ρ	0.086	-0.005	-0.057	-0.076	-0.179
	95% CI	−0.11 to 0.28	−0.20 to 0.19	−0.25 to 0.14	−0.27 to 0.12	−0.36 to 0.02
	p-value	0.395	0.960	0.573	0.451	0.074
Modic changes	Spearman ρ	0.275	0.541	0.364	0.330	0.492
	95% CI	0.08 to 0.45	0.37 to 0.67	0.17 to 0.53	0.14 to 0.50	0.32 to 0.63
	p-value	0.006*†	<0.001*†	<0.001*†	0.001*†	<0.001*†
Multifidus fat infiltration	Spearman ρ	0.280	0.557	0.632	0.438	0.157
	95% CI	0.08 to 0.45	0.39 to 0.69	0.48 to 0.75	0.26 to 0.59	−0.04 to 0.34
	p-value	0.005*†	<0.001*†	<0.001*†	<0.001*†	0.118
Erector spinae fat infiltration	Spearman ρ	0.187	0.370	0.466	0.248	0.214
	95% CI	−0.01 to 0.37	0.18 to 0.53	0.29 to 0.61	0.05 to 0.43	0.02 to 0.40
	p-value	0.063	<0.001*†	<0.001*†	0.013*†	0.032*
Psoas fat infiltration	Spearman ρ	0.171	0.269	0.341	0.167	-0.023
	95% CI	−0.03 to 0.36	0.07 to 0.44	0.15 to 0.51	−0.03 to 0.35	−0.22 to 0.17
	p-value	0.089	0.007*†	0.001*†	0.098	0.821

*p < 0.05. †Remained significant after Benjamini–Hochberg false discovery rate correction across all 65 tests (q < 0.05). 95% CI calculated using Fisher's z-transformation.

Age was positively correlated with disc degeneration at all levels (all p < 0.001 and FDR-significant), with a moderate correlation at L1-L2 (ρ = 0.658), strong correlations from L2-L3 to L4-L5 (ρ = 0.705–0.747), and a moderate correlation at L5-S1 (ρ = 0.444). The Modic score was positively correlated with disc degeneration at all levels (ρ = 0.275–0.541). Multifidus fat infiltration correlated with disc degeneration from L1-L2 to L4-L5 (ρ = 0.280–0.632) but not at L5-S1. Erector spinae fat infiltration correlated with disc degeneration from L2-L3 to L4-L5 (ρ = 0.248–0.466); its correlation at L5-S1 (ρ = 0.214; p = 0.032) did not remain significant after FDR correction. Psoas fat infiltration correlated with disc degeneration only at L2-L3 and L3-L4 (ρ =

0.269 and 0.341). Dorsal subcutaneous fat thickness was not significantly correlated with disc degeneration at any level (ρ = −0.179 to 0.086), with 95% CIs (−0.36 to 0.28) that exclude moderate positive correlations.

Table 4. Spearman Correlations of Age and MRI Features with Severity of Central Canal Stenosis (n = 100 per level)

Independent Variable	Correlation Parameter	L1-L2	L2-L3	L3-L4	L4-L5	L5-S1
Age	Spearman ρ	0.104	0.179	0.361	0.405	0.064
	95% CI	−0.09 to 0.29	−0.02 to 0.36	0.17 to 0.53	0.22 to 0.56	−0.13 to 0.26
	p-value	0.305	0.075	<0.001*†	<0.001*†	0.525
Dorsal subcutaneous fat thickness (mm)	Spearman ρ	0.011	-0.073	0.061	-0.012	-0.006
	95% CI	−0.19 to 0.21	−0.27 to 0.13	−0.14 to 0.25	−0.21 to 0.18	−0.20 to 0.19
	p-value	0.911	0.468	0.547	0.908	0.956
Modic changes	Spearman ρ	-0.092	0.490	0.347	0.254	-0.081
	95% CI	−0.28 to 0.11	0.31 to 0.63	0.16 to 0.51	0.06 to 0.43	−0.27 to 0.12
	p-value	0.363	<0.001*†	<0.001*†	0.011*†	0.424
Multifidus fat infiltration	Spearman ρ	0.162	0.212	0.187	0.174	0.051
	95% CI	−0.04 to 0.35	0.01 to 0.39	−0.01 to 0.37	−0.02 to 0.36	−0.15 to 0.25
	p-value	0.108	0.034*	0.062	0.083	0.615
Erector spinae fat infiltration	Spearman ρ	0.105	0.112	0.219	0.256	0.073
	95% CI	−0.09 to 0.30	−0.09 to 0.30	0.02 to 0.40	0.06 to 0.43	−0.13 to 0.27
	p-value	0.299	0.267	0.028*	0.010*†	0.469
Psoas fat infiltration	Spearman ρ	0.009	0.337	0.392	0.123	-0.095
	95% CI	−0.19 to 0.21	0.15 to 0.50	0.20 to 0.55	−0.08 to 0.31	−0.29 to 0.10
	p-value	0.926	0.001*†	<0.001*†	0.223	0.348

*p < 0.05. †Remained significant after Benjamini–Hochberg false discovery rate correction across all 65 tests (q < 0.05). 95% CI calculated using Fisher's z-transformation.

Age was correlated with stenosis grade at L3-L4 (ρ = 0.361, weak) and L4-L5 (ρ = 0.405, moderate), both FDR-significant, but not at the other levels. The Modic score correlated with stenosis grade at L2-L3 (ρ = 0.490), L3-L4 (ρ = 0.347), and L4-L5 (ρ = 0.254), but not at L1-L2 or

L5-S1. Associations with paraspinal muscle fat infiltration were weak and level-specific: psoas at L2-L3 (ρ = 0.337) and L3-L4 (ρ = 0.392) and erector spinae at L4-L5 (ρ = 0.256) remained FDR-significant, whereas erector spinae at L3-L4 (ρ = 0.219; p = 0.028) and multifidus at L2-L3 (ρ = 0.212; p = 0.034) were nominally significant but did not survive FDR correction. Dorsal subcutaneous fat thickness was not correlated with stenosis grade at any level (ρ = −0.073 to 0.061). Differences in significance between levels were not formally tested and should not be interpreted as evidence of level-specific biological effects.

Table 5. Spearman Correlation of Lumbar Disc Degeneration (Hypothesized Intermediate Variable) with Severity of Central Canal Stenosis (n = 100 per level)

Intermediate Variable	Correlation Parameter	L1-L2	L2-L3	L3-L4	L4-L5	L5-S1
Disc degeneration (mod. Pfirrmann)	Spearman ρ	0.242	0.372	0.464	0.450	-0.035
	95% CI	0.04 to 0.42	0.18 to 0.53	0.28 to 0.61	0.27 to 0.60	−0.23 to 0.16
	p-value	0.015*†	<0.001*†	<0.001*†	<0.001*†	0.729

*p < 0.05. †Remained significant after Benjamini–Hochberg false discovery rate correction across all 65 tests (q < 0.05). 95% CI calculated using Fisher's z-transformation.

Disc degeneration, examined as a hypothesized intermediate variable, was positively correlated with stenosis grade from L1-L2 to L4-L5 (ρ = 0.242–0.464; all FDR-significant), with weak correlations at L1-L2 and L2-L3 and moderate correlations at L3-L4 and L4-L5, but not at L5-S1 (ρ = −0.035; p = 0.729). These are bivariate mediator–outcome correlations; no formal mediation (indirect-effect) analysis was performed.

Discussion

This study examined level-specific associations among age, paraspinal muscle fat infiltration, Modic changes, dorsal subcutaneous fat thickness, lumbar disc degeneration, and MRI-defined central canal stenosis, with disc degeneration examined as a hypothesized intermediate variable. Age, the Modic score, and fat infiltration of the multifidus and erector spinae were correlated with disc degeneration at most levels, whereas their correlations with stenosis grade were weaker and confined to selected mid-lumbar levels. Disc degeneration correlated with stenosis from L1-L2 to L4-L5 but not at L5-S1, and dorsal subcutaneous fat thickness was not significantly associated with either outcome at any level. Because only bivariate correlations were estimated, these findings describe a pattern of interrelated MRI features and cannot establish mediation or causal ordering.

The most robust findings were the correlations of age with disc degeneration at all five levels and the weak-to-moderate correlations of age with stenosis at L3-L4 and L4-L5, all of which remained significant after FDR correction. Ligamentum flavum hypertrophy and osteophyte formation are known to contribute to canal narrowing at these mobile segments (Kushchayev et al., 2018; Kwon et al., 2022; A.-M. Wu et al., 2017), but these structures were not measured here, so this explanation is external context rather than an observed pathway. Dorsal subcutaneous fat thickness increased from proximal to distal levels, which may simply reflect regional body contour, and it was not associated with disc degeneration or stenosis. This null result should not be generalized to adiposity as a biological construct: a single sagittal thickness is a noisy proxy that was not normalized for body size, body mass index was unavailable, and measurement error may have attenuated associations. Previous work has distinguished subcutaneous fat from intramuscular fat infiltration, which is associated with muscle atrophy and dysfunction (Noonan & Brown, 2021; Wesselink et al., 2023; Yu et al., 2026), but metabolic activity was not measured in this study.

Disc degeneration and Modic scores were highest at the lower lumbar levels, whereas mean DSCSA decreased distally. The Modic score correlated with stenosis grade at L2-L3, L3-L4, and L4-L5, but not at L1-L2 or L5-S1. Modic types represent distinct phenotypes, with type I associated with edema and inflammation, type II with fatty marrow replacement, and type III with sclerosis (Liu et al., 2026; Zhu et al., 2025), and inflammation was not measured directly; these correlations should therefore not be interpreted as evidence that endplate inflammation causes canal narrowing. Moreover, Modic changes and disc degeneration are closely interrelated manifestations of the disc–endplate complex (Rajasekaran et al., 2025), so Modic changes may be a consequence or co-manifestation of disc degeneration rather than an upstream exposure.

Fat infiltration of the multifidus and erector spinae correlated with disc degeneration at most levels, consistent with community-based (Teichtahl et al., 2015) and clinical (Shi et al., 2022) MRI studies. However, correlations with stenosis grade were weak and heterogeneous: psoas at L2-L3 and L3-L4 and erector spinae at L4-L5 remained significant after FDR correction, whereas multifidus at L2-L3 and erector spinae at L3-L4 did not. Muscle fat infiltration may reduce dynamic spinal stability (Faur et al., 2019), but no functional, strength, or biomechanical measure was obtained, so this mechanism is not directly supported by the present data. Reverse causation is plausible, because pain and reduced activity associated with degeneration may lead to disuse and secondary fatty infiltration. Psoas fat infiltration may also reflect systemic sarcopenia, physical activity, hip pathology, or age rather than local lumbar biomechanics. The absence of significant correlations at L5-S1 may reflect differences in central canal anatomy, the distribution of stenosis grades, or chance, and could not be explained by the present data.

The pattern of exposure–disc and disc–stenosis correlations is compatible with, but does not demonstrate, a pathway in which disc degeneration lies between muscle or endplate changes and canal narrowing. Several mechanisms have been proposed in the literature, including the release of proinflammatory cytokines (Chen et al., 2023), impaired nutrient diffusion through the endplate (Rajasekaran et al., 2025), and age-related cellular senescence of the nucleus pulposus (Bu et al., 2024), but none was measured here. Because the data are cross-sectional, disc degeneration may precede, follow, or co-evolve with paraspinal fatty infiltration and Modic changes (Jiang et al., 2024), and all may be parallel manifestations of aging. Formal testing would require multivariable, multilevel mediation models with temporally ordered data. Likewise, the absence of significant direct correlations at some levels does not establish the absence of a direct pathway, as measurement error, limited power, nonlinear effects, confounding, and segmental heterogeneity could obscure associations.

These findings are hypothesis-generating. They suggest that paraspinal muscle and endplate features co-occur with disc degeneration and, to a lesser extent, with central canal narrowing on routine lumbar MRI. Whether reporting these features adds diagnostic or prognostic value beyond standard stenosis assessment and age, and whether interventions targeting muscle quality or endplate changes alter disc degeneration or stenosis progression, remain to be established in longitudinal and interventional studies. Because this study used visual Goutallier grading, its findings do not validate quantitative fat-fraction sequences.

This study has several limitations. First, the cross-sectional design precludes causal inference and does not allow the temporal order of muscle, endplate, disc, and canal changes to be established; reverse causation is possible. Second, only bivariate correlations were estimated: no multivariable, multilevel, or formal mediation analysis was performed, the correlated structure of five levels within each patient was not modeled, and age, which was correlated with most variables, was not adjusted for, so confounding by age is likely. Third, 65 correlations were tested; although FDR correction was applied, isolated findings may be due to chance. Fourth, important structural determinants of stenosis (ligamentum flavum thickness, facet hypertrophy, disc bulging, osteophytes, spondylolisthesis, and alignment) and potential confounders (body mass index, diabetes, smoking, physical activity, occupation, and prior spinal surgery) were not measured. Fifth, imaging grades were assessed by readers who were not blinded, without a reliability assessment; Modic type was analyzed as an ordinal score although the types are

distinct phenotypes; and DSCSA captures central canal stenosis only. Sixth, the single-center MRI-referral cohort with degenerative findings was relatively young (mean age 48.9 years) and predominantly female and lacked clinical symptom data, which introduces referral and spectrum bias and limits generalizability to symptomatic lumbar spinal stenosis. Future studies should use prospective longitudinal designs, multilevel mediation models adjusted for age and body habitus, quantitative fat-fraction imaging, and reliability-tested measurements.

Conclusion

In this single-center MRI-referral cohort, age, Modic changes, and paraspinal muscle fat infiltration were associated with lumbar disc degeneration at most levels and with MRI-defined central canal stenosis mainly at mid-lumbar levels, and disc degeneration was associated with stenosis from L1-L2 to L4-L5. Dorsal subcutaneous fat thickness was not significantly associated with disc degeneration or stenosis in this sample. These findings describe a pattern of interrelated MRI features that is consistent with, but does not demonstrate, a role of disc degeneration as an intermediate factor. Longitudinal, multilevel studies with formal mediation analysis, adjustment for confounders, and reliability-tested measurements are needed before clinical or protocol recommendations can be made.

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DECLARATION OF INTEREST

There is no conflict of interest in this study.

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