

Incident or Worsening Spine Disease and Low Back Pain: A Systematic Review of Longitudinal Studies

Hanadi A. Albadi, Tatiana M. Martin, Lorena E. Juarez, Schuyler Hilton, Jennifer C. Westrick, John T. Martin
Rush University Medical Center, Chicago, IL; john_martin@rush.edu

Disclosures: Hanadi A. Albadi (N), Tatiana M. Martin (N), Lorena E. Juarez (N), Schuyler Hilton (N), Jennifer C. Westrick (N), John T. Martin (N)

INTRODUCTION: This study aimed to synthesize longitudinal evidence on the relationship between degenerative lumbar spine disease and incident low back pain (LBP) in adult general populations, evaluating whether incident or worsening degenerative changes are associated with LBP. Given the prevalence of spine disease in asymptomatic populations [1] and the prominent psychosocial risk factors for low back pain [2], we hypothesized that such changes have little or no association with LBP, addressing the need for clearer evidence to inform patient counseling and management.

METHODS: A systematic review was performed in accordance with PRISMA guidelines. PubMed/MEDLINE, Embase, Scopus, CINAHL, and Google Scholar were searched from inception to February 17, 2025. After removing 2,163 duplicates, 2,036 unique records were screened. Of 110 full-text articles assessed for eligibility, nine met all inclusion criteria. Eligible studies were prospective cohort designs that included baseline and follow-up imaging (MRI, CT, or X-ray) of the lumbar spine and tracked LBP longitudinally. Populations included adults aged 18 years and older with or without LBP at baseline, excluding studies from niche populations (athlete subgroups, etc.). Imaging features assessed included disc degeneration (DD), Modic changes (MC), Schmorl's nodes (SM), disc bulge, disc protrusion, disc extrusion, annular fissures (AF), disc height narrowing (DHN), osteophytes (OST), spondylolisthesis, facet joint osteoarthritis (FOA), central canal stenosis (CCS), disc signal loss (DSL), and high-intensity zone (HIZ). Outcomes were incident or worsening LBP in association with incident spine changes and baseline image findings as predictors of future LBP. Findings were synthesized descriptively due to heterogeneity in design and outcome definitions.

RESULTS: Nine studies (N ≈ 2,115; unique N ≈ 1,578, accounting for overlapping cohorts) [3-11] met inclusion criteria, with follow-up durations ranging from 1 to 16 years. Baseline populations ranged from asymptomatic groups, such as Veterans Affairs outpatients [3, 4] and hospital personnel [5], to mixed (with and without LBP) cohorts such as the TwinsUK registry [8, 9] and Johnston County Osteoarthritis cohort [10]. When baseline imaging findings were evaluated as predictors of future LBP, significant associations were reported for Pfirrmann summary scores (1/1), OST (1/1), and spine OA (1/1), while disc protrusion in one study [4] was protective (HR 0.5). In contrast, DSN (0/1), FOA (0/1), spondylolisthesis (0/1), DD (0/2), and MC (0/2) showed no associations. When new imaging findings during follow-up were analyzed as predictors of incident or worsening LBP, AF were significant in 1/1 study [3], whereas DD, bulge, HIZ, MC, and spondylolisthesis were consistently non-significant or inconclusive due to sparse data. Finally, when worsening of existing degenerative findings was examined, two studies [5, 6] found no significant associations for progression of DD, MC, bulge, or HIZ with incident LBP.

DISCUSSION: Due to the small number of studies, limited sample sizes, and substantial heterogeneity in populations, pain definitions, and statistical methods, no definitive conclusion can be drawn regarding the association between spinal changes and low back pain. Standardized definitions, harmonized imaging protocols, and large-scale longitudinal studies are needed to clarify the role of specific degenerative features in predicting LBP.

SIGNIFICANCE/CLINICAL RELEVANCE: These findings highlight the uncertainty surrounding the interpretation of degenerative spine changes on imaging. Clinicians should be cautious in attributing symptoms to such findings in isolation, and further evidence is required before imaging can be reliably used to guide prognostic decisions in LBP.

REFERENCES: [1] Brinjikji (2015) Am J Neuroradiol; [2] Huang (2023) JOR Spine; [3] Suri (2014) BMC Musculoskelet Disord; [4] Jarvik (2005) Spine; [5] Tonosu (2017) PLOS One; [6] Elfering (2002) Spine; [7] Aavikko (2024) Eur Spine J; [8] Munir (2018) Spine; [9] Määttä (2015) Spine; [10] Goode (2022) Arthritis Care Res (Hoboken); [11] Savage (1997) Eur Spine J.

Table 1 Overview of Included Studies (n = 9)

Author (Year) Country	Population	FU (y)	Image	N FU	N LBP BL	Mean age sex %	Pain Definition	Analyzed disc pathology	Result Baseline predictors of future LBP	Result Incident pathology and incident LBP	Result Worsening pathology and incident LBP
Suri (2014) US	VA, no severe LBP or any sciatica in last 4M	3	MRI	123	0	BL: 53 F: 13%	chronic LBP: ≥ moderate = ≥2 FU leg pain, any at ≥1 FU	AF, FOA, DHN, bulge, protrusion, extrusion, CCS, Desiccation, nerve contact, spondylolisthesis Endplate changes Same as Suri (2014)	NR	P < 0.05: AF* P > 0.05: all other pathologies	NR
Jarvik (2005) US	VA, no severe LBP or any sciatica in last 4M	3	MRI	131	0	BL: 54 F: 13%	PFI score >2 for LBP or >1 for leg pain at 21 FU	Same as Suri (2014)	P < 0.05: protrusion (HR: 0.5) P > 0.05: CCS, nerve root contact NR: other findings	Significance analysis not possible due to sparse new imaging findings	NR
Tonosu (2017) JP	Hospital personnel with previous but no current LBP	10	MRI	49	0	FU: 44 F: 51%	LBP/buttock = leg pain, in past month, = medical consult	DD, MC, bulge, HIZ, spondylolisthesis	P < 0.05: None P > 0.05: DD, DD, bulge, HIZ, spondylolisthesis NR: MC	P < 0.05: None P > 0.05: DD, DD, bulge, HIZ, NR: MC P < 0.05: None P > 0.05: DD, MC	P < 0.05: None P > 0.05: DD, NR: MC P < 0.05: None P > 0.05: DD, MC
Elfering (2002) CH	adults from trauma clinic for minor injuries with no prior consult for LBP	5	MRI	41	0	35.9 NR	self-reported occurrence, duration, frequency, and work/medical impact	DD, MC	NR	P < 0.05: None P > 0.05: DD, MC	P < 0.05: None P > 0.05: DD, MC
Aavikko (2024) FI	School-based cohort	16	MRI	41	19	BL: 18 FU: 34 F: 51%	Self-reported history of LBP over the past 16 years by age 34	Pfirrmann summary score (PSS)	P < 0.05: PSS	NR	NR
Munir (2018) UK	TwinsUK unselected for LBP	10	MRI	414	NR	BL: 53 F: 96%	Severe disabling LBP lasting >1M (lifetime history)	Endplate defect, MC, DD	NR	NR	NR
Määttä (2015) UK	TwinsUK unselected for LBP	10	MRI	429	NR	BL: 54 F: 95%	Severe disabling LBP lasting >1M (lifetime history)	DD, MC, SN	NR	NR	NR
Goode (2022) US	Johnston County Cohort	5.5	X-ray	798	278	NR F: 67%	"yes/no" to LBP on most days; if "yes": mild, moderate, or severe	Spine OA, OST, DSN, FOA, spondylolisthesis	Incident LBP: P < 0.05: OST, spine OA P > 0.05: DSN, FOA, Spondylolisthesis Worsening LBP: P < 0.05: OST P > 0.05: other 4 findings	NR	NR
Savage (1997) UK	Working men from 5 occupations	1	MRI	89	NR	NR (-36) M: 100%	Never LBP before 12M, within last 12M, monthly	DD, bulge, protrusion, facet hypertrophy, nerve root compression	NR	Significance analysis not possible due to sparse new imaging findings	NR

* only after additional adjustment for depression and arthritis; F: female; NR: not reported; VA: Veterans Affairs outpatients; BL: baseline; FU: follow-up; LBP: low back pain; PFI: Pain Frequency Index; MC: Modic changes; HIZ: high intensity zone; AF: annular fissures; DHN: disc height narrowing; DSL: disc signal loss; DD: disc degeneration; SN: Schmorl's nodes; CCS: central canal stenosis; FOA: facet joint osteoarthritis; OST: osteophytes; DSN: disc space narrowing; HR: hazard ratio

Figure 1 PRISMA flowchart

