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## Low Back Pain and Lumbar Spine Osteoarthritis: How Are They Related?

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### Abstract

Lumbar spine osteoarthritis (OA) is very common, with estimates of prevalence ranging from 40–85 %. The process of degeneration of the spine has commonly been classified as OA (disc space narrowing together with vertebral osteophyte formation); however, anatomically, the facet joint is the only synovial joint in the spine that has a similar pathological degenerative process to appendicular joints. Low back pain (LBP) is also a common condition, with nearly 80 % of Americans experiencing at least one episode of LBP in their lifetime. The complex relationship between spine radiographs and LBP has many clinical and research challenges. Specific conservative treatments for spine degeneration have not been established; there has, however, been recent interest in use of exercise therapy, because of some moderate benefits in treating chronic LBP. An understanding of the relationship between spine degeneration and LBP may be improved with further population-based research in the areas of genetics, biomarkers, and pain pathways.

### Keywords

Low back pain; Osteoarthritis; Spine osteoarthritis; Disc space narrowing; Intervertebral disc degeneration; Vertebral osteophytes; Facet joint osteoarthritis; Epidemiology; Treatment; Risk factors; Prevalence; Gender; Race

### Introduction

Osteoarthritis (OA) is the most common form of arthritis, affecting an estimated 27 million adults in the US [1]. The prevalence of OA has increased over the past two decades, and increases in life expectancy and obesity, both risk factors for OA, have led to concerns over

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the public health consequences of OA [1]. Arthritis in general is also a leading cause of disability and a significant cause of reduced quality of life [2, 3]. Commonly thought of as a disease of the peripheral joints (i.e., hips, knees, and hands), spine OA is often ignored in discussions of the prevalence and effect of OA on disability and function [4]. However, estimates of lumbar spine OA are high, ranging from 40–85 %, the large range being primarily because of study differences in definitions, distribution of age and other demographic factors, and recruitment of subjects [5].

Approximately 80 % of Americans experience at least one episode of LBP during their lifetime, making frequency of LBP second only to the common cold [6, 7]. Utilization of health care services resulting from LBP is high, and total social cost is estimated to be greater than 100 billion dollars per year in the United States [8]. Aside from prevalence and cost, the effect of LBP on the workplace is substantial. Approximately 149 million workdays per year are lost as a result of LBP [9], making it the most common reason for time off from work [10,11]. There is no doubt that LBP has a substantial effect on the US health care and workforce system, affecting more than 30 % of community-dwelling adults in a given year [12] and remaining one of the most common reasons for physician visits [13]. Because of its high prevalence and effect on health services, the spinal anatomical origins of LBP are of great interest to researchers and clinicians. To address prevention and treatment, understanding the etiology of LBP is important, because it may differ within the large majority of patients in whom we describe the etiology as mechanical or non-specific origin.

Although many imaging techniques can be used to diagnose and quantify OA, plain film radiographs are a commonly utilized technique because they are relatively inexpensive and easily administered [14]. Specific clinical reasons for use of plain film radiographs for individuals seeking care for LBP include addressing danger signals, for example malignancy, fracture, or infection, improving patient satisfaction, and improving understanding of the origin of LBP. In the absence of radicular symptoms, current guidelines suggest plain film radiography is a reasonable initial imaging option for LBP [15]. A steady rise in the use of plain film radiographs in spine care has also occurred [16]; this may be because of the increasing prevalence of LBP [17\*]. Use of plain films of the spine is common, irrespective of the stage or chronicity of LBP. Carey and colleagues [18], in a stratified probability sample of North Carolina residents, found that 45.8 % reported having spinal radiographs in the previous year, despite a mean duration of 9.8 years with chronic impairing LBP. The clinical decisions correlating radiographic findings and LBP are complex and poorly understood.

The association between radiographic spine degeneration and LBP has been debated since the 1970s [19]. Interest in spine OA and its effect on pain, function, and disability are increasing, and new research improving our understanding of spine OA's prevalence, risk factors, and associations with LBP has been conducted in recent years. The purpose of this review is to summarize this current evidence while describing the challenges of inconsistent spine OA definitions and to report recent conservative treatment approaches for LBP. This review concludes by highlighting recommendations for future spine OA and LBP research to address these two commonly related conditions with substantial public health importance.

## Pathophysiology and/or Anatomy

Individual radiographic features of the spine, commonly studied and referred to as the “three-joint complex”, are the structures of vertebral osteophytes (OST), facet joint OA (FOA), and disc space narrowing (DSN) from intervertebral disc degeneration [20]. All of these spinal structures have adequate nerve supply capable of generating LBP.

The vertebral facet joints (zygapophyseal joints) are synovial joints with the typical features of hyaline cartilage over subchondral bone, a synovial membrane, and a joint capsule [21]. Facet joint OA is a multifactorial process, and it has been thought that the presence of intervertebral disc degeneration leads to a greater load and motion at the facet joint, resulting in degenerative changes similar to those seen in other synovial joints [20]. However, the presence of facet joint OA has been found to be present even in the absence of intervertebral disc degeneration [22].

Situated between two vertebral bodies, the intervertebral disc is made up of two main regions: the soft inner nucleus pulposus and the firm outer collagenous annulus fibrosis [23]. The collagen content of the intervertebral disc consists of both type I and II collagen, with the nucleus containing only type II whereas the annulus contains both types I and II [24]. Changes to the disc collagen content can occur naturally with aging, a process commonly referred to as intervertebral disc degeneration. These aging-related changes include a decrease in aggrecan, water, and collagen content [25], resulting in DSN on plain film radiographs.

A vertebral osteophyte is a bony outgrowth that arises from the periosteum at the junction of bone and cartilage [26]. Osteophyte formation in the vertebral column has been shown to be a general indicator of age [27]. Osteophytes, however, may form without overt cartilage damage, implying they may form in an otherwise healthy joint [28]. However, the presence of disc space narrowing is highly associated with osteophyte formation [29].

The process of degeneration in the spine is thought to be initiated by disc degeneration; this disc degeneration is hypothesized to result in segmental instability that increases the load on the facet joints and leads to cartilage alterations [21]. This process has been clinically debated with little research support. Recently, Suri and colleagues [30\*] reported results from an ancillary project using 435 participants in the Framingham Heart Study. Using computed tomography (CT), they report that, for most individuals, there is ordered progression of spine degeneration beginning with the intervertebral disc. They also remark that increasing age, body mass index (BMI), and female sex may be related to isolated facet joint degeneration in some individuals [30].

## Spine Osteoarthritis or Degeneration?

Osteoarthritis is the clinical outcome of a disease process that results in structural and functional failure of synovial joints. This process has been characterized by damage to articular cartilage, subchondral bone alteration, a synovial inflammatory response, and an overgrowth of bone and cartilage [31]. In the spine, both the presence of intervertebral disc degeneration and osteophyte formation at the same vertebral level has been used to define lumbar spine OA, otherwise known as spondylosis [32]. Intervertebral disc degeneration and osteophyte formation may not share the same pathophysiological process of degeneration or have the anatomical synovial structures necessary to collectively meet the definition of OA. This has led to challenges and discussion of spine degeneration research because there is no consensus on whether this combination of features (DSN and OST) in the spine constitutes OA or a separate phenomenon [33]. As such, a clinically relevant definition that combines spine features to accurately represent spine OA is still unknown [34] and may not be necessary. The facet joint, however, is a synovial joint with a process of degeneration that may be associated with OA in appendicular joints [6]. Our recent work, using data from the Johnston County OA Project, found a statistically significant association between radiographic knee OA and hand OA and facet joint OA, but no significant association between DSN and hip, knee, or hand OA, or between vertebral OST and hip and hand OA

[35]. Findings such as these suggest the pathophysiological course of degeneration in the spine differs, depending on the radiographic feature, from that of hip, knee, and hand OA.

## Prevalence: Gender and Race Differences

The presence of these radiographic spine features is quite common, as evidenced by community-based prevalence of lumbar spine DSN between 50 and 64 % and vertebral OST prevalence as high as 75–94 % [34–37]. Our recent work is the only study to quantify the prevalence of radiographic FOA in a community-based population, revealing the prevalence affecting at least one lumbar level to be 57.9 % [35\*\*].

The prevalence of these radiographic features has been found to differ by gender and race. Most recent studies agree that OST is more prevalent [34, 35, 37] and severe [35, 37] in men whereas prevalence of DSN is greater in women [34, 35, 37]. Our recent work is the first to describe these features across a sample of African Americans (AA) and Caucasians [35]. We found the prevalence of all these radiographic features was significantly lower for AA than for Caucasians, most notably in the odds of FOA (OR = 0.45; 95 % CI 0.32, 0.62). The reason for this may be related to racial differences in occupational exposure, BMI, physical activity demands, or anatomical differences. As such, there is still much to be learned about the reasons for these race and gender differences and radiographic changes in the lumbar spine.

## Associations With Low Back Pain

By far, most epidemiological studies addressing the association between LBP and spine degeneration have been cross-sectional. Most of these cross-sectional studies have been practice-based, using select or small samples that have limited generalizability. Recently, a series of population-based cross-sectional studies have specifically set out to determine the association between lumbar spine radiographic features and LBP (Table 1). Cross-sectional designs with the outcome of LBP have been criticized for their lack of uniformity in LBP questions [38]. Despite the varying definitions of LBP, and different grading and coding schemes and settings across recent studies, a modest association between DSN and LBP has been consistently found. Conversely, the associations between OST and LBP have been weaker, and most are not significant. The association between radiographic FOA and LBP has only been reported in one study without a significant association with LBP, similar to a recent finding by Kalichman and colleagues [39] that indicates an insignificant association between FOA confirmed by CT scan and LBP.

An topic that is particularly understudied is the association between spine radiographic features and physical function. de Schepper et al. [34\*\*] found, in some instances, stronger associations between reduced physical function and disability and DSN (OR = 1.9; 95 % CI 1.4, 2.6) as measured with the Health Assessment Questionnaire. In this same cohort, Scheele et al. [40\*\*] recently found a strong association among those with both morning stiffness and LBP with DSN (OR = 2.5; 95 % CI 1.9, 3.4). Our findings with regard to physical function are similar to those of de Schepper et al., measured by use of a condition-specific questionnaire (Roland-Morris Back Pain and Disability Questionnaire). We have found, using data from the Johnston County OA Project ( $n = 1,633$ ), that after adjusting for age, BMI, and gender, mean levels of perceived disability because of LBP increase significantly ( $p < 0.05$ ) across severity of DSN. (Fig. 1; unpublished data).

## Risk Factors

The vague diagnostic classifications and multidimensional, recurrent nature of LBP create challenges for longitudinal designs aimed at determining the temporal relationship between

radiographic spine features and LBP. These challenges are compounded by the remaining questions related to the etiologic process of spine degeneration. Spine degeneration is a process that occurs slowly in some individuals and more rapidly in others [25]. The age at which the process of spine degeneration begins is largely unknown and may depend on the individual spine. Our recent cross-sectional findings indicate that 88 % of participants had at least mild OST with a mean age of 59.7 years, suggesting that this particular degenerative process may begin much earlier than that of DSN or FOA, for which the prevalence was 57.6 % and 57.9 %, respectively. These different factors make studying the temporal relationship between spine degeneration and LBP difficult.

Two previous longitudinal studies, consisting of women only, have found few predictive risk factors for either DSN or OST [33, 41]. Notable predictors of DSN progression from these studies have been the presence of baseline DSN [41] and the presence of baseline hip or knee OA [33]. The presence of baseline DSN was not found to be a risk factor for future back pain [42]. Recently, Muraki and colleagues [32\*\*], using data from the Research on Osteoarthritis/Osteoporosis Against Disability (ROAD), longitudinally determined the incidence of radiographic, Kellgren–Lawrence (KL) graded, lumbar spondylosis (presence of DSN and OST at the same vertebral level) and LBP in a Japanese community-based population. With a 3.3-year follow-up, the incidence of KL 2 grade lumbar spondylosis was high—50 % in men and 34.4 % in women. The presence of KL 3 grade spondylosis has been found to be a moderately significant risk factor for LBP (OR = 1.26; 95 % CI 1.03, 1.69). This risk, however, was stronger when three or more KL 3 levels were affected in both men (OR = 1.69; 95 % CI 1.03, 2.76) and women (OR = 1.77; 95 % CI 1.34, 2.34).

There is little consistency between studies of risk factors for future spine degeneration or LBP; some studies suggest baseline degeneration as a significant risk factor for future LBP whereas others do not. Ideally, such knowledge of risk factors could be used to develop primary prevention techniques for these outcomes; however, this topic is largely understudied, although we already have sufficient information to make clinical recommendations for the treatment of LBP.

## Current Conservative Treatment Options

Given the effect of LBP on the US healthcare system, workplace, and individual quality of life, there is great need to better understand predictors and etiologic factors of LBP. Most research has focused on treating symptoms and functional impairment of LBP rather than on understanding the mechanisms underlying the anatomic and functional changes we currently call spine degeneration and their relationship to symptoms and functional impairment. Currently, our understanding of the treatment for LBP is, stated simply, that some activity is better than no activity [43, 44]. Exercise therapy is an activity which has long been a treatment option for LBP, with Cochrane and other reviews indicating some effectiveness for treating chronic low back pain [45]. Specifically, treatment of LBP with yoga has attracted substantial interest in recent years [46–48]. A recent randomized clinical trial by Sherman and colleagues [47\*\*] found that outcomes for the yoga intervention group were superior to those for the self-care group, but no significant difference was found from the stretching group for chronic LBP. The functional outcomes found in the yoga and stretching group remained statistically superior to those for the self-care group after a follow-up of 26 weeks. These findings suggest it is the physical rather than psychological aspect of yoga that has moderate benefits for chronic LBP. This trial did not specifically target patients with intervertebral disc degeneration and excluded patients with severe intervertebral disc degeneration, because some patients with severe disc degeneration may not tolerate exercise therapy [48]. The use of conservative treatment to prevent spine degeneration or conservative treatment for spine degeneration as a primary technique for treating LBP has

not yet been reported in the literature. Reasons for this may be the small number of longitudinal studies, inconsistencies in predictors, and weak risk factors.

Furthermore, there are few secondary prevention intervention techniques specifically aimed at treating symptomatic spine degeneration. Injection therapy has been a popular treatment technique targeting spine features for LBP, and Karppinen et al. [48] have recently summarized evidence on this therapy. Facet joint injection therapy to treat LBP has increased dramatically in recent years. Among Medicare beneficiaries, intervention for facet joint pain has increased substantially with annual growth of 60 % from 1997 to 2006 [49]. However, there is little evidence to support the use of this therapy, with recent practice guidelines recommending against the use of facet joint steroid injections [50].

## Future Research Directions

### Biological Markers, Genetics and Pain Pathways

What future research areas are ahead for understanding of spine degeneration and LBP? The topic of spine degeneration seems to follow the research trends of OA in the knee and hip. A recent review of the literature indicates several serum and urine biomarkers may be useful in understanding the etiology of knee and hip OA [51]. Gruber and Hanley [52] indicated that biological markers for intervertebral disc degeneration that could be used to correlate patients' status regarding pain and symptoms would be especially meaningful. Minimum work has been performed on this topic, but a few small studies of select samples have found associations between DSN and C-terminal cross-linked telopeptide (CTX-II), a biomarker of type II collagen degradation [53, 54]. Our recent work led to an interesting result regarding DSN, cartilage oligomeric matrix protein (COMP), and LBP [55\*\*]. Among those with LBP ( $n = 265$ ), there was a strong positive association between COMP and DSN (OR = 1.82; 95 % CI 1.02, 3.27) that was not observed in those without LBP (OR = 0.65; 95 % CI 0.35, 1.20). Therefore, it is possible that elevated COMP levels reflect the ongoing process of degeneration in the intervertebral disc, as evidenced by DSN, and results in associated symptoms that have been reported for this degenerative process.

Another topic of increasing interest is understanding the possible involvement of genetics in spine degeneration and LBP. Disc degeneration may be partially explained by genetic factors [5] and several review articles have already summarized these genetic factors extensively [56–60]. A relationship between LBP and intervertebral disc degeneration has been established, and several studies have identified genetic risk factors for lumbar disc degeneration [60]. However, most of these studies are cross-sectional with small sample sizes, and longitudinal, population-based studies are needed to confirm these findings.

Understanding the involvement of pain pathways in OA is beginning to become clearer as studies are discovering more about central pain sensitization in knee OA [61, 62]. Our recent work measuring pressure–pain threshold tolerance among participants with knee and/or hip OA or knee and/or hip symptoms indicates that those with higher pain thresholds are significantly less likely to report knee or hip symptoms, and plain film radiographs were not significantly associated with pressure–pain threshold in either joint [63]. This is also true for other conditions with musculoskeletal components, for example fibromyalgia [64, 65]. Use of the pressure–pain threshold to improve our understanding of central and peripheral pain sensitization is not new to the study of LBP [66, 67]. However, its use with radiographic findings of the lumbar spine has not been studied at the population level and may improve our understanding of nervous system pathways in this complex relationship between spine degeneration and LBP.

## Conclusions

The radiographic features of spine degeneration are common and are associated with LBP and reduced physical function. Therefore, facet joint OA and degeneration of the spine resulting in DSN and OST formation should not be overlooked in the discussion of OA and population estimates of prevalence and disease burden. This is particularly true of the facet joint, which may follow an OA-related degenerative process similar to that of appendicular synovial joints. However, recent population-based studies have been unable to link FOA with LBP and the multidimensional nature of LBP may cloud these associations. In recent years population-based studies have consistently demonstrated that DSN is associated with LBP, and recent longitudinal evidence suggests it may be a risk factor for LBP. Current treatment has not focused on primary or secondary prevention of LBP resulting from intervertebral disc degeneration, but evidence suggests that exercise therapy in general, including stretching and yoga, are good treatments for LBP. The different associations of LBP, physical function, and radiographic features in the spine are indicative of the importance of individual assessment of radiographic features and measured outcomes in future studies. Given the multidimensional nature of LBP, determining the association between spine degeneration and both LBP and reduced physical function may prove more worthwhile and clinically applicable in understanding the burden of spine degeneration than self-reported LBP alone. Finally, the role of genetics, biological markers, and understanding pain pathways in spine degeneration and LBP may be crucial to understanding the complex relationship between these two conditions.

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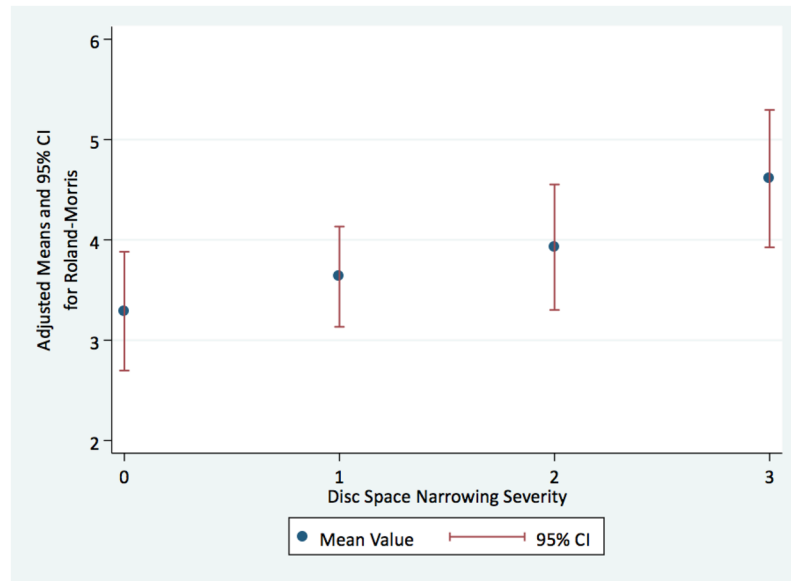


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**Fig. 1.** Adjusted mean values for the Roland–Morris Low Back Pain and Disability Questionnaire across severity of disc space narrowing. Findings from the Johnston County Osteoarthritis Project (unpublished)

**Table 1**

Associations between low back pain and spine radiographic features

Study (year) Ref.	Setting	Sample size (n)	DSN OR (95 % CI)	OST OR (95 % CI)	FOA OR (95 % CI)
Pye et al. (2004) [37]	Aberdeen, UK	585	1.7 (1.1, 2.4)	0.7 (0.4, 1.4)	N/A
Muraki et al. (2009) [36]	Japan	2,288	<sup>a</sup> Men = 1.44 (0.89, 2.38) Women = 1.80 (1.38, 2.37)	<sup>b</sup> Men = 1.15 (0.70, 1.92) Women = 0.99 (0.69, 1.42)	N/A
de Schepper et al. (2010) [34]	Rotterdam, Netherlands	2,809	1.4 (1.1, 1.7)	1.2 (1.0, 1.5)	N/A
Goode et al. (2012) [35]	North Carolina, US	840	1.37 (1.04, 1.80)	1.11 (0.84, 1.48)	0.84 (0.61, 1.15)

OR, odds ratio; CI, confidence interval; N/A, not applicable; DSN, disc space narrowing; OST, osteophytes; FOA, facet joint osteoarthritis

<sup>a,b</sup>Used Kellgren-Lawrence grading atlas:

<sup>a</sup><sub>K-L</sub> 2 and

<sup>b</sup><sub>KL</sub> 3