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Predictors of Lumbar Spine Degeneration and Low Back Pain in the Community: The Johnston County Osteoarthritis Project

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Abstract

Objective: To determine the incidence and worsening of lumbar spine structure and low back pain (LBP) and whether they are predicted by demographic characteristics or clinical characteristics or appendicular joint osteoarthritis (OA).

Methods: Paired baseline (2003-2004) and follow-up (2006-2010) lumbar spine radiographs from the Johnston County Osteoarthritis Project were graded for osteophytes (OST), disc space narrowing (DSN), spondylolisthesis, and presence of facet joint OA (FOA). Spine OA was defined as at least mild OST and mild DSN at the same level for any level of the lumbar spine. LBP, comorbidities, and back injury were self-reported. Weibull models were used to estimate hazard ratios and 95% confidence intervals (CI) of spine phenotypes accounting for potential predictors including demographics, clinical characteristics, comorbidities, obesity, and appendicular OA.

Results: Obesity was a consistent and strong predictor of incidence of DSN (HR=1.80, 95%CI 1.09–2.98), spine OA (HR=1.56, 95%CI 1.01–2.41), FOA (HR=4.99, 95%CI 1.46–17.10), spondylolisthesis (HR=1.87, 95%CI 1.02–3.43), and LBP (HR=1.75, 95%CI 1.19–2.56), and worsening of DSN (HR=1.51, 95%CI 1.09–2.09) and LBP (HR=1.51, 95%CI 1.12–2.06). Knee OA was a predictor of incident FOA (HR=4.18, 95%CI 1.44–12.2). Spine OA (HR=1.80, 95%CI 1.24–2.63) and OST (HR=1.85, 95%CI 1.02–3.36) were predictors of incidence of LBP. Hip OA (HR=1.39, 95%CI 1.04–1.85) and OST (HR=1.58, 95%CI 1.00–2.49) were predictors of LBP worsening.

Conclusion: Among the multiple predictors of spine phenotypes, obesity was a common predictor for both incidence and worsening of lumbar spine degeneration and LBP.

Chronic low back pain (LBP) impacts over 31 million Americans at any given moment[1] and has increased threefold in prevalence over a 10-year period.[2] The traditional “gateway” to interventions for LBP is diagnostic clinical imaging.[3] This is especially true within the primary care setting, where although plain film radiographs are not generally recommended by guidelines,[4] they are nonetheless frequently performed for examining whether lumbar spine structure is linked to LBP.[5,6] Improved understanding of the relationship between lumbar spine imaging and LBP is critically important,[7-13] as discordance between spine degeneration and LBP may lead to additional tests and referrals, some of which may have questionable benefit.[14]

Disc space narrowing (DSN) from degeneration of the intervertebral disc, vertebral osteophytes (OST) formation, facet joint osteoarthritis (FOA), and spondylolisthesis are potential sources of nociceptive pain in the lower back. Cross-sectional studies have linked lumbar spine degeneration with demographic and clinical characteristics.[7-9,15,16] However, longitudinal community-based studies are sparse, with most including only women, a considerable length of follow-up (approximately 9 years), and limited LBP examination.[10,12,13] Muraki et al.,[17] using data from a Japanese cohort, identified that sex was a significant predictor of incidence of lumbar spine degeneration; more severe

spine degeneration was also a significant predictor of LBP. However, differences between Japanese and US lifestyles may result in different predictors of incidence and worsening of lumbar spine degeneration and LBP.

To our knowledge, no other community-based study within the US has examined the incidence, worsening, and the longitudinal relationship between demographic and clinical characteristics and appendicular joint OA as predictors of radiographic lumbar spine degeneration and LBP within the same cohort. Therefore, our objective was to: 1) describe the incidence and worsening of lumbar spine vertebral OST, DSN, FOA, spondylolisthesis, and LBP, and 2) determine demographic or clinical characteristics and appendicular joint OA predictors of incidence and worsening of lumbar spine degeneration and LBP. We hypothesized that there would be 1) multiple factors predictive of degeneration, 2) multiple factors predictive of LBP, and 3) few (if any) factors predictive of both degeneration and LBP. These hypotheses are driven by previous research which suggests that factors predictive of structural changes are not the same factors as those predictive of LBP.[17-19] However, we intentionally posit a general hypothesis as we believe several factors are likely to be predictive of these outcomes.

Patients and Methods

Participants

Details of the sampling strategy and recruitment methods used for the Johnston County Osteoarthritis Project (JoCo OA) are described elsewhere.[7,20] This ongoing longitudinal study of OA includes African American (nearly one-third of the cohort) and white participants living in a largely rural county in North Carolina. Civilian, noninstitutionalized residents aged ≥45 years from six townships in Johnston County were enrolled between 1991 and 1998 (n=3187, Original Cohort), and additional residents were enrolled in 2003–2004 (n=1015, Enrichment Cohort). Since the Enrichment Cohort aimed to supplement the sample for African Americans and younger participants, participants enrolled during 2003–2004 tended to be younger (mean age 59.3 vs. 65.8 years) and more likely to be African American (40% vs. 28%) than the Original Cohort participants at first follow-up (1999–2003); the two groups did not differ according to sex.[21] Participants in JoCo OA completed follow-up clinical and interview data collection approximately every 5 years, with 1,695 participants seen during the 2006–2010 clinic visit (time point T2). All participants in JoCo OA have provided informed consent for participation, and JoCo OA has been continuously approved by the institutional review boards of the University of North Carolina and the Centers for Disease Control and Prevention in Atlanta, Georgia.

Radiographic Spine Structure

Lumbar spine radiographs were included in the JoCo OA study for participants at the T2 2006–2010 clinic visit (n=1685). There were 819 returning participants at the T3 time point (2013–2015) who completed lumbar spine radiographs. By protocol, women of reproductive age (<50 years) were excluded from having lumbar spine radiographs. Lumbar spine radiographs were performed with the participant lying on his/her left side, a common position for clinical radiographs, with the central beam centered at the lumbar

spine. The Burnett Atlas[22] was used to grade lumbar spine radiographic features of FOA, DSN, and OST. FOA was graded as absent or present at each lumbar level, while DSN and OST were graded in a semi-quantitative fashion (0=none, 1=mild, 2=moderate, and 3=severe). Spine OA was defined as the presence of at least mild OST and mild DSN at the same vertebral level,[23,24] which is similar to studies that define spine degeneration with the Kellgren-Lawrence (K-L) atlas.[13,17] Spondylolisthesis was graded based on the translation of the vertebrae relative to the diameter of the affected intervertebral disc space, with 0=no listhesis, 1=≤25%, 2=26%−50%, 3=51%−75%, 4=76%−100%, and 5=>100% translation. All lateral lumbar spine radiographs were graded at each lumbar level by an experienced single bone and joint radiologist (JBR) with an intra-rater reliability of kappa=0.73 for FOA, 0.89 for DSN, and 0.90 for OST.[25]

LBP

LBP was ascertained at the clinical interview by asking participants to answer “yes” or “no” to the question “On most days do you have pain, aching or stiffness in your lower back?” Those participants who reported “yes” were also asked to quantify the severity of their symptoms as “mild,” “moderate,” or “severe.”

Demographic and Clinical Characteristics

Demographic data were collected by clinical interview and examination, including age, sex, and race (African American/white). Clinical characteristics included self-reports of diabetes, high blood pressure, back injury, and history of cigarette smoking, as well as body mass index (BMI) at the time of interview (calculated from height measured without shoes and weight measured with a balance beam scale).

Appendicular Joint OA

The protocol for conducting appendicular joint OA radiographs has been described in detail elsewhere.[26,27] All knee, hip, and hand radiographs were read for K-L[28] score by the same radiologist (JBR). Inter-rater and intra-rater reliability have been reported previously with a kappa of 0.86 and 0.89, respectively, for both the hip and knee.[26] Hip and knee OA for these analyses was defined as a K-L score of 2-4 in at least one extremity. Hand OA was defined as K-L grade of 2-4 in at least one distal interphalangeal of one extremity with at least two other interphalangeal joints or carpometacarpal joints affected (K-L grade 2-4) across both hands.[27]

Statistical Analysis

Descriptive statistics were generated for the total sample. Incidence was defined as the absence of a specific radiographic feature at all levels of the lumbar spine at baseline and the presence of that feature at any level of the lumbar spine at follow-up. Worsening was defined as ≥1-unit increase in severity from baseline to follow-up for OST, DSN, and spondylolisthesis. Incidence was measured for all radiographic features, whereas worsening was not measured for FOA since this was measured on a dichotomous scale. LBP was considered incident if the participant reported “no” LBP at baseline but “yes” at follow-up. LBP was considered worsening if there was a ≥ 1-unit increase in severity from baseline

status to follow-up. Those with baseline severe LBP were excluded as they were unable to have a 1-unit increase in symptoms. Since the prevalence varied for each spine feature at baseline, the incidence and worsening sample sizes varied accordingly (Figure 1).

Our outcomes were interval-censored because the exact time of occurrence was not observed, but rather only whether the event occurred between time points. In addition, follow-up intervals were of varying length for JoCo OA participants (on average, 5.5 years). Due to these features of our data, we selected Weibull models to estimate hazard ratios accounting for potential predictors with corresponding 95% confidence intervals (CI). All models reported are multivariable and included demographic variables (age, race, and sex), clinical characteristics (diabetes, high blood pressure, smoking status, and BMI) and appendicular joint OA (knee, hip, and hand OA) predictors simultaneously. When FOA was the outcome, we also included spine OA; likewise, when spine OA, DSN, or OST was the outcome, we included FOA. We explored several pairwise interaction terms between each BMI interval and diabetes and demographic (sex, race, and age) and clinical characteristics (BMI, smoking, and diabetes), but we did not identify any significant interactions. Similar to other studies, we conducted post-hoc stratified analyses of the relationships between upper (L1-3) and lower (L4-5) lumbar levels for spine OA and FOA. Those results are provided in Supplemental Table 1. In addition, we compared differences in outcomes and predictors between participants in this study and those who were lost to follow-up (Supplemental Table 2). Finally, we conducted a simple sensitivity analysis to determine if our definition of spine OA would be influenced by the severity of OST and DSN. All analyses were conducted in SAS 9.4 (SAS Institute Inc., Cary, NC), and alpha was set at 0.05.

Results

Figure 1 illustrates the selection of participants in JoCo OA for both structure and symptoms for these analyses. For our structure outcomes, approximately 50% did not have lumbar spine radiographic data due to being lost to follow-up or failure to return for the clinic follow-up visit. A large proportion had baseline prevalent lumbar spine structural abnormality (54.9% with spine OA and 78.4% with FOA) or LBP (34.8%). Some were missing or refused lumbar spine radiographs at follow-up for lumbar spine structure (n=1 for FOA and n=1 for spine OA and n=6 spondylolisthesis), and some had missing covariate data for lumbar spine structure (n=28 for spine OA and spondylolisthesis, and n=17 for FOA). For our symptom outcome of LBP, approximately 50% had missing LBP data due to being lost to follow-up or failure to return for the clinic follow-up visit. After missing covariate data (n=21), 499 participants were eligible for incidence analysis and 638 were eligible for analysis of worsening.

Table 1 describes the baseline demographic and clinical characteristics as well as appendicular joint OA and lumbar spine degenerative factors. Just over two-thirds (67.9%) were women, and 31.8% were African American. OST and FOA were common at baseline: 84.3% and 78.4%, respectively; DSN and spondylolisthesis occurred less frequently: 26.1% and 17.8%, respectively. A majority (54.7%) of the participants were obese, a small percentage (1.6%) reported a history of back injury, and approximately 35% reported the

presence of LBP. The proportions of knee (39.6%), hip (35.5%), and hand OA (32.0%) were similar among participants.

The number of participants at risk for incident lumbar spine degeneration is described in Supplemental Table 3. A large proportion of participants developed OST at any level of the lumbar spine (59.0%). The incidences of DSN and spine OA were similar at 38.1% and 31.4%, respectively. The incidences of any FOA and spondylolisthesis were 10.2% and 9.1%, respectively. Approximately 34% of participants had a worsening of OST, 24% a worsening of DSN, and 8% a worsening of spondylolisthesis.

Table 2 describes the demographic predictors for incidence of lumbar spine degeneration in multivariable models including demographic, clinical characteristics, and OA variables as potential predictors simultaneously. Women were more likely to develop spondylolisthesis (HR=2.16, 95% CI 1.07–4.34). Obesity was a risk factor for incidence of DSN (HR=1.80, 95% CI 1.09–2.98), FOA (HR=4.99, 95% CI 1.46–17.10), spine OA (HR=1.56, 95% CI 1.01-2.41) and spondylolisthesis (HR=1.87, 95% CI 1.02–3.43). The presence of diabetes was a protective factor for the incidence of DSN (HR=0.54, 95% CI 0.30–0.97). Smokers were 39% less likely to develop DSN (HR=0.61, 95% CI 0.38–0.98) and 40% less likely to develop spine OA (HR=0.60, 95% CI 0.39–0.91). Knee OA was a risk factor for the incidence of FOA (HR=4.18, 95% CI 1.44–12.2). However, hip and hand OA were not risk factors for radiographic changes in the lumbar spine. In our sensitivity analysis of spine OA coding, we did not identify that the severity of OST or DSN changed the results.

Table 3 describes the predictors for worsening of lumbar spine degeneration in multivariable models including demographic, clinical characteristics, and OA variables simultaneously as potential predictors. Women had a 46% increased hazard of worsening of DSN (HR=1.46, 95% CI 1.02–2.08). Obesity was a risk factor for worsening of DSN (HR=1.51, 95% CI 1.09–2.09) but a protective factor for vertebral OST worsening (HR=0.75, 95% CI 0.57–0.99). The presence of diabetes was a risk factor for the worsening of vertebral OST (HR=1.38, 95% CI 1.00–1.92).

Table 4 describes the predictors for incidence and worsening of low back pain in multivariable models including demographic, clinical characteristics, and OA variables simultaneously as potential predictors. Obesity was a strong risk factor of both LBP incidence (HR=1.75, 95% CI 1.19–2.56) and worsening (HR=1.51, 95% CI 1.12–2.06). Having mild or moderate LBP at baseline was a predictor for LBP worsening. Participants with hip OA at baseline were 39% more likely to progress in LBP severity (HR=1.39, 95% CI 1.04–1.85). Baseline OST and spine OA were predictors of incident LBP (HR=1.85, 95% CI 1.02–3.36; HR=1.80, 95% CI 1.24–2.63, respectively). The presence of vertebral OST at baseline was also a predictor of worsening LBP (HR=1.58, 95% CI 1.00–2.49).

The results for the post-hoc stratified analysis of the incidence of upper and lower lumbar spine OA and FOA are described in Supplemental Table 1. Findings were similar to our primary analysis of the lumbar spine degeneration features. In addition, African-Americans were 59% less likely (HR=0.41, 95% CI 0.24–0.72) to have incident upper lumbar spine OA with symptoms and 53% less likely (HR=0.47, 95% CI 0.28–0.79) to have lower FOA.

Those with self-reported high blood pressure were 59% more likely (HR=1.59, 95% CI 1.01–2.51) to have incident upper spine OA.

Discussion

We determined the incidence and worsening of radiographic lumbar spine features commonly used in clinical practice to help better delineate relationship between spine structure and LBP. Obesity was a strong predictor for nearly all lumbar spine features and LBP. OST and spine OA had prognostic value as demonstrated by their prediction of LBP incidence and worsening. These findings may have important implications for clinical practice, especially where diagnostic imaging is being used for better understanding the relationship between lumbar spine degeneration and LBP.

We identified that obesity was a predictor of both the incidence and worsening of LBP. Muraki and colleagues[17] did not find obesity to be a significant predictor of incident LBP; however, their Japanese study population had a considerably lower average BMI compared to ours. Since our cohort had a large proportion of participants considered to be obese, these findings may not be generalizable to other community-based studies with a much lower proportion of obese participants. For radiographic features commonly examined during initial visits for LBP, we found that spine OA was a significant risk factor for incident LBP, similar to findings of others.[17] The baseline presence of at least mild OST was a significant predictor of LBP incidence and worsening. Although OST is common among the population, the presence of OST among those with LBP may be an indicator of continued or worse mechanical LBP. We also identified hip OA as a significant predictor of LBP worsening. Some have supported a “hip-spine syndrome” whereby influences from the presence of hip symptoms or OA have resulted in LBP.[29-34] To our knowledge, this is the first study to report a longitudinal relationship between baseline hip OA and LBP worsening.

Obesity was a strong and consistent predictor for the incidence of DSN, spine OA, FOA, and spondylolisthesis, and only for worsening of DSN. Muraki et al.[17] found that BMI was a significant risk factor for lumbar spine degeneration. However, our study also includes lumbar spine FOA and spondylolisthesis, which were not included in their study. Our findings support cross-sectional studies from both our group and others that indicate an association between obesity and FOA.[7,14,35] Due to the older age of our sample in JoCo OA, over 75% of participants already had FOA at baseline. As such, the small number of incident cases for FOA limits these findings. The finding that obesity was a protective factor for the worsening of OST is difficult to explain. One reason may be related to the large number of prevalent OST at baseline that limited the number at risk during follow-up. As such, this sample may be a more “resistant” group to OST development. Similar to our findings, a Framingham Study analysis[36] determined a higher proportion of women developed spondylolisthesis.

We identified some interesting relationships between diabetes and smoking and the incidence and worsening of DSN. Smoking has been associated with intervertebral disc disease in twin studies,[37] while other community-based studies[17-19] have found no significant association with spine degeneration. Our study, however, identified that

smokers were significantly less likely to have incident DSN. It has been suggested that inadequate statistical control of BMI may be one factor that leads to this inverse association; however, this was not the case in our study. The inverse association of smoking with DSN is concordant with the inverse association of smoking with knee OA found in many studies.[38,39] Self-reported presence of diabetes was identified as a significant risk factor for worsening of OST. This may be related to impaired glucose tolerance, which has been linked to ankylosing hyperostosis in the spine.[40] However, we also identified self-reported diabetes as inversely associated with incidence of DSN. We explored any potential differences between those participants self-reporting insulin use for diabetes treatment, based on the study by Shirinsky and Shirinsky[41] . In this prior study, they found that medication-treated diabetes was protective for knee OA progression. However, we did not identify any significant differences between these two groups. It is possible that other anti-diabetic medications—such as metformin, which has both antioxidant and anti-senolytic effects—might help explain the inverse association of diabetes and lumbar spine degeneration. The relationship between diabetes and DSN does appear to be focused more in the upper lumbar spine than the lower lumbar spine. A biologic rationale is not known for the BMI-independent inverse association of diabetes and smoking with DSN, but may be related to potential underlying nicotine effects or cellular mechanisms.[39] In addition, there may be other non-biological reasons that could explain these findings. We did not adjust our findings for multiple comparisons, and several of these findings are nearly statistically significant by our established threshold. In addition, while our models included several potential predictors simultaneously, we did not include all potential confounders that might influence our findings, and therefore we cannot rule out the potential for residual confounding.

Our study has several strengths but is not without limitations. Lateral lumbar spine radiographs could lead to non-differential misclassification of FOA status since lateral views may underestimate the occurrence of FOA. However, prevalence estimates of FOA based on lateral spine radiography[7] are similar to those previously reported based on computed tomography scans.[35] The JoCo OA protocol excluded women of childbearing age from having lumbar spine radiographs to prevent unnecessary radiation exposure; therefore, the results may not be generalizable to this subgroup. While the average length of follow-up of 5.5 years is ideal for examining degeneration, it limits the examination of incident and worsening of LBP since it may be quite variable over a long period. We measured the presence and severity of LBP but not how LBP interfered with daily activity. In addition, our question for LBP also includes pain, aching, and stiffness, which may overestimate the true incidence of LBP since stiffness may be present without pain. We did not include lower back-specific functional measures or account for previous LBP episodes, widespread pain, or psychosocial factors that are known to be associated with incident or progressive LBP.[42] Our study had over 40% loss to follow-up of cohort participants, which may limit generalizability since those lost to follow-up were more likely to be older and to have a BMI less than 30, self-reported diabetes, high blood pressure, and appendicular joint OA (knee, hip and hand OA). However, the primary reason for loss to follow-up was participant death. The loss to follow-up we experienced over this time frame may influence the direction and strength of our estimates relative to the true population values. This may be the case for

some estimates without clear statistical significance such as the relationship we identified between DSN and diabetes. In addition, there was a high prevalence of OST and FOA among participants based upon the average age of the cohort. As such, future studies should consider cohorts with younger participants to enhance generalizability.

In conclusion, our study is unique in that it provides estimates of lumbar spine incidence and worsening of radiographic features commonly used in clinical practice to examine spine health. The predictors identified in this study are commonly used in routine primary care for participants with LBP. The finding that obesity, existing lumbar spine degeneration, and appendicular hip OA are predictive of future LBP may assist in the ongoing efforts to decrease LBP in the community.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Significance and Innovations

- This longitudinal study of 819 participants over an average of 5.5 years found that obesity and appendicular joint osteoarthritis were significant predictors of the incidence and worsening of both lumbar spine degeneration and low back pain.
- Since obesity is a modifiable risk factor, efforts to decrease it could lessen the development and worsening of lumbar spine degeneration and low back pain.

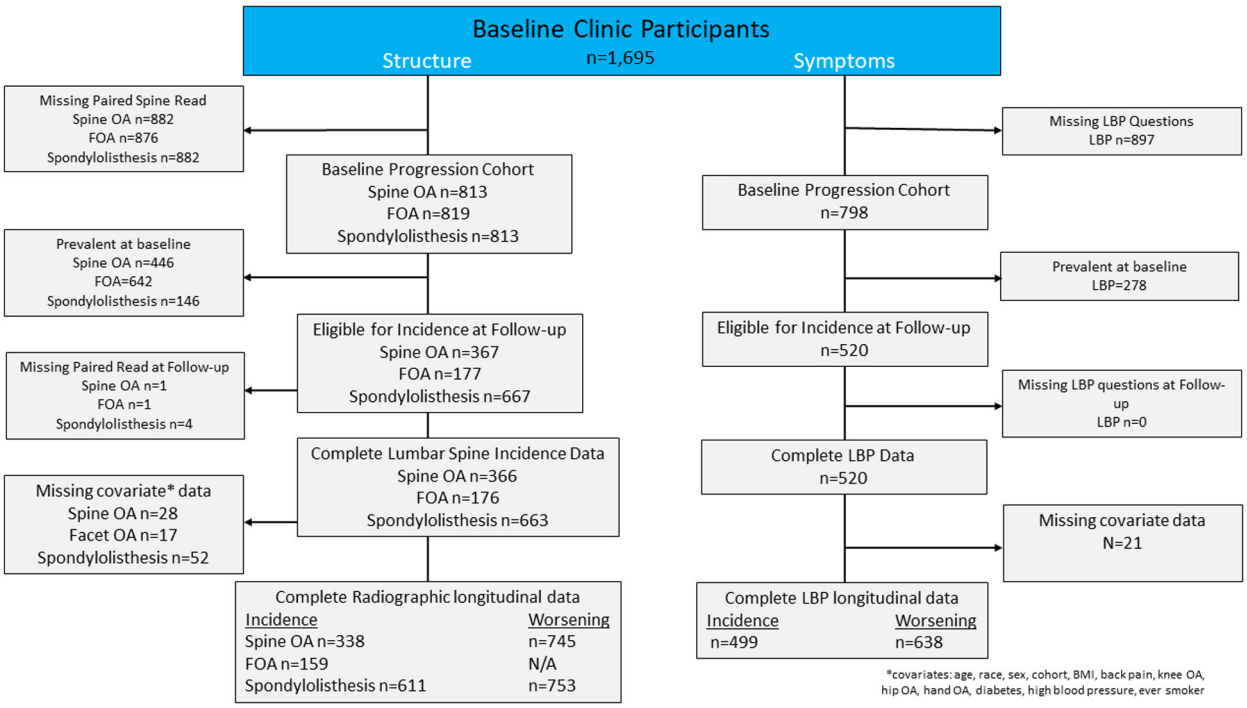

Figure 1.
Flow Diagram.

Table 1

Characteristics of participants at baseline with paired lumbar spine radiographs (N=819)

	n/N	% (95% CI)
Radiographic Spine Outcomes		
OST	691/819	84.3 (81.9-86.9)
Grade 0 vs 1-3		
Grade 0	128/819	15.6 (13.2-18.3)
Grade 1	511/819	62.4 (59.0-65.70
Grade 2	154/819	18.8 (16.2-21.7)
Grade 3	26/819	3.2 (2.1-4.6)
DSN	579/819	26.1 (23.0-29.3)
Grade 0 vs 1-3		
Grade 0	240/819	29.3 (26.2-32.6)
Grade 1	364/819	44.4 (41.0-47.9)
Grade 2	214/819	26.1 (23.2-9.3)
Grade 3	1/819	0.12 (0.0-0.7)
Spondylolisthesis	146/813	17.8 (15.3-20.6)
Grade 0	673/813	82.2 (79.4-84.7)
Grade 1	139/813	17.0 (14.5-19.7)
Grade 2	5/813	0.61 (0.2-1.4)
Grade 3	2/813	0.24 (0.03-0.9)
Grade 4	0/813	N/A
Grade 5	0/813	N/A
Spine OA present	446/813	54.9 (51.4-58.3)
FOA present	642/819	78.4 (75.6-81.2)
Demographic Predictors		
Age		
<55	34/819	4.2 (2.9-5.8)
55<65	371/819	45.3 (41.9-48.9)
65+	414/819	50.5 (47.1-54.0)
Sex - female	556/819	67.9 (65.0-71.0)
Race - African American	260/819	31.8 (28.6-35.1)
Clinical Characteristic Predictors		
Obesity (BMI≥30)	448/819	54.7 (51.3-8.1)
Diabetes	160/795	20.1 (17.3-23.0)
High blood pressure	499/796	62.7 (59.3-66.1)
Smoking	410/812	50.5 (47.0-54.0)
Back injury	13/811	1.6 (1.0-2.0)
Back pain	278/798	34.8 (31.5-38.1)
None	520/798	65.2 (61.0-69.1)
Mild	96/798	12.0 (9.5-15.1)
Moderate/severe	182/798	22.8 (19.5-26.6)

	n/N	% (95% CI)
Appendicular Joint OA Predictors		
Knee	318/803	39.6 (36.2-43.0)
Hip	287/809	35.5 (32.2-38.8)
Hand	260/813	32.0 (28.8-35.3)

BMI=body mass index; DSN=disc space narrowing; FOA=facet joint osteoarthritis; N/A=not applicable; OA=osteoarthritis; OST=osteophytes.

Table 2

Baseline demographics, clinical characteristics, and appendicular joint OA as predictors of incident lumbar spine degeneration in Weibull models

	OST	DSN	Spine OA	FOA	Spondylolisthesis
	N=114	N=220	N=338	N=159	N=611
Demographics					
Women vs. men	2.31 (0.88-6.04)	1.33 (0.79-2.23)	1.26 (0.78-2.04)	0.50 (0.14-1.75)	**2.16 (1.07-4.34)**
African American vs. white	1.19 (0.60-2.37)	0.69 (0.41-1.17)	0.81 (0.52-1.28)	0.96 (0.29-3.23)	1.20 (0.66-2.18)
Age 55-65 y vs. age <55 y	0.41 (0.11-1.58)	1.05 (0.34-3.23)	2.51 (0.75-8.42)	0.69 (0.03-14.9)	0.67 (0.19-2.41)
Age 65+ y vs. age <55 y	0.38 (0.09-1.55)	1.24 (0.37-4.10)	2.17 (0.62-7.58)	1.09 (0.05-26.1)	0.96 (0.26-3.59)
Clinical Characteristics					
Obesity	0.63 (0.31-1.27)	**1.80 (1.09-2.98)**	**1.56 (1.01-2.41)**	**4.99 (1.46-17.1)**	**1.87 (1.02-3.43)**
Diabetes	0.51 (0.17-1.57)	**0.54 (0.30-0.97)**	0.71 (0.42-1.18)	0.82 (0.20-3.35)	0.69 (0.34-1.39)
High blood pressure	0.80 (0.42-1.50)	0.76 (0.48-1.21)	0.81 (0.54-1.22)	0.51 (0.16-1.56)	1.26 (0.69-2.29)
Smoking	0.87 (0.49-1.56)	**0.61 (0.38-0.98)**	**0.60 (0.39-0.91)**	1.04 (0.35-3.11)	0.79 (0.46-1.38)
Back injury	0.57 (0.07-4.59)	0.56 (0.07-4.43)	0.72 (0.17-3.02)	NR	1.82 (0.24-14.0)
Appendicular Joint OA					
Knee OA	1.28 (0.62-2.65)	0.92 (0.54-1.57)	0.95 (0.62-1.47)	**4.18 (1.44-12.2)**	0.88 (0.50-1.53)
Hip OA	0.61 (0.34-1.10)	0.80 (0.48-1.34)	0.91 (0.59-1.38)	0.41 (0.13-1.27)	1.28 (0.74-2.22)
Hand OA	1.02 (0.56-1.84)	1.16 (0.68-1.97)	1.31 (0.84-2.02)	0.58 (0.16-2.09)	1.20 (0.66-2.19)

DSN=disc space narrowing; FOA=facet joint osteoarthritis; OA=osteoarthritis; OST=osteophytes; NR= not reported due to small sample size.

Data shown are HR (95% CI). Weibull models include sex, race, age, body mass index, diabetes, high blood pressure, smoking, and back injury, as well as knee, hip, and hand OA predictors simultaneously. Additionally, DSN and spine OA models included FOA presence, and FOA models included spine OA presence.

Table 3

Baseline demographics, clinical characteristics, and appendicular joint OA as predictors of lumbar spine degeneration worsening in Weibull models

	OST N=730	DSN N=754	Increased Levels of Spine OA N=745	Spondylolisthesis N=753
Demographics				
Women vs. men	0.85 (0.63-1.13)	**1.46 (1.02-2.08)**	1.22 (0.92-1.62)	1.93 (0.96-3.87)
African American vs. white	1.04 (0.77-1.40)	0.79 (0.55-1.13)	0.91 (0.69-1.21)	1.19 (0.66-2.13)
Age 55-65 y vs. age <55 y	0.84 (0.41-1.74)	1.40 (0.55-3.60)	**2.95 (1.07-8.15)**	0.67 (0.19-2.40)
Age 65+ y vs. age <55 y	0.89 (0.43-1.88)	1.29 (0.49-3.38)	**3.01 (1.08-8.42)**	0.90 (0.24-3.33)
Clinical Characteristics				
Obesity	**0.75 (0.57-0.99)**	**1.51 (1.09-2.09)**	1.24 (0.96-1.62)	1.77 (0.97-3.22)
Diabetes	**1.38 (1.00-1.92)**	0.85 (0.56-1.28)	0.77 (0.56-1.08)	0.81 (0.40-1.62)
High blood pressure	0.88 (0.67-1.16)	0.88 (0.64-1.20)	1.13 (0.87-1.47)	1.15 (0.64-2.04)
Smoking	1.00 (0.76-1.30)	1.08 (0.79-1.47)	0.95 (0.74-1.23)	0.77 (0.44-1.35)
Back injury	0.58 (0.18-1.83)	0.61 (0.15-2.49)	0.59 (0.19-1.85)	1.41 (0.19-10.6)
Appendicular Joint OA				
Knee OA	0.97 (0.74-1.28)	0.84 (0.61-1.16)	1.00 (0.78-1.30)	0.83 (0.48-1.45)
Hip OA	0.91 (0.69-1.19)	0.72 (0.52-1.00)	0.77 (0.60-1.01)	1.11 (0.64-1.91)
Hand OA	0.98 (0.73-1.32)	1.06 (0.75-1.48)	1.10 (0.84-1.43)	1.05 (0.58-1.90)

DSN=disc space narrowing; OA=osteoarthritis; OST=osteophytes.

Data shown are HR (95% CI). Weibull models include sex, race, age, body mass index, diabetes, high blood pressure, smoking, and back injury, as well as knee, hip, and hand OA predictors simultaneously.

Table 4

Baseline demographics, clinical characteristics, appendicular joint OA, and lumbar spine degeneration as predictors of incident and worsening low back pain in Weibull Models

	Low Back Pain Incidence (None to Mild or Greater) N=509	Low Back Pain Worsening (1-Unit Change From Baseline; Excludes Severe at Baseline) N=652
Demographics		
Women vs. men	1.19 (0.79-1.79)	1.35 (0.97-1.87)
African American vs. white	0.66 (0.43-1.01)	0.81 (0.58-1.12)
Age 55-65 y vs. age <55 y	0.70 (0.26-1.85)	0.68 (0.33-1.43)
Age 65+ y vs. age <55 y	0.55 (0.20-1.50)	0.66 (0.31-1.42)
Clinical Characteristics		
Obesity	**1.75 (1.19-2.56)**	**1.51 (1.12-2.06)**
Diabetes	0.68 (0.40-1.16)	0.92 (0.64-1.32)
High blood pressure	1.33 (0.91-1.95)	1.30 (0.96-1.76)
Smoking	0.69 (0.47-1.01)	0.83 (0.62-1.11)
Low back injury	1.85 (0.42-8.10)	2.39 (0.69-8.27)
Mild low back pain to moderate and severe	NR	**5.35 (3.62-7.92)**
Moderate low back pain to severe	NR	**2.26 (1.55-3.29)**
Appendicular Joint OA		
Knee	1.40 (0.97-2.02)	1.15 (0.86-1.53)
Hip	1.08 (0.75-1.57)	**1.39 (1.04-1.85)**
Hand	1.19 (0.79-1.77)	0.97 (0.71-1.33)
Spine Degeneration		
OST	**1.85 (1.02-3.36)**	**1.58 (1.00-2.49)**
DSN	1.52 (0.99-2.33)	1.11 (0.80-1.53)
Spine OA	**1.80 (1.24-2.63)**	1.27 (0.95-1.70)
FOA	1.06 (0.69-1.63)	1.05 (0.73-1.51)
Spondylolisthesis	1.14 (0.71-1.82)	1.12 (0.78-1.62)

BMI=body mass index; DSN=disc space narrowing; NR= not reported due to small sample size; OA=osteoarthritis; OST=osteophytes.

Data shown are HR (95% CI). Weibull models include sex, race, age, body mass index, diabetes, high blood pressure, smoking, and back injury, as well as knee, hip, and hand OA predictors simultaneously.