Published in final edited form as:

Comput Methods Biomech Biomed Engin. 2016 July ; 19(9): 1009–1018. doi:
10.1080/10255842.2015.1088524.

Comparative Role of Disc Degeneration and Ligament Failure on Functional Mechanics of the Lumbar Spine

Arin M. Ellingson, Ph.D.^{*}, Miranda N. Shaw, Ph.D., Hugo Giambini, Ph.D., and Kai-Nan An, Ph.D.

Biomechanics Laboratory, Division of Orthopedic Research, Mayo Clinic, Rochester, MN 55905, United States

Abstract

Understanding spinal kinematics is essential for distinguishing between pathological conditions of spine disorders, which ultimately lead to low back pain. It's of high importance to understand how changes in mechanical properties affect the response of the lumbar spine, specifically in an effort to differentiate those associated with disc degeneration from ligamentous changes, allowing for more precise treatment strategies. To do this the goals of this study were twofold: 1) develop and validate a finite element (FE) model of the lumbar spine and 2) systematically alter the properties of the intervertebral disc and ligaments to define respective roles in functional mechanics. A three-dimensional non-linear FE model of the lumbar spine (L3-Sacrum) was developed and validated for pure moment bending. Disc degeneration and sequential ligament failure was modeled. Intersegmental range of motion (ROM) and bending stiffness was measured. The prediction of the FE model to moment loading in all three planes of bending showed very good agreement, where global and intersegmental ROM and bending stiffness of the model fell within one standard deviation of the *in vitro* results. Degeneration decreased ROM for all directions. Stiffness increased for all directions except axial rotation, where it initially increased then decreased for moderate and severe degeneration, respectively. Incremental ligament failure produced increased ROM and decreased stiffness. This effect was much more pronounced for all directions except lateral bending, which is minimally impacted by ligaments. These results indicate that lateral bending may be more apt to detect the subtle changes associated with degeneration, without being masked by associated changes of surrounding stabilizing structures.

Keywords

finite element; lumbar spine; biomechanics; disc degeneration; ligament failure

Introduction

Low back pain is the most prevalent musculoskeletal impairment in the United States affecting 70–85% of the population annually¹. The causes of low back pain are often ambiguous and contributing tissues are difficult to discern. Intervertebral disc degeneration

^{*}Corresponding Author: Arin M. Ellingson, Ph.D., Mayo Clinic, 200 First Street SW, Rochester, MN 55905, Phone: 507-538-0215, Fax: 507-284-5075, ellingson.arin@mayo.edu; ellin224@umn.edu.

and ligament failure has been identified as contributors to low back pain^{2,3}. During clinical examinations, lumbar functional motion is evaluated to determine these mechanisms of pain. It's important to understand how changes in the surrounding soft tissues, such as intervertebral disc (IVD) and ligaments, affect motion characteristics to be able to isolate the specific structure instigating spinal instability^{4,5}.

Flexion/extension has the largest range of motion, and therefore is the most monitored from a clinical perspective⁴. Recent research eluded coronal plane motion may be more sensitive in detecting the subtle changes that occur in response to diminished disc health⁶⁻⁸. Particularly, it is important to understand the changes that are associated with disc degeneration from those that are ligamentous in nature as it will alter the course of treatment. Isolation of the specific underlying pathological tissue may allow for more precise treatment strategies, especially as novel biologics and other treatments are made available for disc and ligament repair.

A finite element (FE) approach allows for the alteration of various parameters to systematically understand the role of each structure and condition on the spine's biomechanical response. Disc degeneration has been the subject of many lumbar FE models⁹⁻¹¹, however very few studies have investigated the role of ligamentous pathology or failure^{12,13}. Moreover, these studies only included a single spinal unit, rather than a longer spinal segment. The addition of adjacent segments alters the boundary conditions of the system and may therefore have an impact on the findings. Also, to the author's knowledge, no studies have aimed to define the comparative role between disc and ligamentous health on the mechanical function. It is of high importance to understand how alterations in the mechanical properties of each structure affect the response of the lumbar spine.

The goals of this study were twofold: 1) to develop and validate a three-dimensional non-linear FE model of the lumbar (L3-Sacrum), and 2) to systematically alter the soft tissue (IVD and ligaments) properties to define each tissue's respective role in range of motion (ROM) and bending stiffness. We hypothesize disc degeneration will cause a decrease in ROM and an increase in stiffness, where progressive ligament failure will result in an increase in ROM and a decrease in stiffness. Furthermore, we hypothesized disc degeneration has a larger impact relative to ligament failure for flexion, extension, and axial rotation compared to lateral bending.

Methods

Development of Finite Element Model

A fresh, frozen human cadaveric spine (male, age 52 yrs.) was obtained from the Mayo Anatomical Bequest program. The specimen was visually and radiographically screened for significant deformity and other anatomic anomalies. The specimen was kept frozen at -20°C until the day prior to imaging. The specimen was thawed to room temperature and kept moist with toweling soaked with saline throughout preparation and scanning. A high-resolution computer tomography (CT) scan was obtained on a Siemens Helical CT Scanner

(Siemens Corp., Munich, Germany) and reconstructed with a pixel size of 0.293 mm and 0.4 mm slice thickness (Figure 1).

Vertebral bodies were segmented using an automated thresholding mask for bone using Mimics (v12.3 Materialise, Ann Arbor, MI). Manual segmentation was then performed on each image slice to create a solid mask of the vertebral and all bony features. A three-dimensional model was generated based on the masks and surfaces were smoothed for meshing. IVDs were generated using the wrap function in 3-matic (Materialise, Ann Arbor, MI).

The final 3-D geometry is shown in Figure 1. The Stereolithography (STL) surface model (L3-Sacrum) was exported and generation of the mesh was performed in Hypermesh (v10.0 Altair Engineering, Inc., Troy, MI, USA). With 71,477 elements, the following meshes were created (Table 1) and evaluated using ABAQUS (v6.13, Dassault Systèmes, Providence, RI):

Vertebrae—The vertebral body was defined as a cancellous bone core surrounded by a 0.5 mm thick cortical bone shell^{14–17}. Vertebral bodies and pedicles were constructed of 3-D first-order hexahedral elements. Posterior elements were constructed of 3-D second-order tetrahedral elements. The transition elements connecting the posterior tetrahedral elements to the hexahedral pedicle elements were 3-D second-order pyramid elements. Due to the complexity of the sacrum, the mesh was constructed of 3-D second-order tetrahedral elements. The transition elements between the S1 endplate and sacral tetrahedral elements consisted of 3-D second-order pyramid elements.

Vertebral Endplate—The vertebral endplates were defined as a 0.5 mm thick layer of elements on the superior and inferior surfaces of the vertebral bodies^{14,15,18}. The region connected to the nucleus pulposus (NP) was defined as the cartilaginous endplate, while the region over the annulus fibrosus (AF) was the bony endplate. The endplates were represented by 3-D first-order hexahedral elements.

Intervertebral Disc—Discs was modeled as a composite of a solid matrix with embedded fibers in concentric rings. The NP was surrounded by seven AF lamellae, containing two evenly spaced layers of fibers oriented at approximately $\pm 30^\circ$ to the horizontal plane^{4,19}. The fibers were defined using 3-D two-node truss elements. Collagenous fiber content varies from 23% in the outermost layer to 5% in the innermost layer^{20,21}. Thus, the cross-sectional area of the truss elements was determined based on the annular layer volume and the number of elements to satisfy the fiber content distribution. The fiber thickness and stiffness increased in the radial direction (Table 1). A “no compression” option was defined for the AF such that the elements resist tension only. The solid matrix of the AF was modeled as a neo-Hookean solid, while the NP was linearly elastic.

Facet Joint—The inferior and superior facets were lined with a thin layer of cartilage on the articular surface. This cartilaginous layer was simulated with the “softened contact” parameter in ABAQUS. This contact parameter adjusts force transfer between the nodes exponentially, depending on the initial gap size. An initial gap of 0.1 mm was assumed²². Upon closure of the gap, the stiffness of the joint assumed the same stiffness as the

surrounding bone²³. To simulate the frictionless surface of the cartilage, zero friction was applied during surface slippage.

Ligaments—All major ligaments were represented in the intact spine model (Figure 2): anterior longitudinal (ALL), posterior longitudinal (PLL), intertransverse (ITL), ligamentum flavum (LF), interspinous (ISS), supraspinous (SSL), and capsular (CL). The ligaments were modeled with 3-D two node truss elements and assigned nonlinear hypoelastic material properties, which allow axial stiffness to be a function of axial strain (Table 2). At initially low strains the ligaments exhibit low stiffness, but as the strains increase the ligament stiffness increases. The cross-sectional areas of each ligament were obtained from literature. All elements representing the ligaments were aligned in the anatomical fiber direction and unstressed at the unloaded state⁴.

Validation

Eighteen osteoligamentous cadaveric lumbar spines (L3-Sacrum) were acquired from the University of Minnesota Bequest Program (age: 53.2 ± 15.5 yr). Disc degeneration grade was assessed via the Pfirrmann scale from T2 weighted MRI²⁴. Specimens were embedded in polymethylmethacrylate (PMMA) and tested using a 6-axis Spine Kinetic Simulator (8821 Biopuls, Instron, Norwood, MA)²⁵. Pure moments of 7 Nm in flexion/extension, lateral bending, and axial rotation were applied with no axial preload. The inferior portion was unconstrained in the anterior-posterior and lateral directions to minimize shear forces. Input moments and forces were controlled by a 6 degree-of-freedom load cell (AMTI M4380, Watertown, MA). Three cycles of bending were performed sinusoidally (0.015 Hz), and the final cycle was used for analysis. Load and moment data were collected on two six-axis load cells at 100 Hz. Intersegmental angular displacements were recorded at 100 Hz using a motion analysis 5-camera system (Vicon MX-F40NIR, Vicon Motion Systems, Centennial, CO) capturing a 4-ball infrared reflecting marker set attached to each vertebral body. Anatomical landmarks were digitized and local coordinate systems were established for each vertebral body and Euler angles were computed for each direction of bending in accordance with ISB recommendations²⁶. ROM and stiffness were quantified for each functional spinal unit²⁷.

Loading and Boundary Condition of Finite Element Model

Loading of the FE model mimicked that of the experimental protocol for direct comparison and validation. Moments of up to 7 Nm were applied to the superior vertebral body (L3), which was allowed to translate in the axial direction. The sacrum was allowed only to linearly translate in the anterior-posterior and lateral directions to minimize inferior shear forces. Displacement of four independent nodes on each vertebral body were recorded and used to establish local coordinate systems for each vertebra and compute range of motion and bending stiffness similar to the experimental protocol.

Pathological Conditions

Intervertebral Disc Degeneration—Discs were included from the entire degeneration spectrum (7 grade 1, 12 grade 2, 17 grade 3, 11 grade 4, 7 grade 5) for the cadaveric

validation (Figure 3). Healthy was defined as grade 1 and 2; moderate as grade 3, and severe as grade 4 and 5.

In silico degeneration was simulated in three stages as Healthy, Moderate, and Severe corresponding to Pfirrmann grades of 1, 3, and 5, respectively for all levels. Degeneration was modeled only by altering the compressive material properties of the solid matrix for each the AF (neo-Hookean) and NP (linear elastic). The material properties were changed as a function of degeneration severity using published values (Table 3)^{10,23}. ROM and bending stiffness was calculated for each evaluation.

Ligament Failure—To investigate the role of ligamentous pathology, sequential ligament failure –total absence of the structure – was implemented in four stages: Whole Model, Remove SSL/ISL/ITL/LF, Remove FCL, and Remove ALL/PLL (Figure 4). The effect of ligament failure was examined for each stage of degeneration, and ROM and bending stiffness was measured.

Validation

Validation of the FE model was performed with cadaveric experimental work. Intersegmental and global mean and standard deviations of ROM and bending stiffness were calculated for the experimental work as well as the FE models²⁷. This resulted in the average of 18 cadaveric specimens, having a normal distribution of disc health, and 3 FE models – Healthy, Moderate, and Severe.

Results

Intersegmental (L3-L4, L4-L5, L5-Sacrum) and global (L3-Sacrum) ROM and bending stiffness averaged throughout degeneration showed very good agreement between the FE model outputs and experimental findings for flexion, extension, lateral bending, and axial rotation (Figure 5). Table 4 shows the experimental response to degeneration.

Degeneration resulted in a progressive decrease in modeled ROM for all bending directions, whereas stiffness increased only for flexion, extension, and lateral bending (Table 5). Compared to the healthy discs, moderate and severe degeneration decreased Flexion ROM 10% and 45%; extension ROM 15% and 57% lateral bending ROM 25% and 68%, respectively. A larger change in stiffness was noted, particularly for lateral bending, displaying a 28% increase from healthy to moderate and 194% increase from healthy to severe degenerative. Whereas flexion and extension – from healthy to severe – only increased 22% and 62%, respectively. Axial rotation stiffness increased initially for moderate degeneration, then decreased when modeled as severely degenerated. Figure 6 displays the L4-L5 level, but these trends were observed for all levels. Healthy to severe intersegmental ROM ranges are as follows (L3-L4, L4-L5, L5-S1): flexion (3.09–1.97, 4.64–2.57, 3.89–2.46), extension (3.87–2.27, 6.42–2.76, 6.09–3.04), lateral bending (3.90–1.32, 3.79–1.21, 3.42–1.27), and axial rotation (2.05–1.49, 2.76–1.79, 1.98–1.57).

Incremental ligament failure produced an increase in ROM and decrease in stiffness. Table 5 displays the percent changes in the biomechanical response from the initial model for the

L4-L5 level with moderate degeneration. Healthy and moderate degeneration exhibited similar results to each other. Severe degenerative discs shows the same overall general trend, but the response was less pronounced. Regarding the moderately degenerated discs, flexion was primarily restrained by the FCL, accounting for approximately 80% of the increase in ROM and 60% of the stiffness decreased observed. No change was measured for extension when removing the SSL, ISL, ITL, or LF. A 12.5 % increase in ROM and 25% decrease in stiffness occurred when removing the FCL. Following complete removal of all ligaments, there was a 41% increase in ROM and 45% decrease in stiffness during extension. Lateral bending, however, only experienced small changes in ROM and stiffness after removal of the surrounding ligaments (2% increase in ROM; 2% decrease in stiffness). Axial rotation was affected by all groups of ligament removal. Figure 7 graphically shows the changes associated with ligament failure for all stages of degeneration.

Discussion

A three-dimensional, non-linear FE model of the lumbar spine (L3-Sacrum) was generated with geometry based on a high resolution CT scan of a 52 year-old male cadaver spine. The initial portion of this study focused on the development and validation of a lumbar spine FE model capable of accurately representing the biomechanical responses observed throughout degeneration by comparison with a similar cohort of cadaveric specimens spanning the degeneration spectrum. The prediction of the FE model to moment loading in all three planes of bending showed very good agreement, where global and intersegmental ROM and bending stiffness of the model fell within one standard deviation of the *in vitro* results.

Three grades of degeneration (healthy, moderate, severe) were modeled by changing the compressive material properties of the NP and surrounding AF in accordance with values obtained in the literature^{10,23}. Worsening degeneration decreased ROM for all directions. The results for flexion, extension, and lateral bending are consistent with findings of our experimental work used for validation⁷. They also compare with previous studies; however these show an increase in ROM for axial rotation^{28,29}. Other studies have shown conflicting ROM reports, including the conventional Kirkaldy-Willis model describing a progressive increase in ROM until restabilization. A majority of the differences can be explained by the method of determining degeneration (MRI, radiographs, and dissection) and inclusion of active musculature³⁰. These discrepancies in axial rotation may be due to our model not including facet morphology or degeneration, which has been shown to be associated with disc degeneration impacting axial rotation mechanics^{31,32}. These may also explain the poor agreement between extension ROM between the model and experimental work, where only a slight change was observed experimentally, but a twofold decrease was observed computationally. Bending stiffness increased for flexion, extension, and lateral bending, but decreased for axial rotation. These findings are directly supported by our experimental work⁷. The published literature also substantiates these results, where it is reported that degeneration significantly increases the stiffness of each functional spinal unit for all directions except axial rotation, where it is shown to decrease^{33–35}.

The second portion of the study focused on ligament failure and, as hypothesized, progressive ligament failure caused incremental increased ROM and decreased stiffness

response in all bending directions. Though, this relationship was quite muted for the lateral bending direction. Previous studies have investigated the role of ligament failure on a single L4-L5 spinal unit and similarly reported an increase in motion with loss of surrounding stabilizing structures^{12,13}. Each of these studies observed a larger response in the lateral bending direction than reported herein. The inclusion of adjacent segments or differences in loading and boundary conditions may have been the reason these dissimilarities arose. The largest change measured in this study for flexion occurred following the failure of the facet capsular ligament, whereas the removal of the ALL resulted in the largest increase in ROM during extension indicating their respective importance during sagittal plane motion. Interestingly, axial rotation ROM incrementally increased stepwise, whereas the most superficial ligaments (SSL/ISL/ITL/LF) predominately affected stiffness. Lateral bending was the least affected, where only a minor (2%) alteration as observed at the L4-L5 level (Table 5).

Modeling of degeneration altered the mechanical function of the lumbar. Even though flexion-extension provides substantially more motion and is the focus of many clinical exams, alterations due to degeneration were particularly prominent during lateral bending. There was a 68% decrease in ROM and 194% increase in stiffness at the L4-L5 level from healthy to severe degeneration. Of higher importance is the ability to detect early changes of degeneration from a functional standpoint. Lateral bending displayed a higher percent change between healthy and moderate than any other bending direction (Table 5). Even though flexion-extension is the focus of functional motion as it relates to spinal health, the effects of degeneration are less noticeable. Furthermore, the changes observed *in vivo* with respect to degeneration are often combined with ligamentous degradation. Lateral bending function has little to no influence from these structures as demonstrated by progressive ligament failure. The importance of lateral bending, and associated changes with spinal health, are often overlooked. Indeed, coronal plane bending has been the focus of several recent publications highlighting its ability in detecting instability and changes associated with degeneration⁶.

As is the case with all *in silico* studies, there are several limitations that need to be addressed. Simplifications in regard to geometrical structures, in particular the ligaments and components of the disc, may lead to idealized results. Additionally, simplified material properties were utilized, and in future studies more appropriate viscoelastic material properties of the IVD will be included to quantify the neutral zone during cyclical bending activities. The degenerative model did not include decreased disc height, annular tears, ligamentous laxity, osteophytes, or facet deterioration due to software limitations to be addressed in subsequent analyses. This study did not contain a follower load because the experimental set-up used for validation did not permit. Future studies aim to incorporate these advancements and investigate the three-dimensional helical axis patterns, off-axis bending, IVD and facet contact pressures, as well as cartilaginous endplate perturbations when including physiological compressive and shear forces.

A properly generated and validated FE model can be a powerful tool to non-invasively assess a variety of clinical conditions and questions. The present study has developed a robust sophisticated FE model of the lumbar spine (L3-Sacrum) that accurately represents

the mechanical output through IVD degeneration. Progressive ligament failure was modeled and mechanical output was compared to degeneration effects, where it was shown that coronal plane motion was sensitive to changes in disc health, but not ligament pathology. These results indicate that lateral bending may be more apt to detect the subtle changes associated with degeneration, without being masked by associated changes of surrounding stabilizing structures.

Acknowledgments

Funding was provided through NIH/NIAMS grants T32 AR050938 and T32 AR056950.

References

1. Andersson GB. Epidemiological features of chronic low-back pain. *Lancet*. Aug 14; 1999 354(9178):581–585. [PubMed: 10470716]
2. Adams MA. Biomechanics of back pain. *Acupunct Med*. Dec; 2004 22(4):178–188. [PubMed: 15628775]
3. Urban JP, Roberts S. Degeneration of the intervertebral disc. *Arthritis Res Ther*. 2003; 5(3):120–130. [PubMed: 12723977]
4. White, AA.; Panjabi, MM. *Clinical biomechanics of the spine*. Philadelphia: Lippincott; 1978.
5. Posner I, White AA 3rd, Edwards WT, Hayes WC. A biomechanical analysis of the clinical stability of the lumbar and lumbosacral spine. *Spine (Phila Pa 1976)*. Jul-Aug;1982 7(4):374–389. [PubMed: 7135070]
6. Zhao F, Pollintine P, Hole BD, Dolan P, Adams MA. Discogenic origins of spinal instability. *Spine (Phila Pa 1976)*. Dec 1; 2005 30(23):2621–2630. [PubMed: 16319748]
7. Ellingson AM, Mehta H, Polly DW, Ellermann J, Nuckley DJ. Disc degeneration assessed by quantitative T2* (T2 star) correlated with functional lumbar mechanics. *Spine (Phila Pa 1976)*. Nov 15; 2013 38(24):E1533–1540. [PubMed: 23921323]
8. Ellingson AM, Nuckley DJ. Altered helical axis patterns of the lumbar spine indicate increased instability with disc degeneration. *J Biomech*. Jan 21; 2015 48(2):361–369. [PubMed: 25481221]
9. Natarajan RN, Williams JR, Andersson GB. Modeling changes in intervertebral disc mechanics with degeneration. *J Bone Joint Surg Am*. Apr; 2006 88(Suppl 2):36–40. [PubMed: 16595441]
10. Ruberte LM, Natarajan RN, Andersson GB. Influence of single-level lumbar degenerative disc disease on the behavior of the adjacent segments--a finite element model study. *J Biomech*. Feb 9; 2009 42(3):341–348. [PubMed: 19136113]
11. Rohlmann A, Zander T, Schmidt H, Wilke HJ, Bergmann G. Analysis of the influence of disc degeneration on the mechanical behaviour of a lumbar motion segment using the finite element method. *J Biomech*. 2006; 39(13):2484–2490. [PubMed: 16198356]
12. Alapan Y, Demir C, Kaner T, Guclu R, Inceoglu S. Instantaneous center of rotation behavior of the lumbar spine with ligament failure. *J Neurosurg Spine*. Jun; 2013 18(6):617–626. [PubMed: 23600587]
13. Heuer F, Schmidt H, Klezl Z, Claes L, Wilke HJ. Stepwise reduction of functional spinal structures increase range of motion and change lordosis angle. *J Biomech*. 2007; 40(2):271–280. [PubMed: 16524582]
14. Silva MJ, Wang C, Keaveny TM, Hayes WC. Direct and computed tomography thickness measurements of the human, lumbar vertebral shell and endplate. *Bone*. Jul-Aug;1994 15(4):409–414. [PubMed: 7917579]
15. Edwards WT, Zheng Y, Ferrara LA, Yuan HA. Structural features and thickness of the vertebral cortex in the thoracolumbar spine. *Spine (Phila Pa 1976)*. Jan 15; 2001 26(2):218–225. [PubMed: 11154545]

16. Fazzalari NL, Parkinson IH, Fogg QA, Sutton-Smith P. Antero-postero differences in cortical thickness and cortical porosity of T12 to L5 vertebral bodies. *Joint Bone Spine*. May; 2006 73(3): 293–297. [PubMed: 16488642]
17. Goel VK, Grauer JN, Patel T, et al. Effects of charite artificial disc on the implanted and adjacent spinal segments mechanics using a hybrid testing protocol. *Spine (Phila Pa 1976)*. Dec 15; 2005 30(24):2755–2764. [PubMed: 16371899]
18. Cvijanovic O, Bobinac D, Zoricic S, et al. Age- and region-dependent changes in human lumbar vertebral bone: a histomorphometric study. *Spine (Phila Pa 1976)*. Nov 1; 2004 29(21):2370–2375. [PubMed: 15507797]
19. Goel VK, Monroe BT, Gilbertson LG, Brinckmann P. Interlaminar shear stresses and laminae separation in a disc. Finite element analysis of the L3-L4 motion segment subjected to axial compressive loads. *Spine (Phila Pa 1976)*. Mar 15; 1995 20(6):689–698. [PubMed: 7604345]
20. Shirazi-Adl A, Ahmed AM, Shrivastava SC. A finite element study of a lumbar motion segment subjected to pure sagittal plane moments. *J Biomech*. 1986; 19(4):331–350. [PubMed: 3711133]
21. Schmidt H, Kettler A, Heuer F, Simon U, Claes L, Wilke HJ. Intradiscal pressure, shear strain, and fiber strain in the intervertebral disc under combined loading. *Spine (Phila Pa 1976)*. Apr 1; 2007 32(7):748–755. [PubMed: 17414908]
22. Shirazi-Adl A. Finite-element evaluation of contact loads on facets of an L2-L3 lumbar segment in complex loads. *Spine (Phila Pa 1976)*. May; 1991 16(5):533–541. [PubMed: 2052996]
23. Schmidt H, Heuer F, Drumm J, Klezl Z, Claes L, Wilke HJ. Application of a calibration method provides more realistic results for a finite element model of a lumbar spinal segment. *Clin Biomech (Bristol, Avon)*. May; 2007 22(4):377–384.
24. Pfirrmann CW, Metzdorf A, Zanetti M, Hodler J, Boos N. Magnetic resonance classification of lumbar intervertebral disc degeneration. *Spine (Phila Pa 1976)*. Sep 1; 2001 26(17):1873–1878. [PubMed: 11568697]
25. Wheeler DJ, Freeman AL, Ellingson AM, et al. Inter-laboratory variability in in vitro spinal segment flexibility testing. *Journal of Biomechanics*. 2011; 44(13):2383–2387. [PubMed: 21764061]
26. Wu G, Siegler S, Allard P, et al. ISB recommendation on definitions of joint coordinate system of various joints for the reporting of human joint motion—part I: ankle, hip, and spine. *International Society of Biomechanics. J Biomech*. Apr; 2002 35(4):543–548. [PubMed: 11934426]
27. Wilke HJ, Wenger K, Claes L. Testing criteria for spinal implants: recommendations for the standardization of in vitro stability testing of spinal implants. *Eur Spine J*. 1998; 7(2):148–154. [PubMed: 9629939]
28. Mimura M, Panjabi MM, Oxland TR, Crisco JJ, Yamamoto I, Vasavada A. Disc degeneration affects the multidirectional flexibility of the lumbar spine. *Spine (Phila Pa 1976)*. Jun 15; 1994 19(12):1371–1380. [PubMed: 8066518]
29. Kettler A, Rohlmann F, Ring C, Mack C, Wilke HJ. Do early stages of lumbar intervertebral disc degeneration really cause instability? Evaluation of an in vitro database. *Eur Spine J*. Apr; 2011 20(4):578–584. [PubMed: 21125299]
30. Kirkaldy-Willis WH, Farfan HF. Instability of the lumbar spine. *Clinical orthopaedics and related research*. May. 1982 (165):110–123. [PubMed: 6210480]
31. Fujiwara A, Tamai K, Yamato M, et al. The relationship between facet joint osteoarthritis and disc degeneration of the lumbar spine: an MRI study. *Eur Spine J*. 1999; 8(5):396–401. [PubMed: 10552323]
32. Fujiwara A, Lim TH, An HS, et al. The effect of disc degeneration and facet joint osteoarthritis on the segmental flexibility of the lumbar spine. *Spine (Phila Pa 1976)*. Dec 1; 2000 25(23):3036–3044. [PubMed: 11145815]
33. Houghton VM, Lim TH, An H. Intervertebral disk appearance correlated with stiffness of lumbar spinal motion segments. *AJNR Am J Neuroradiol*. Jun-Jul; 1999 20(6):1161–1165. [PubMed: 10445464]
34. Zirbel SA, Stolworthy DK, Howell LL, Bowden AE. Intervertebral disc degeneration alters lumbar spine segmental stiffness in all modes of loading under a compressive follower load. *Spine J*. Sep; 2013 23(9):1134–1147. [PubMed: 23507531]

35. Brown MD, Holmes DC, Heiner AD. Measurement of cadaver lumbar spine motion segment stiffness. *Spine (Phila Pa 1976)*. May 1; 2002 27(9):918–922. [PubMed: 11979161]
36. Shirazi-Adl SA, Shrivastava SC, Ahmed AM. Stress analysis of the lumbar disc-body unit in compression. A three-dimensional nonlinear finite element study. *Spine (Phila Pa 1976)*. Mar; 1984 9(2):120–134. [PubMed: 6233710]
37. Natarajan RN, Williams JR, Andersson GB. Recent advances in analytical modeling of lumbar disc degeneration. *Spine (Phila Pa 1976)*. Dec 1; 2004 29(23):2733–2741. [PubMed: 15564922]
38. Skaggs DL, Weidenbaum M, Iatridis JC, Ratcliffe A, Mow VC. Regional variation in tensile properties and biochemical composition of the human lumbar annulus fibrosus. *Spine (Phila Pa 1976)*. Jun 15; 1994 19(12):1310–1319. [PubMed: 8066509]
39. Ferguson SJ, Ito K, Nolte LP. Fluid flow and convective transport of solutes within the intervertebral disc. *J Biomech*. Feb; 2004 37(2):213–221. [PubMed: 14706324]
40. Chazal J, Tanguy A, Bourges M, et al. Biomechanical properties of spinal ligaments and a histological study of the supraspinal ligament in traction. *J Biomech*. 1985; 18(3):167–176. [PubMed: 3997901]
41. Pintar FA, Yoganandan N, Myers T, Elhagediab A, Sances A Jr. Biomechanical properties of human lumbar spine ligaments. *J Biomech*. Nov; 1992 25(11):1351–1356. [PubMed: 1400536]
42. Panjabi MM, Greenstein G, Duranceau J, Nolte LP. Three-dimensional quantitative morphology of lumbar spinal ligaments. *J Spinal Disord*. Mar; 1991 4(1):54–62. [PubMed: 1807531]

Figure 1. Lumbar Spine Finite Element Development

A: Segmented high-resolution CT scan of the lumbar spine used for the model. **B:** Three-dimensional vertebral and disc geometry. **C:** L3-Sacrum finite element mesh.

Figure 2. Lumbar spine mesh with included spinal ligaments and section view of the vertebral body, endplates, and intervertebral disc with fibers.

Figure 3. Representative T2 weighted MR images of discs from experimental work with corresponding Pfirrmann grade²⁴

Eighteen cadaveric specimens were included with discs from the entire degeneration spectrum were included (7 grade 1, 12 grade 2, 17 grade 3, 11 grade 4, 7 grade 5).

Figure 4.
Model description with progressive ligament failure

Figure 5. Summary statistics comparing Model and Experimental output for validation
Mean and standard deviations of range of motion and bending stiffness were calculated for cadaveric experimental work across the degenerative spectrum as well as the FE model for Healthy, Moderate, and Severe degeneration.

Figure 6. L4-L5 Range of Motion and Bending Stiffness Throughout Degeneration from FE model

ROM progressively decreased with degeneration for all bending directions. Stiffness increased for flexion, extension, and lateral bending. Axial rotation stiffness increased initially for moderate degeneration, then decreased when modeled as severely degenerated.

Figure 7. L4-L5 Range of Motion and Bending Stiffness with progressive ligament failure from FE model

ROM incrementally increased and stiffness decreased with ligament failure for all bending directions. Lateral bending displayed only minor changes.

Table 1

Material Properties used in the Finite Element Model

Element Set	Number of Elements	ABAQUS Element Library	Type	Elastic Modulus (MPa)	Poisson's Ratio, ν	Cross-Sectional Area (mm ²)
Vertebral Cortical Bone ^{1,2}	1130	C3D8		12000	0.30	
Vertebral Cancellous Bone ^{1,2}	10849	C3D8		100	0.20	
Vertebral Bony Endplate ^{3,4}	1862	C3D8		4000	0.30	
Posterior Bone ^{1,2}	13766	C3D8 C3D10 C3D20		3500	0.25	
Sacrum & Coccyx ^{1,2}	29295	C3D10 C3D20		3500	0.25	
Annular Layers ^{4,5,6}	3990	C3D8	Neo-Hooke See Table 3			
Annular Fibers ^{1,7}	7980	T3D2		71.5 – 110	0.30	0.03475 – 0.1373
Nucleus Pulposus ^{4,5,6}	1455	C3D8		See Table 3		
Cartilage Endplate ⁸	679	C3D8		5	0.17	
Apophyseal Joints ⁴	732			Softened, 3500		
¹ 19;						
² 36;						
³ 15;						
⁴ 23;						
⁵ 37;						
⁶ 10;						
⁷ 38;						
⁸ 39						

Table 2

Ligament Material Properties used in the finite element model

Ligaments	Number of Elements	ABAQUS Element Library Type	Elastic Modulus (MPa)	Cross-Sectional Area (mm ²)
Anterior Longitudinal ^{1,2}	180	T3D2	15.6 – 20.0	66
Posterior Longitudinal ^{1,2}	115	T3D2	10.0 – 20.0	25
Intertransverse ¹	34	T3D2	12.0 – 58.7	2
Ligamentum Flavum ^{1,2}	19	T3D2	13.0 – 19.5	39
Interspinous ^{1,2,3}	17	T3D2	9.8 – 12.0	30
Supraspinous ^{1,2,3}	9	T3D2	8.8 – 15.0	30
Capsular ^{1,3,4}	97	T3D2	7.50 – 33.0	44

¹19,²40,³41,⁴42

Table 3

Intervertebral Disc Material Properties used in the Finite Element model (AF: Annulus Fibrosus; NP: Nucleus Pulposus). AF was modeled as a neo-Hookean solid and the NP was as linearly elastic.

Disc Health	Elastic Modulus (MPa)	Poisson's Ratio, ν
Healthy ¹	AF: $c_{10} = 0.25$; $D_1 = 0.78$	AF: 0.40
	NP: 1MPa	NP: 0.4999
Moderate ²	AF: $c_{10} = 0.348$; $D_1 = 0.3$	AF: 0.40
	NP: 1.26 MPa	NP: 0.45
Severe ¹	AF: $c_{10} = 1.13$; $D_1 = 0.16$	AF: 0.40
	NP: 1.66 MPa	NP: 0.40

¹10;

²23

Table 4

ROM and Bending Stiffness Mean (SD) from Experimental Work

		Experimental Output		
		Healthy	Moderate	Severe
ROM (deg)	Flex	3.85 (1.21)	3.16 (1.50)	2.87 (1.38)
	Ext	4.73 (2.42)	4.63 (1.33)	4.52 (1.71)
	LB	3.19 (0.96)	2.92 (1.02)	2.15 (1.02)
	AR	1.62 (1.00)	1.91 (0.79)	1.91 (1.00)
Stiffness (Nm/deg)	Flex	2.35 (0.86)	3.03 (0.86)	3.11 (1.53)
	Ext	3.02 (0.87)	3.18 (0.88)	3.04 (1.08)
	LB	2.83 (0.67)	3.52 (1.42)	4.46 (2.57)
	AR	6.99 (3.47)	6.26 (3.32)	5.84 (2.92)

Table 5

L4-L5 mechanical response resulting from disc degeneration (% change from Healthy) and ligament failure (% change from Whole) – determined from FE model.

		Ligament Failure						
		Degeneration						
		Healthy	Moderate	Severe	Whole	Remove SSL/ISL/ITL/LF	Remove FCL	Remove ALL/PLL
ROM (deg)	Flex	4.64	4.16 (10.3)	2.57 (44.6)	4.16	4.40 (5.8)	7.69 (84.9)	7.75 (86.3)
	Ext	6.42	5.46 (15.0)	2.76 (57.0)	5.46	5.46 (0)	6.14 (12.5)	7.70 (41.0)
	LB	3.79	2.86 (24.5)	1.21 (68)	2.86	2.86 (0)	2.86 (0)	2.92 (2.1)
	AR	2.76	2.56 (7.2)	1.79 (35.1)	2.56	2.85 (11.3)	3.54 (38.3)	4.05 (58.2)
Stiffness (Nm/deg)	Flex	2.54	2.61 (2.8)	3.09 (21.7)	2.61	2.46 (5.8)	0.94 (64.0)	0.92 (64.8)
	Ext	1.64	1.68 (2.5)	2.66 (62.2)	1.68	1.68 (0)	1.26 (25.0)	0.91 (45.8)
	LB	1.96	2.50 (27.6)	5.77 (194.4)	2.47	2.47 (0)	2.46 (0.4)	2.41 (2.4)
	AR	4.99	5.48 (9.8)	4.23 (15.2)	5.48	3.05 (44.3)	1.72 (68.6)	1.67 (70.0)