

Simulation of the Disc Degeneration with a Poroelastic Finite Element Model

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Abstract

To predict the degeneration process in the intervertebral disc, a finite element model of the spinal motion segment model was developed. The relationship between the biomechanical characteristics of fluid and solid matrix of the disc and cancellous core of the vertebral body, modeled as 20 node poroelastic elements, during the degeneration process was investigated. Excess von Mises stress in the disc element was assumed to be a possible source of degeneration under compressive loading condition. Recursive calculation was continued until the desired convergence was attained by changing the permeability and void ratio of those elements. The degenerated disc model showed that relatively small compressive stresses were generated in the nucleus elements compared to normal disc. Its distribution along the sagittal plane was consistent with a previously reported experimental result. Contrasts to this result, pore pressures in the nucleus were higher than those in the normal disc. Total stress, sum of compressive stress and pore pressure, indicated similar values for two different models. This study presented a new approach to study the likely mechanism responsible for the initiation and progression of the degenerative process within the intervertebral disc.

Keywords: Disc; Degeneration; FE model; Poroelastic element; Void ratio

1. Introduction

Intervertebral discs (IVD) are of considerable medical interest because damaged and degenerated discs are believed to be the most leading source of the severe pain (Kuslich et al., 1991). In the biology, disc degeneration is a chronic remodeling process that results in alterations of matrix composition and decreased cellularity. The gel (sometimes called the notochordal sheath) loss of disc inwards leads to faster degeneration and disc space narrowing is the most sensitive sign for disc degeneration (Ghosh, 1988). Disc degeneration is used to describe situations

where the internal structure shows pathological changes with or without prolapse. Degeneration, which can only be seen in sectioned discs, involves disorganization of the lamellar structure, ruptures (either internal or penetrating), sometimes calcification of the disc centre, and a brown discoloration that is often, if not always, due to hemorrhage.

Disc degeneration is a very complex phenomenon that will require extensive research both biomechanically and biochemically before it can be fully understood. Biochemically, researchers were trying to find the cause of disc degeneration using several techniques, including studies of mechanical effects on the cell activity and cell viability (Natarajan et al., 2004). The disc degeneration is a cascading event that is often attributed to the cumulative damage caused

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by heavy physical work or vibration. Analysis of degeneration process using experimental method has been performed in lumbar disc biomechanics. Kim *et al.* (2003) investigated the origin of chondrocytes forming fibrocartilage in the nucleus pulposus and the mechanisms of transition. They suggested that the newly formed fibrocartilage nucleus pulposus is the true normal state of the adult nucleus pulposus, and that disc degeneration ensues after the end stage of transition (the formation of the fibrocartilage nucleus pulposus). The use of analytical models has been suggested as a way to overcome the limitations of experimental investigations. In the recent past, three-dimensional (3D) finite element (FE) models of human spinal segments have been developed, and it has become possible to analyze the effect of disc degeneration (Kim *et al.*, 1991; Natarajan *et al.*, 1994). While these biomechanical studies provide some insight into the types of injuries, these studies are limited in their ability to identify tissue failure initiation and propagation. Due to the poroelastic characteristics of the fluid in the IVD and cancellous core, it is not enough to analyze the initiation and progression of disc degeneration with continuum element as many investigators have done.

In the current study, spinal one motion segment, modelled as 3D poroelastic FE model, was developed to incorporate the biomechanical interaction between the solid skeletal structure and fluid in the IVD, and the degeneration process in the IVD was performed with this developed model.

2. Methods

2.1 Poroelastic FE model

A FE models were developed to study the disc degeneration and its associated changes in biomechanical properties. A simplified 3D nonlinear FE model of the L3–L4 vertebral bodies and disc, previously developed (Lee *et al.*, 2000) using computed tomography (CT) transverse sections, was modified to incorporate posterior element. (Fig. 1)

For the cortical shell of the vertebral body, 8 node shell elements were used. Posterior elements including facet contacts were also added to the model. The cancellous core of the vertebral body and the endplate were simulated using homogeneous 20-node brick elements. 27 different porosities (Keller *et al.*, 1989) and permeability were assigned to the grouped block of the vertebral body which mimics the real

Table 1. Material properties used to simulate various spinal elements in the model.

Material	Young's modulus (MPa)	Initial Permeability (m^4/Ns)	Poisson's Ratio	Initial Void Ratio
Cortical bone	10000	-	0.25	
Cancellous bone	100	5.00E-06 ~ 2.00E-05	0.25	4.0–5.2
Posterior bone	3500	-	0.25	
Endplate	10000	5.00E-12	0.25	4.0
Annulus	5	1.00E-14	0.45	3.0
Nucleus	2	2.00E-13	0.49	6.0

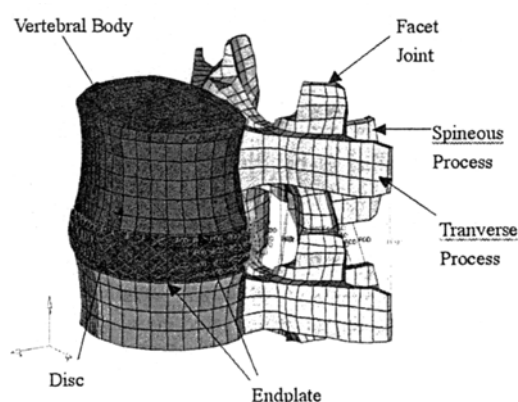


Fig. 1. Developed motion segment FE model used for the analysis.

vertebral body structure. To simulate the force resistance mechanism in the IVD, using the fluid flow between the vertebral body and the disc via the endplate, the cancellous core and the vertebral endplate were modelled as poroelastic material. The IVD was modelled as composed of nucleus pulposus and annulus fibrosus to study disc degeneration. The annulus fibrosus was made of a porous and fiber reinforced material. The orientation of the annulus fibers, modeled as 3D tension-only truss elements in individual lamellas, alternated with successive layers positioned at 70° and -70° in relation to the vertical axis. The ground substance of the annulus fibrosus and nucleus pulposus was modeled as brick elements with 20-node quadratic displacement and linear pore pressure. Table 1 shows material properties used to simulate various spinal elements.

The vertebral bodies are connected by the anterior and posterior longitudinal ligaments, parallel to the axis of the spine, as well as by the disc. Further

ligaments connecting the vertebral arches of adjacent vertebrae, e.g., the ligament flavum, interspinous ligament and supraspinous ligament were also included in the model. Facet joint, modeled as contact elements, are conventional synovial joints in which the capsular ligaments confer further stability on the spine.

2.2 Degeneration process

Generated nucleus pressure under various loading condition can be balanced by tension in the collagen fibers of the annulus, and this tends to uniformly distribute the stress generation in the IVD. We were trying to analyze overall degeneration process on the motion segment and not to predict absolute stresses with a particular region. Therefore, relatively high compressive force (1200 N, three times of the trunk weight) was chosen for this analysis, although combined loading is believed to produce relatively high risk on the IVD because of its geometrical uniqueness. Von Mises stress in the disc element was investigated to be a possible source of degeneration. Elements with excess stress more than the average value were selected as degenerated element, to which degenerated material properties were assigned. The initial void ratio of the annulus matrix was assumed to be 3. This corresponded to the 71% water content of the total tissue weight in the annulus. This agreed with the measurement of Kraemer *et al.* (1985), in which the percentage varied between 70% and 75%. The nucleus pulposus also was modeled as a poroelastic element, with an initial void ratio of 6, which implied an 85% fluid content for a normal disc.

Degeneration process of the IVD was studied by changing the void ratio and permeability of the degenerated element. While iterating the degeneration process of the IVD, proper void ratio of an annulus fibrosus and a nucleus pulposus was determined by using the water content of the IVD at different ages, as referred to the measurement of Kraemer *et al.* (1985). The water content of the annulus fibrosus was about 74% in the first decade of life, decreased to 67% by the age of third decade, and then increased to 70% by the age of sixth decade. That of the nucleus pulposus also decreased from 85% in the first decade of life to 75% in the sixth decade. In our calculation, the void ratios of each decade were selected for each iteration procedure, hereupon, element showed excessive stress value changed its void ratio of next

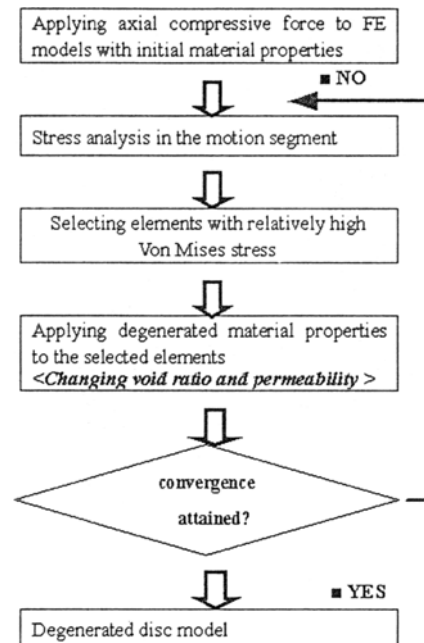


Fig. 2. Analysis algorithm of the disc degeneration.

decade. Recursive calculation was continued until the desired convergence was attained by changing the permeability and porosity of those elements, which could be predicted from the previous iteration.

The permeability (k) of annulus and nucleus was assumed using Li *et al.* (1999)' Eq. (1) and calculated by the value of void ratio (e).

$$k = k_0 \exp\left(M \frac{e - e_0}{1 + e_0}\right) \quad (1)$$

where, k_0 and e_0 , indicated initial value of permeability and void ratio respectively. M was a constant used to match material properties.

Figure 2 shows an analysis algorithm of the disc degeneration in the present study.

Swelling pressure was not considered. A zero pressure boundary condition was applied at the peripheral surface of the disc. The general purpose finite element program ABAQUS (Hibbit, Karlsson and Sorenson, Inc., version 6.4) was used for the execution of the models.

3. Results

Degeneration process of the IVD was analysed in a poroelastic spinal motion segment FE model. The

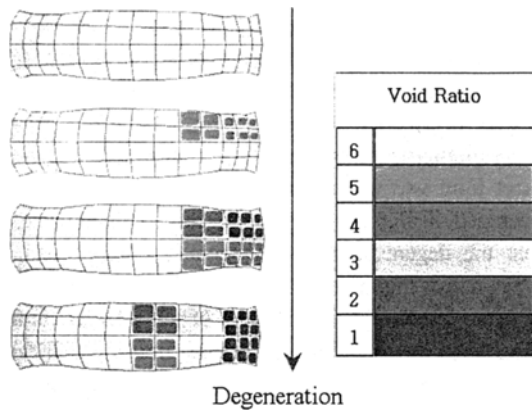


Fig. 3. Progress of the disc degeneration (figure shows sagittal plane of the disc).

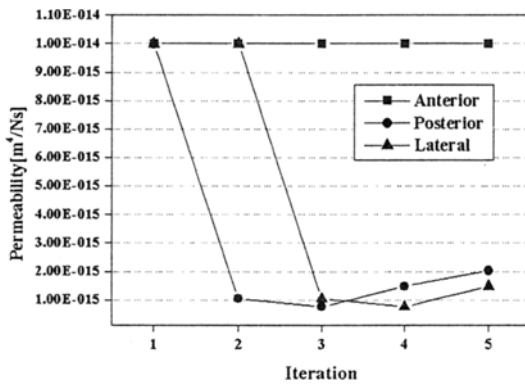


Fig. 4. Permeability changes in the annulus.

degeneration, as shown in Fig. 3, initiated at the posterior region of the nucleus and then progressed to the centre of the nucleus.

Degeneration was also generated in the posterior region of the annulus. It was originated upper layer of the annulus and propagated to the lower layer of the annulus. As shown in Fig. 4, the permeability in the annulus was dramatically changed at the posterior region, followed by the lateral region of the annulus. The permeability decreased with water content (void ratio) and that the water content varied inversely with iteration step i.e., degeneration step.

Distribution of compressive stress and pore pressure along the midagittal plane of the disc are shown in Fig. 5. Maximum compressive stress was found at the region of the posterior annulus of de-generated disc. The degenerated disc model showed that relatively small compressive stresses were generated in the nucleus elements compared to the normal disc.

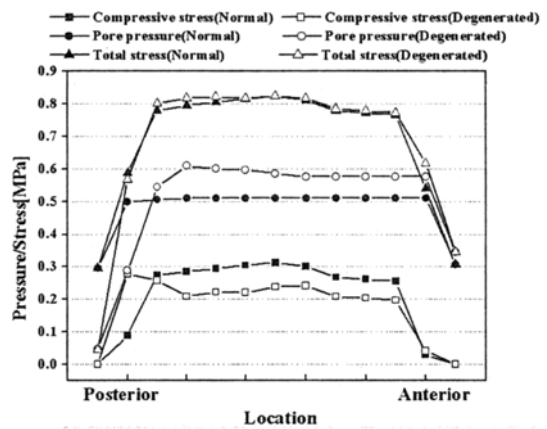


Fig. 5. Variations of pore pressure, compressive stress, and total compressive stress (sum of the two) in the midsagittal plane of the disc.

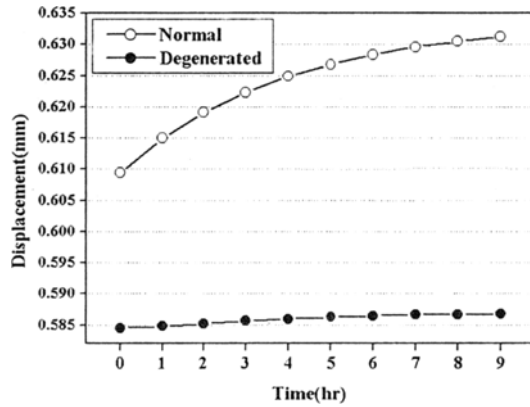


Fig. 6. Creep responses in normal and degenerated disc.

Contrasts to this result, pore pressures in the nucleus were higher than those in the normal disc. Total stress, sum of compressive stress and pore pressure, indicated similar values for two different models (normal and degenerated discs).

The predicted temporal variation of axial displacement under compression load indicated that the response markedly stiffened when the degeneration progressed. The void ratio dependent permeability substantially increased the disc stiffness by increasing the resistance of the fluid flow in the IVD. Figure 6 shows the creep phenomena of the normal and degenerated discs. The pore pressure decreased with time as ground structure deforms. Relatively hard-ened ground structure due to degeneration provided small variation of axial displacement with time.

4. Discussion

Degeneration process of IVD was studied with a poroelastic spinal motion segment FE model by changing the void ratio and permeability. In this study, as processed in degeneration, relatively high von Mises stress, compared with those of normal disc, was generated at posterior region which can explain the outset of disc degeneration at this region. As shown in Fig. 3, the degeneration was progressed to the center of the nucleus pulposus. According to the experimental observation, all form of fibrocartilage lamellas and fibers found in the nucleus pulposus had originated and migrated from the cartilage endplate and proceeded in a centripetal direction (Lotz et al., 1998). Compressive experiment (Lotz and Chin, 2000) also indicated that extensive cell death was noted in the notochordal cells with in the nucleus pulposus at the long time duration and high stress.

As the degeneration proceeded, the permeability in the posterior region of annulus decreased to $1.0 \times 10^{-15} \text{ m}^4/\text{Ns}$ that was closed to the previous experimental observation (Gu et al., 1999). The hydraulic permeability has been determined from both compressive loading and direct permeation test with values of 0.2 to $2 \times 10^{-15} \text{ m}^4/\text{Ns}$. In the degeneration process, we could not consider the incidence of the tear in the ground matrix of the annulus, and consequently our result was only compared with an experimental result in radial direction. This permeability change followed by the degeneration should have a further influence on the cell nutrition and biomechanical etiology of disc failure.

Same as most of disc degeneration studies, our analysis also focused on the physical stress and overt tissue damage. Significant structural failure such as radial fissure, prolapse, annular protrusion, and disc space narrowing accompanied in the disc degeneration, were not considered even though those biological factors accelerate disc degeneration. Therefore, calculated total compressive stress showed almost same values as shown in the Fig. 5. Previous experimental researches (Brown et al., 2002; Ralph et al., 2006) also indicated that there were no significant stiffness differences between normal and degenerated disc, even though neutral zone of the degenerated discs were greater than those of the normal discs. Relatively large pore pressure was generated in the degenerated disc compared to normal disc, whereas approximately 25% of compressive stress was re-

duced in the solid structure of degenerated disc. Pollintine et al. (2004) also showed similar results by measuring compressive stress in the disc after reducing discs water content. They found that the proportion of compressive force transmitted by the facet joint was increased from less than to 20% to averaged 49% in the degenerated disc. This indicates that the degeneration process makes pore pressure play more important role to resist against the external force. The fluid content of the intervertebral disc is important in determining its mechanical response and also its transport and biologic properties. Also increased hydrostatic pressure (pore pressure) directly affects matrix protein gene expression, synthesis rate, and degradation of the IVD (Lotz and Chin, 2000).

The loss of fluid content accompanied by the disc degeneration affects the creep pattern of the disc. Reducing the water content of the nucleus makes the disc better adapted to withstand high compressive forces, because the nucleus is then less likely to prolapse through the posterior annulus (Adams et al., 1987). As shown in Fig. 6, this supports the predicted variation of axial displacement in both normal and degenerated discs. However, increasing compressive stress brought about annulus disorganization in the inner and middle annulus (Lotz et al., 1998), and then the mechanical property change will be produced at this region. It will provide different creep pattern. The authors are pursuing these studies further, and hope to report their findings in the future.

This study presented a new approach to study the likely mechanism responsible for the initiation and progression of the degenerative process within an IVD. Even though, this model has several shortcomings, it has the potential to provide significant insight into the relationship between mechanical factors and the degenerative changes within the IVD.

5. Conclusion

Use of the finite element technique incorporating fluid contents with poroelastic modeling to address the degeneration process in the IVD is presented. The current analysis shows how disc degeneration affects these pore pressure and solid skeleton compressive stress distributions. Variation of the role of pore pressure and stress in the solid structure provide new insights into the mechanical function of degenerated IVD.

It is generally believed that disc degeneration affects on the osteoporosis in the vertebral body and this change also affects on the disc degeneration in vice versa. Therefore, we are now extending our analysis to incorporate these phenomena for more realistic outcomes.

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References

- Adams, M. A., Dolan, P. and Hutton, W. C., 1987, "Diurnal Variations in the Stresses on the Lumbar Spine," *Spine*, Vol. 12, pp. 130~137.
- Brown, M. D., Holmes, D. C. and Heiner, A. D. 2002, "Measurement of Cadaver Lumbar Spine Motion Segment Stiffness," *Spine*. Vol. 27, pp. 918~922.
- Ghosh, P. and SC, B., 1988, "The Biology of the Intervertebral Disc," *CRC Press, USA*, pp. 99~105.
- Gu, W. Y., Mao, X. G., Foster, R. J., Weidenbaum, M. and Mow, V. C., Rawlins, B. A., 1999, "The Anisotropic Hydraulic Permeability of Human Lumbar Annulus Fibrosus," *Spine*, Vol. 24, pp. 2449~2455.
- James, C. I., Lori, A. S., Robert, J. F., Bernard, A. R., Mark, W. and Van, C. M., 1998, "Degeneration Affects the Anisotropic and Nonlinear Behaviors of Human Annulus Fibrosus in Compression," *J. Biomech.*, Vol. 31, pp. 535~544.
- Keller, T. S., Hansson, T. H., Abram, A. C., Spengler, D. M. and Panjabi, M. M., 1989, "Regional Variation in the Compressive Properties of Lumbar Vertebral Trabeculae-Effect of Disc Degeneration," *Spine*, Vol. 14, pp. 1012~1019.
- Kim, K. W., Lim, T. H., Kim, J. G., Jeong, S. T., Masuda, K. and An, H. S., 2003, "The Origin of Chondrocytes in the Nucleus Pulposus and Histologic Findings Associated with the Transition of a Notochordal Nucleus Pulposus to a Fibrocartilaginous Nucleus Pulposus in Intact Rabbit Intervertebral Discs," *Spine*, Vol. 28, pp. 982~990.
- Kim, Y. E., Goel, V. K., Weinstein, J. N. and Lim, T., 1991, "Effect of Disc Degeneration at One Level on the Adjacent Level in Axial Mode," *Spine*, Vol. 16, pp. 331~335.
- Kraemer, J. D., Kolditz, M. and Gowin, R., 1985, "Water and Electrolyte Content of Human Intervertebral Discs Under Variable Load," *Spine*, Vol. 10, pp. 69~71.
- Kuslich, S. D., Ulstrom, C. L. and Michael, C. J., 1991, "The Tissue Origin of Low Back Pain and Sciatica," *Orthop. Clin. N. Am.*, Vol. 22, pp. 181~187.
- Lee, C. K., Kim, Y. E., Lee, C. S., Hong, Y. M., Jung, J. M. and Goel, V. K., 2000, "Impact Response of the Intervertebral Disc in a Finite-element Model," *Spine*, Vol. 25, pp. 2431~2439.
- Li, L. P., Soulhat, J., Buschmann, M. D. and Shirazi-Adl, A., 1999, "Nonlinear Analysis of Cartilage in Unconfined Ramp Compression Using a Fibril Reinforced Poroelastic Model," *Clin. Biomech.*, Vol. 14, pp. 673~682.
- Lotz, J. C. and Chin, J. R., 2000, "Intervertebral Disc Cell Death is Independent on the Magnitude and Duration of Spinal Loading," *Spine*, Vol. 25, pp. 1477~1483.
- Lotz, J. C., Colliou, O. K., Chin, J. R., Ducan, N. A. and Liebenberg, E., 1998, "Compression-induced Degeneration of the Intervertebral Disc ; An in Vivo Mouse Model and Finite Element Study," *Spine*, Vol. 23, pp. 2493~2506.
- Natarajan, R. N., Ke, J. H. and Andersson, G. B. J., 1994, "A Model to Study the Disc Degeneration Process," *Spine*, Vol. 19, pp. 259~265.
- Natarajan, R. N., Williams, J. R. and Andersson, G. B. J., 2004, "Recent Advances in Analytical Modeling of Lumbar Disc Degeneration," *Spine*, Vol. 29, pp. 2733~2741.
- Pollintine, P., Przybyla, A. S., Dolan, P. and Adams, M. A., 2004, "Neural Arch Load-bearing in Old and Degenerated Spines," *J. of Biomech.*, Vol. 37, pp. 197~204.
- Ralph, E. Gay, R. E., Ilharreborde, B., Zhao, K., Zhao, C. and An, K. N., 2006, "Sagittal Plane Motion in the Human Lumbar Spine: Comparison of the in Vitro Quasistatic Neutral Zone and Dynamic Motion Parameters," *Clin. Biomech.*, Vol. 21, pp. 914~949.