Original Article

Initiation and Progression of Mechanical Damage in the Intervertebral Disc under Cyclic Loading using Continuum Damage Mechanics Methodology: A Finite Element Study

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Abstract

It is difficult to study the breakdown of disc tissue over several years of exposure to bending and lifting by experimental methods. There is also no finite element model that elucidates the failure mechanism due to repetitive loading of the lumbar motion segment. The aim of this study was to refine an already validated poro-elastic finite element model of lumbar motion segment to investigate the initiation and progression of mechanical damage in the disc under simple and complex cyclic loading conditions. Continuum damage mechanics methodology was incorporated into the finite element model to track the damage accumulation in the annulus in response to the repetitive loading. The analyses showed that the damage initiated at the posterior inner annulus adjacent to the endplates and propagated outwards towards its periphery under all loading conditions simulated. The damage accumulated preferentially in the posterior region of the annulus. The analyses also showed that the disc failure is unlikely to happen with repetitive bending in the absence of compressive load. Compressive cyclic loading with low peak load magnitude also did not create the failure of the disc. The finite element model results were consistent with the experimental and clinical observations in terms of the region of failure, magnitude of applied loads and the number of load cycles survived.
1. Introduction

Low back pain is a major health condition affecting every population worldwide (Andersson, 1999). It can lead to decreased quality of life, diminished physical activity and psychological distress (Deyo and Tsui-Wu, 1987; Deyo et al., 2011). Intervertebral disc degeneration is associated with low back pain (Cheung et al., 2009; Luoma et al., 2000; Samartzis et al., 2011; Savage et al., 1997). Appearance of annular lesions has been suggested (Osti et al., 1992; Sharma et al., 2009a; Sharma et al., 2009b; Vernon-Roberts et al., 2007) as the first sign of the disc degeneration process. Epidemiological studies have identified frequent bending and lifting as a major risk for disc prolapse (Kelsey et al., 1984; KUMAR, 1990). Damage to disc structure has been reported in response to cyclic loading of the motion segment by a number of studies involving human cadavers and animal models (Adams and Hutton, 1983; Adams and Hutton, 1985; Adams et al., 2000; Goel et al., 1988; Hansson et al., 1987; Liu et al., 1983; Liu et al., 1985; Yoganandan et al., 1994). Yu et al. (2003) reported presence of irregular fibers, buckling and bleeding in the porcine annulus in response to compressive cyclic loading. Gordon et al. (1991) reported disc herniation in 14 cadaveric lumbar motion segments, subjected to combination of flexion, axial rotation and compression for an average duration of 36,750 cycles. Liu et al. (1983) subjected cadaveric lumbar motion segments to cyclic axial loads ranging from 37-80% of their failure load limit for up to 10,000 cycles. Disc injury was reported in 2 of 11 specimens while all the specimens experienced endplate or vertebral bone cracking. Parkinson and Callaghan, (2009) conducted a series of in-vitro fatigue testing on porcine motion segments to understand the failure mechanism. They concluded that cyclic flexion/extension bending results in the failure of the disc while large cyclic compressive loading fractures the vertebral body. Average numbers of load cycles for disc injury were reported to be 9000 as compared to 930 for vertebral bone fracture. Marshall and McGill, (2010) showed that cyclic
flexion/extension bending of porcine motion segments caused nucleus tracking through the posterior annulus, while cyclic axial rotation resulted in the radial delamination of the annulus. In case of the human cadaver studies, it is difficult to obtain a large number of specimens without disc degeneration or pre-existing annular disruptions. With current imaging techniques it is not possible to identify the location and extent of damage during different stages of testing without interruptions. It is difficult if not impossible to apply complex loadings that are representative of daily life activities in the cadaver testing setup. These limitations make it hard to track the initiation and progression of structural damage in the intervertebral disc under complex loading conditions in the experimental setup.

Finite element (FE) modeling has been used extensively to explore the spine biomechanics. However most of the FE models of the spine are employed to elucidate the spine kinematics under single load cycle (Goel VK et al, 1995; Argoubi M et al, 1996; Rohlmann et al, 2006; Little JP et al, 2007; Schimdt et al, 2007; Galbusera et al, 2011). Damage to disc structure had been studied using FE models but there is no FE study for lumbar spine that investigates the degradation of the disc due to cyclic loading to the best of the authors’ knowledge (Shirazi-Adl A, 1989; Natarajan RN et al, 1994; Schimdt H et al, 2009). Initiation and progression of structural damage can be tracked in a motion segment by employing user written codes in conjunction with the FE model. The purpose of this study was to employ continuum damage mechanics methodology to predict damage initiation and progression in the disc under cyclic loading using a poro-elastic FE model of a lumbar motion segment. The current analysis was restricted to the damage analyses in the annulus only. The FE model considered annulus as a single continuum body reinforced by collagen fibers instead of multilayered structure. It was hypothesized that the (a) number of load cycles to disc failure will decrease as the motion
segment is subjected to complex loading rather than uni-axial compressive loading and (b) damage will initiate and progress preferentially in the posterior region of the disc under all loading conditions.

2. Materials and Method

2.1 3D Poro-Elastic Finite Element Model of L4/L5 Lumbar Motion Segment

A previously validated (Natarajan et al., 2006; Natarajan et al., 2008; Williams et al., 2007) three dimensional non-linear poro-elastic FE model of a healthy lumbar L4-L5 motion segment was modified for the current study. It included parameters such as porosity, osmotic pressure and the strain dependent permeability. Element and material model information for the FE model are listed in the table 1 and detailed information is included in the appendix. FE analyses were carried out using a commercially available software package ADINA (ADINA R&D Inc., Watertown, Massachusetts).

2.2 Continuum Damage Mechanics

Kachanov, (1999) introduced a concept of damage being continuously distributed throughout the solid and proposed a damage variable as an internal state variable describing the state of degradation of the material. A computational methodology (Verdonschot and Huiskes, 1997) for the prediction of degradation of materials under cyclic loading based on Kachanov’s concept was employed in the current study to investigate the failure progression in the annulus. Continuum damage mechanics formulation along with the FE modeling was employed to simulate the fatigue behavior of the human cortical bone (Taylor et al., 1999). Jeffers et al. (2007) and Lennon et al. (2007) also used it to investigate the cement mantle failure and loosening of femoral components in total hip arthroplasty respectively.

2.3 Application of Continuum Damage Methodology to Lumbar Spine FE Model
In the FE model, annulus was divided into 1,920 elements. Element properties were calculated at eight integration points distributed within the element. At the beginning of the analysis each integration point in the elements representing annulus was assigned a value of zero for the damage variable $d$ representing its healthy state (Figure 1). The loading was applied to the FE model in incremental steps. At the maximum load step, principal tensile stress was calculated at each integration point in the annulus elements. The number of load cycles to failure ($N$) was calculated at each integration point in the annulus using a Stress-Failure (S-N) curve. The lowest number of cycles to failure ($N_{\min}$) corresponded to the integration point with the highest tensile stress value. Damage $d$ at each integration point was incremented as

$$(d_i)_t = (d_i)_{t-1} + \left(\frac{N_{\min}}{N_i}\right)_t$$

Where $i$ represent the integration point and $t$ represent the iteration number.

When damage $d$ for an integration point reached a predefined limit, the corresponding integration point in the element was assumed unable to share any load. The elastic modulus at the damaged integration points was reduced to a predetermined value thus introducing the degradation of the material at that location in the annulus. Even though the damage to the tissue occur only in the direction of tensile principal stress, the algorithm assumes damage equally occurs in all the three principal directions at each integrating point within an element. The number of load cycles required to cause the given damage in the annulus was equal to $N_{\min}$. The stiffness matrix was then updated. The same loading was again applied to the motion segment and damage was incremented for each integration point following the above procedure. The damage initiation and progression was tracked by recording the damaged integration points. The procedure was implemented by introducing a FORTRAN code in the ADINA subroutine (“User
Supplied Material”) that allowed changing elastic modulus at each integration point of the annulus.

2.4 Stress-Failure (S-N) Curve for Annulus

The S-N curve for the annulus was developed by using the data from a cyclic cadaver study carried out by Green et al. (1993). They tested 22 annulus slices from the anterior and posterior regions of the lumbar discs (age range 19–71 years) under different magnitudes of tensile stress for up to 10,000 cycles. In situ tensile strength of the annulus was then estimated based on the size of the specimens. The number of cycles to failure at different magnitudes of stress for individual specimens were plotted. A curve fit based on the power-law represents the S-N curve for the annulus (Figure 2). The logic behind using power-law model rather than a linear model as reported for other biological tissues (Schechtman and Bader, 1997; Wang et al., 1995) was to include the effect of endurance limit observed during cyclic testing of annulus fibrosus (Green et al., 1993).

2.5 Effect of Magnitudes of Elastic Modulus and Damage Parameter at Damaged Integrating Points on the Damage Progression

Analyses were carried out to investigate the effect of elastic modulus and damage parameter value at the damaged integration points on the damage accumulation in the annulus. For this the motion segment was subjected to a compressive cyclic loading with a peak load of 800 N. Analyses were conducted by reducing the elastic modulus at the damaged integration points to one tenth, one hundredth and one thousandth of its original value at three different values of damage parameter (0.99, 0.90, and 0.80). In total nine simulations were performed; three different values of damage parameter paired with three different values of elastic modulus at damaged integration points.
A much faster failure progression was observed when the elastic modulus was reduced to one hundredth than if it was reduced to one tenth of its original value (Figure 3). However damage progression rate did not change appreciably when elastic modulus was reduced to one thousandth rather than one hundredth of its original value (Figure 3). Thus the magnitude of the elastic modulus at the damaged integration point had a considerable effect on the rate of damage accumulation.

The damage accumulation was faster with a decreasing value of \( d \) and became slower with an increasing value of \( d \) (Figure 4). However, the difference between the three cases was not appreciable. Thus the damage parameter value at which the integration point was considered degraded did not have a considerable effect on the damage progression rate.

Same conclusions were reached from all the nine combinations. Based on the above findings it was decided to reduce the elastic modulus of the integration point to one hundredth of its original value, if its damage parameter reached a value of 0.90 for subsequent analyses.

### 2.6 Validation of the FE Model Incorporated with Damage Accumulation Formulation

The FE model incorporated with the continuum damage mechanics methodology was validated by comparing the results with the human cadaver study carried out by Gordon et al. (1991). They studied the disc rupture mechanism using 14 human lumbar motion segments (age range 18-65 years) under complex cyclic loading. 12 motion segments were from L1L2, L3L4 and L4L5 levels with four specimens at each level and two specimens were from L2L3 level. Testing was carried out under displacement control. Motion segments were subjected to 7° flexion, 0.93 ± 0.56 mm compression and 1.9 ± 0.6° axial rotation simultaneously. The testing was stopped when a sharp decrease in the forces was observed and the motion segment was considered failed at that load cycle. They reported mean failure cycles of 36,750 with a standard
deviation of 12.612. Ten discs showed annular protrusion and four showed nuclear extrusion in the posterior region. Annular tears were found in all specimens in the posterolateral region. The current FE model was subjected to 7.16° flexion accompanied by 1.09 mm axial compression and 1.67° axial rotation simultaneously in order to compare the results with the in vitro study.

2.7 Loading Conditions

In order to investigate the effect of different modes of loading on damage accumulation in a lumbar disc, simple and complex loadings were applied to the motion segment. Simple loading conditions involve the application of either the axial compressive load or the bending moments in one of the three principal directions (Table 3, Load cases 1-5). Complex loading scenarios were simulated by the application of the bending moments in single or multiple directions along with the compressive load (Load cases 6-10). Compressive load was simulated by applying a uniform pressure on the top of the superior endplate. Bending moments were simulated by applying equal and opposite forces at appropriate points on the top surface of L4 vertebra.

3. RESULTS

The FE model subjected to the loading conditions similar to the in vitro study predicted that the motion segment will require 31,855 cycles to fail for the given loading. The FE model identified damage initiation and progression in the posterior region of the annulus. The current FE study results thus matched well with the cadaver study observations in terms of number of load cycles to failure and location of damage accumulation (Gordon et al, 1991).

Damage accumulation in the annulus with increasing number of load cycles was plotted under different simple and complex loading conditions (Figure 5). The damaged annulus volume increased almost linearly with increasing number of load cycles until the point of failure under
all loading modes. At the failure point an exponential increase in the damaged annulus volume was observed against a very small increase in the number of load cycles. The number of load cycles to failure decreased as the motion segment was subjected to bending moments in addition to the compressive load. The failure load cycle was identified by sharp increase in failure volume against a very small increase in number of load cycles.

Application of 6 Nm moments in the three principal directions without any compressive load (Load cases 3-5) did not create the failure of the disc, regardless of the number of applied load cycles. Similarly, cyclic compressive loading with a peak load of 400 N (Load case 1) did not fail the disc. The FE model predicted the failure of the disc in 50,798 load cycles under cyclic compressive loading with a peak load of 800 N (Load case 2). Introduction of 6 Nm moments in the three principal directions in concert with the cyclic compressive load (Load cases 6-8) decreased the number of load cycles to failure by 50% (flexion), 32% (lateral bending) and 18% (axial rotation) as compared to the uni-axial cyclic compressive loading (Load case 2). Application of 6 Nm moments in flexion, lateral bending and axial rotation simultaneously along with the compressive cyclic load (Load case 10), reduced the number of load cycles to failure by 71% as compared to the cyclic compressive loading (Load case 2).

FE model predicted the initiation of damage at the posterior region of the inner annulus next to the inferior endplate and progressed towards outer periphery under all loading conditions considered (Figure 6). Damage was also identified at the mid disc height in the posterior annulus which did not propagate beyond few inner annulus layers. Introduction of bending moments caused the damage to progress preferentially in the posteriolateral region of the annulus.

Even though the failure of annular fibers based on maximum strain value was also included in the analyses, no fiber damage was observed in any of the simulations.
4. DISCUSSION

A poro-elastic FE model of L4/L5 lumbar motion segment incorporated with continuum damage mechanics methodology was presented that predicted structural damage in the disc under cyclic loading. Results of the validation study were consistent with the experimental observations in terms of the region of failure, magnitude of applied loads and the number of load cycles. In vitro and in vivo human studies have reported occurrence of radial fissures in the posterior annulus (Haefeli et al., 2006; Osti et al., 1992; Sharma et al., 2009a; Sharma et al., 2009b; Vernon-Roberts et al., 2007). Cyclic testing of porcine discs also reported failure in the posterior annulus (Marshall and McGill, 2010; Callaghan and McGill, 2001). Thus damage accumulation in the posterior annulus as predicted by the current FE model matched well with clinical and experimental observations. The numbers of load cycles to failure predicted for complex loading modes were considerably smaller than those for uni-axial loading supporting the first hypothesis. Damage initiated and progressed preferentially in the posterior annulus under all loading conditions simulated in this study validating the other hypothesis.

Goel et al. (1988) subjected 11 cadaveric human lumbar spines T12-S1 to 3Nm flexion moment for up to 9600 cycles. They reported structural failure in none of the discs, which is consistent with the results from the current study. The FE model results showed that the repetitive bending without compressive load and cyclic compressive loading with low peak load magnitude did not create a failure in the disc irrespective of the number of load cycles. These findings compare well with the results presented by Callaghan and McGill, (2001) who observed just an initiation of a fissure in the posterior region in only 1 out of 5 discs subjected to low compressive load. Marshall and McGill, (2010) reported increased annulus damage in porcine discs subjected to combination of axial rotation and flexion/extension than those tested under
flexion/extension alone. This result supports the less number of load cycles to failure predicted under complex loading modes than single axis bending in the current study.

One of the major limitations of the algorithm used here is that even though the damage to the tissue occur only in the direction of tensile principal stress, the algorithm assumes damage equally occurs in all the three principal directions at each integrating point within an element. The current analyses also did not take into account the changes in the viscoelastic characteristics of the annulus due to the fluid flowing in an out of the disc with increasing number of load cycles which is another major limitation of the analyses. Instead, the damage accumulation methodology was employed as it enabled the simulation of large number of load cycles without having to run every load cycle, thus dramatically reducing the computational expense.

Damage accumulation was designed as a linear process which is a fair assumption in absence of any experimentally derived data on damage propagation in the annulus. The analyses further assumed no rest periods between the loading cycles as well as the tissue healing process. Shearing between the annulus layers has been suggested to cause the delamination of the annulus layers (Iatridis and ap Gwynn, 2004; Marshall and McGill, 2010; Schmidt et al., 2009). However, lack of stress-failure curve based on shear stress made it impossible to include the damage mechanism due to shearing.

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Conflict of Interest Statement

None of the authors has any conflict of interest to report.
REFERENCES


