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Finite element investigation of the effect of nucleus removal on vibration characteristics of the lumbar spine under a compressive follower preload

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ABSTRACT

Previous studies have reported the effect of removing the nucleus on biomechanical responses of the human spine to static loadings. However, few studies have dealt with the whole-body vibration condition. The purpose of this study was to investigate the effect of a single-level (L4–L5) nucleus removal on vibration characteristics of the whole lumbar spine in the presence of a physiologic compressive preload, and also to evaluate the preload effect on the vibration characteristics. A 3-D non-linear finite element model of the lumbar spine (L1 to sacrum) subjected to the physiologic conditions of a compressive follower preload was developed and validated. Comparative studies on forced vibration responses between the intact and denucleated models were conducted. The results from the forced-vibration (transient dynamic) analyses considering axial cyclic loading indicated that the nucleus removal increased the dynamic responses at all disc levels. For example, at the denucleated L4–L5 level, after nucleus removal the maximum response values of disc bulge and von-Mises stress in annulus increased by 63.9% and 110.5% respectively, and their vibration amplitudes increased by 97.9% and 139.7% respectively. At other levels, the predicted maximum response values and vibration amplitudes of the stresses and strains also produced 3.1–7.5% and 10.8–30.6% increases respectively due to the nucleus removal, and a relatively larger increase was observed at level L5–S1. It was also found that increasing the preload increased the stresses and strains at all levels but decreased their vibration amplitudes. Nucleus removal at a single level deteriorates the effects of vibration on whole lumbar spine. Also, increasing the preload alters vibration characteristics of the spine. These findings may be useful to provide a guideline for the patients suffering from lumbar disc degeneration to minimize the risk of further injury and discomfort.

1. Introduction

There appears to be a strong link between lumbar intervertebral disc degeneration and low back pain. It has been reported that around 75% of low back pain cases are caused by lumbar disc degeneration (Vaccaro, 2005). A volume loss of the nucleus resulting from decreased proteoglycan and water concentration induces early disc degeneration (Buckwalter, 1995). This volume loss decreases intradiscal pressure and leads to abnormal stress distributions in the annulus fibrosus (Cannella et al., 2014; von Ooij et al., 2003), which further accelerates the degradation and may even result in disc prolapse (Adams et al., 1996; Brinckmann and Grootenboer, 1991; Schmidt et al., 2007). Many investigations have studied the dependency of lumbar disc mechanical behavior on alteration of the nucleus. For example, Dunlop et al. (1984) measured the pressure across the facet joints under compression in vitro and found the pressure was increased to 8.2 MPa from 6.9 MPa after removal of the nucleus. The increased stressing of the facet joints might be the origin of pain after nucleotomy (Ivicsics et al., 2014). Meakin

and Hukins (2000) showed the change in movement direction of the annulus under compressive loading and total removal of the nucleus, with the inner margins bulging outwards in the intact disc, and inwards in the denucleated disc. The increased disc displacement indicated an awkward compressive loading to the annular ring. O'Connell et al. (2011) concluded that nucleotomy altered the internal radial and axial strains of the annulus fibrosus under compressive load in the neutral position, which might lead to its damage and microfractures.

In modern times, people are frequently exposed to whole-body vibration (WBV) in their daily life and work, such as driving a car or operating other vibration machines. Epidemiological studies have suggested that long-term WBV is a major cause of low back pain and degenerative diseases of the spine (Frymoyer et al., 1980; Kelsey and Hardy, 1975). Finite element (FE) studies have also demonstrated that vibration loads are more dangerous for the lumbar spine than static loads with equivalent magnitudes (Goel et al., 1994; Guo et al., 2005). Although the published works (Dunlop et al., 1984; Ivicsics et al., 2014; Meakin and Hukins, 2000; O'Connell et al., 2011) cited in the previous

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paragraph have offered valuable insights into the effect of nucleus removal on the biomechanical behavior of the lumbar spine, most of them mainly focused on studying the effect under static loadings. It is still poorly understood how nucleus removal affects dynamic mechanical behavior of the whole lumbar spine during WBV under a physiologic compressive preload. In addition, investigations have found that the physiologic compressive preload can alter biomechanics of the spine in static loading conditions (Anderson et al., 2016; Du et al., 2016; Rohlmann et al., 2001; Zirbel et al., 2013). For example, in vitro results of Zirbel et al. (2013) showed that the preload increased the lumbar spine segmental stiffness under axial rotation, flexion-extension and lateral bending loadings. However, very few studies have addressed the effect of the preload on vibration characteristics of the whole lumbar spine.

This study aimed to investigate how nucleus removal at a single level affects dynamic response (including disc bulge, von-Mises stress in annulus ground substance and intradiscal pressure) of the whole lumbar spine to the vertical vibration using a FE model of L1–sacrum segments subjected to a physiologic compressive preload. Nucleus removal was used to simulate the injury or severe degeneration in the nucleus (Frei et al., 2001) where intradiscal pressure was as low as zero. This study also examined effect of the preload on the dynamic response for both the intact and denucleated models.

2. Materials and methods

2.1. FE modeling

A 3-D non-linear osteoligamentous FE model of the L1–sacrum

human lumbar spine was constructed based on computed tomography (CT) protocols (Fig. 1). The bony geometry was obtained via high-resolution CT scans of a 48-year-old female volunteer (weight, 58 kg; height, 162 cm) with no lumbar diseases. The pre-processing software ANSA (BETA CAE Systems S.A., Thessaloniki, Greece) was used for meshing in this study. Each vertebra was modeled by accounting for the separation of the cancellous bone and cortical shell (including endplate) (El-Rich et al., 2009) using 4-node tetrahedral and 3-node triangular elements, respectively. There were 96,298 elements used to model the cancellous and cortical bone in the entire model. The intervertebral disc consisted of a nucleus pulposus surrounded by annulus ground substance. The disc was simulated as nearly incompressible and Mooney-Rivlin hyperelastic (Schmidt et al., 2006) by 8-node hybrid hexahedral elements. The annulus substance was reinforced in the radial direction by six fiber layers using 2-node tension-only truss elements oriented at $\pm 30^\circ$ to the horizontal (Ambati et al., 2015). There were 37,704 elements used to model the annulus and nucleus in the entire model. The contact between the facet joints was defined as a surface-to-surface contact with no friction (Barthelemy et al., 2016). The seven major spinal ligaments were represented by 2-node tension-only truss elements. The material properties used in current FE model were taken from the literature (Table 1). The denucleated model was generated by removing the nucleus from the L4–L5 disc. This level was chosen due to its higher prevalence in individuals suffering from disc degeneration (Cheung et al., 2009; Ruberte et al., 2009). The present study was approved by the Science and Ethics Committee of Northeastern University, and an informed consent was obtained from the volunteer.

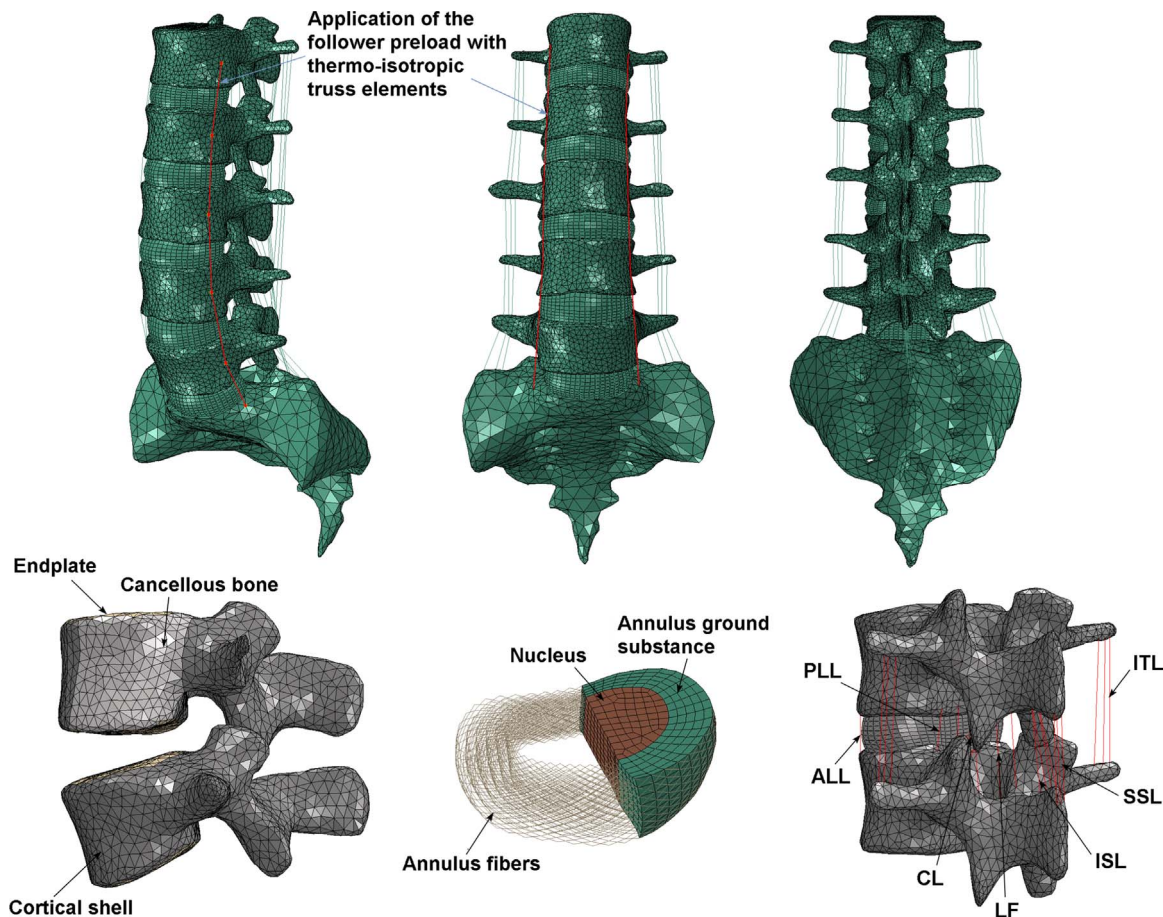


Fig. 1. FE model of the L1–sacrum spinal segments. (ALL = anterior longitudinal ligament, PLL = posterior longitudinal ligament, LF = ligamentum flavum, SSL = supraspinous ligament, ISL = interspinous ligament, ITL = intertransverse ligament, CL = capsular ligament).

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