EFFECTS OF INTERVERTEBRAL DISC DEGENERATION ON THE SURFACE STRAIN DISTRIBUTION OF HUMAN VERTEBRAE

Giulia Cavazzoni[1], **Margherita Pasini**[1], **Luca Cristofolini**[1], **Marco Palanca**[1]
1. *Dept of Industrial Engineering, Alma Mater Studiorum – University of Bologna, Bologna, IT*

Introduction

Intervertebral discs (IVDs) degeneration involves changes in biomechanics, structure of the extracellular matrix, genetics, and cellular activities and can turn into a painful pathological condition [1]. How these changes can affect the load transfer through the vertebrae, and consequently the local mechanical behaviour of the vertebrae is still unclear. In spine segments FE models, IVDs are frequently modelled with simplified mechanical properties [2], which may not be sufficient to describe the IVDs degeneration and apply realistic boundary conditions to the FE model of the vertebrae. The effects of the IVDs degeneration on the adjacent vertebrae can be evaluated experimentally by combining mechanical tests and Digital Image Correlation (DIC) to characterize the external strain field of both vertebrae and IVDs simultaneously. The aim of this study is to evaluate the strain distribution on the vertebral bodies.

Methods

Two T9-L1 spine segments were harvested from human donors (Ethical approval: Prot.n.113043) and imaged with a 3T Magnetic Resonance Imaging (MRI) and a Computed Tomography (CT) to establish the degeneration of the IVDs (IVDs degeneration level = 2) [1] and exclude critical bone pathology. All soft tissues and the anterior ligament were removed without damaging the IVDs. A high-contrast white speckle pattern was prepared on the surface of each specimen. A four-cameras 3D-DIC system (Aramis Adjustable 12M, GOM) was used to measure the strains on the surface of the specimens, at 25 frames per second, with a measurement spatial resolution of 0.07mm. The intact specimens were mechanically tested with a uniaxial testing machine (Instron 8500, 10kN load cell) to induce flexion loads, as defined by [3]. Each spine segment was loaded until the average minimum principal strain on the central vertebra reached approximately 3000$\mu\varepsilon$ (target to remain in elastic regime without damaging the bone). Ten preconditioning cycles up to half of the load corresponding to the target strain were applied. Then, each specimen was loaded monotonically to reach the target strain in 10s, 1s and 0.4s. To simulate a grade 5 degeneration [1] of the IVD, the nucleus of the T11-T12 IVD was entirely removed mechanically [4]. The specimens were tested again, following the same loading protocol. Random errors were evaluated in zero-strain condition as the standard deviation of the strain. The maximum and minimum principal strains were measured and compared before and after the IVD degeneration. Statistical analyses (Wilcoxon test, $p=0.05$) were performed in Prism.

Results

Random errors were smaller than 100$\mu\varepsilon$. The maximum and minimum principal strains in the vertebrae close to degenerated IVD (T11 and T12), were significantly different before and after IVD degeneration (maximum principal strains: 1302$\mu\varepsilon$ vs 1885$\mu\varepsilon$; minimum principal strains: -2711$\mu\varepsilon$ vs -3515$\mu\varepsilon$; $p=0.02$).

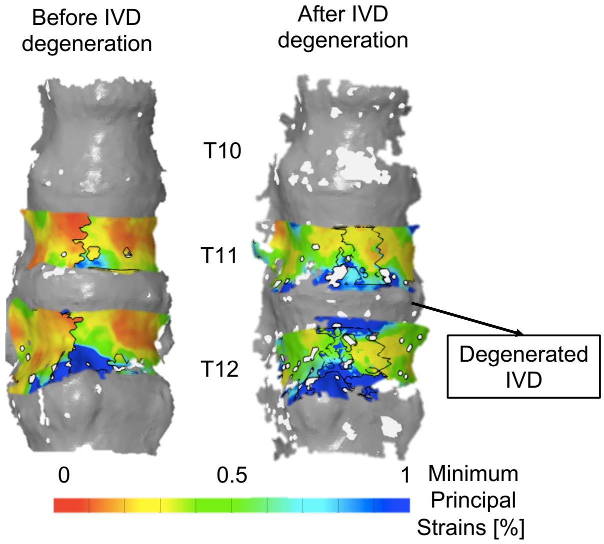

Figure 1: Minimum principal strains [%] evaluated in flexion on a T9-L1 spine segment, before (left) and after (right) T11-T12 IVD degeneration.

Discussion

Results showed that IVD degeneration leads to an increase in the strains experienced by the adjacent vertebral bodies adjacent to the IVD degeneration with respect to the healthy condition (Fig1). Thus, the IVD degeneration causes an alteration in the transmission of loads through the vertebral body which reach larger strains close to failure strains [5]. Further analyses and mechanical tests on other specimens are still on going.

References

1. Pfirmann et al. (2001), *Spine*
2. Argoubi et al. (1996), *JBiomech*
3. Palanca et al. (2021), *Bone*
4. Techens et al. (2020), *MEP*
5. Morgan and Keaveny (2001), *JBiomech*

Acknowledgements

The study was partially funded by the AOSpine Knowledge Forum Associate Research Award (*AOS-KF-TUM-22-003*).