

# EARLY PHYSIOLOGICAL LOADING AFTER ENDPLATE FRACTURE INDUCES CHRONIC DEGENERATIVE CHANGES IN THE ADJACENT DISC IN VITRO

Stefan Dudli<sup>1</sup>, Daniel Haschtmann<sup>2</sup>, Stephen J. Ferguson<sup>1</sup>

<sup>1</sup> *ETH Zurich, Institute for Biomechanics, Switzerland;* <sup>2</sup> *Schulthess Clinic, Spine Center, Switzerland*

## Introduction

Burst fractures are often treated conservatively and with early mobilization [Wood, 2003]. Physiological loading of the burst vertebrae is thought to improve anabolism and to promote a functional repair. However, burst fractures also disrupt the adjacent intervertebral disc and induce disc degeneration (DD) [Kerttula, 2000]. Contrary to bone, the disc has a limited ability for self-healing; hence, posttraumatic loading may be detrimental. In this study, we investigate the effect of early loading of burst spinal segments on the course of DD. We hypothesize that early posttraumatic loading after a burst fracture accelerates DD.

## Methods

72 rabbit spinal segments (disc/endplates + 1/3 of adjacent vertebrae) were harvested from T8/9-T5/6 and assigned to control (n = 36) or trauma groups (n = 36). Burst fractures were induced at day 0 in the trauma group using a dropped-weight device. From day 1 to 28, all specimens were cultured at 37°C and were dynamically loaded daily (~1 MPa nominal pressure, 1 Hz, 2500 cycles). At day 1, 7, 14, and 28, 9 specimens from each group were taken for analysis: histology (n = 2), total disc glycosaminoglycan (GAG) content (n = 3) normalized to DNA, and qPCR of DD marker genes (n = 4) in nucleus pulposus (NP) and annulus fibrosus (AF).

## Results

In the NP, early posttraumatic loading of burst spinal segments strongly increased transcription of catabolic (MMP 1, -3), inflammatory (IL 1, -6, iNOS) and apoptotic (TNF- $\alpha$ , FasL) genes for 7 days (Figure 1). Up-regulation persisted up to 28 days. Trends were similar in the AF, but less pronounced. Despite the moderate and delayed up-regulation of anabolic genes in the NP and AF, a 65 % GAG loss by day 28 was observed. This was in agreement with strong matrix remodelling seen on histological sections.

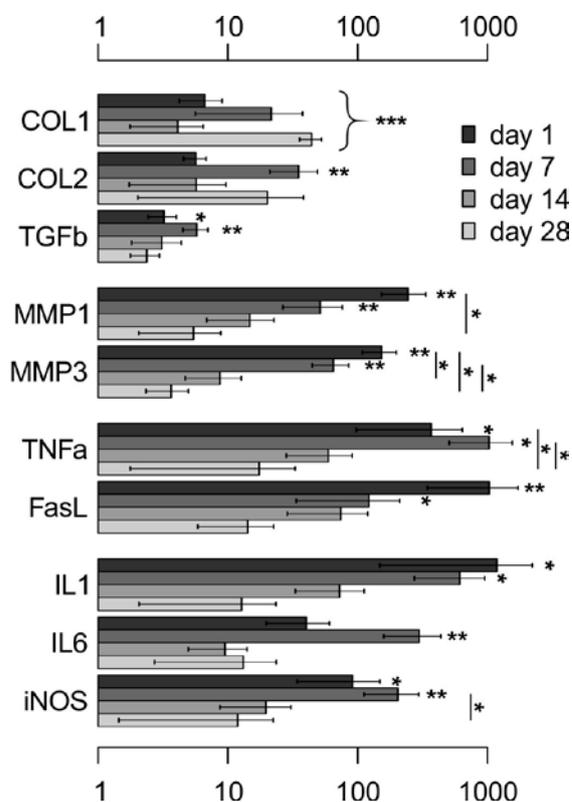


Figure 1: Relative gene transcription in the Nucleus Pulposus of post-traumatically loaded spinal segments normalized to GAPDH and non-traumatized but loaded control specimens.

## Discussion

Physiological compressive loading after burst fracture triggers chronic catabolic, inflammatory, and apoptotic changes in the adjacent disc. Similar studies with burst spinal segments, but without posttraumatic loading, reported no effects on the AF, fast normalization of the catabolic and inflammatory response in the NP, and minor GAG loss [Dudli, 2012]. Therefore, early physiological loading after a burst fracture accelerates the progress of DD.

## References

- Dudli *et al*, J Orthop Res, 30(5):809-16, 2012.
- Kerttula *et al*, J Spine, 25(9):1104-8, 2000.
- Wood *et al*, J Bone Joint Surg Am, 85(5):773-781, 2003