

An *in vivo* nutrient insufficiency induced ovine lumbar intervertebral disc degeneration model

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INTRODUCTION: Intervertebral disc degeneration is believed to play an important role in low back pain. The cells inside the disc rely on diffusion for nutrition and removal of waste products through the endplate¹. Although correlation has been found between occlusion of endplate vascular openings and disc degeneration, causality has never been demonstrated in intact discs². A model for nutrient insufficiency induced disc degeneration is being developed. In this study, we determine if the chosen method of blocking the major nutritional route results in inhibited perfusion and solute transport to the disc.

METHODS: Four sheep will be anaesthetized and the anterior lumbar spine exposed. A ~1cm wide thin slot will be sawn into the vertebrae parallel and adjacent to the endplates overlying the nuclear region of the L2-3 and L4-5 discs. After Ti-foils are inserted into the slots, the inhalation gas mixture will be changed to 70% N₂O and 30% O₂. At 5 min intervals, intranuclear concentrations of O₂ and N₂O will be measured amperometrically. Post-mortem, the vertebral vasculature will be infused with Procion red and thick sections examined to quantify the density of patent endplate capillary buds.

RESULTS: One pilot sheep has been completed. The blocks in the L2-3 disc were made without problems, in L4-L5 disc this was more difficult, and locations of the block were only partially overlying the nucleus. The N₂O diffusion measurements showed a clear inhibition of transport with the diffusion block (Fig. 1). However, O₂ concentrations only significantly differed in the cranial blocked discs (Fig. 2). Although perfusion inhibition has not yet been quantified, sections demonstrated clear decrease in the dye filled endplate capillary buds overlying regions of the blocked endplate.

DISCUSSION & CONCLUSIONS: In this study N₂O is used as a tracer for nutrient diffusion into the disc. It has been shown that it is possible to partially block the diffusion of N₂O into the disc. The defect was less efficient in blocking O₂. This could be because of differences in defects or a different diffusion

rate of O₂. This model will be used to test causality between a diffusion block and disc degeneration in a longer term study.

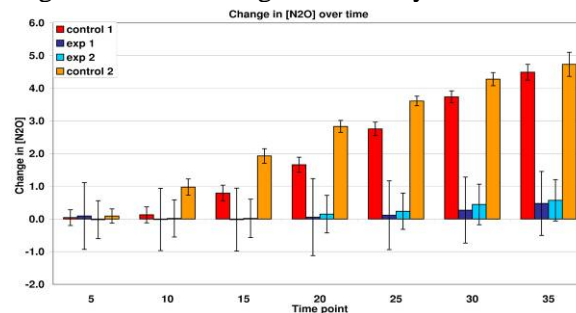


Figure 1. Increase in N₂O concentration over time relative to time 0.

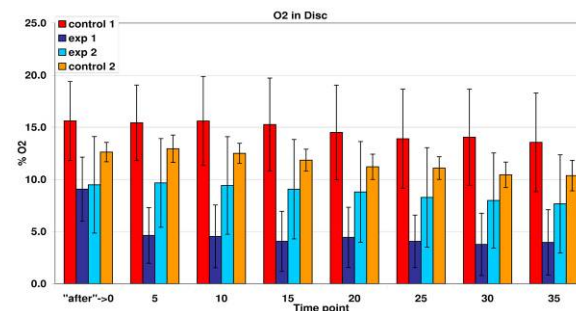


Figure 2. O₂ concentration in the discs.

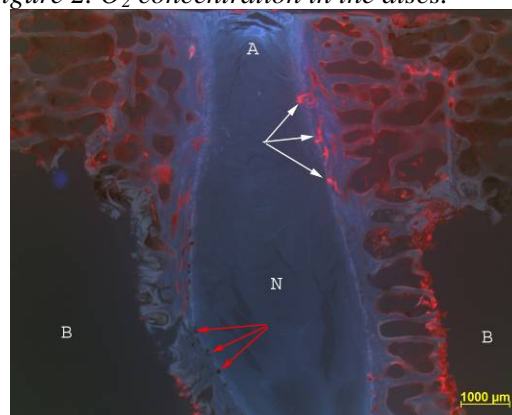


Figure 3. Procion red filled endplate capillary buds (white arrows) and non-filled bud (red arrows). (A=Annulus Fibrosus, N=Nucleus Pulposus, B=Block.)

REFERENCES: ¹ A. Maroudas, R.A. Stockwell, A. Nachemson et al. (1975) *J Anat* **120**:113-130. ² L.M. Benneker, P.F. Heini, M. Alini et al. (2005) *Spine* **30(2)**:167-173

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