

Inflammatory Effects of Severe Burn Injury in Rat Intervertebral Disc

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Background

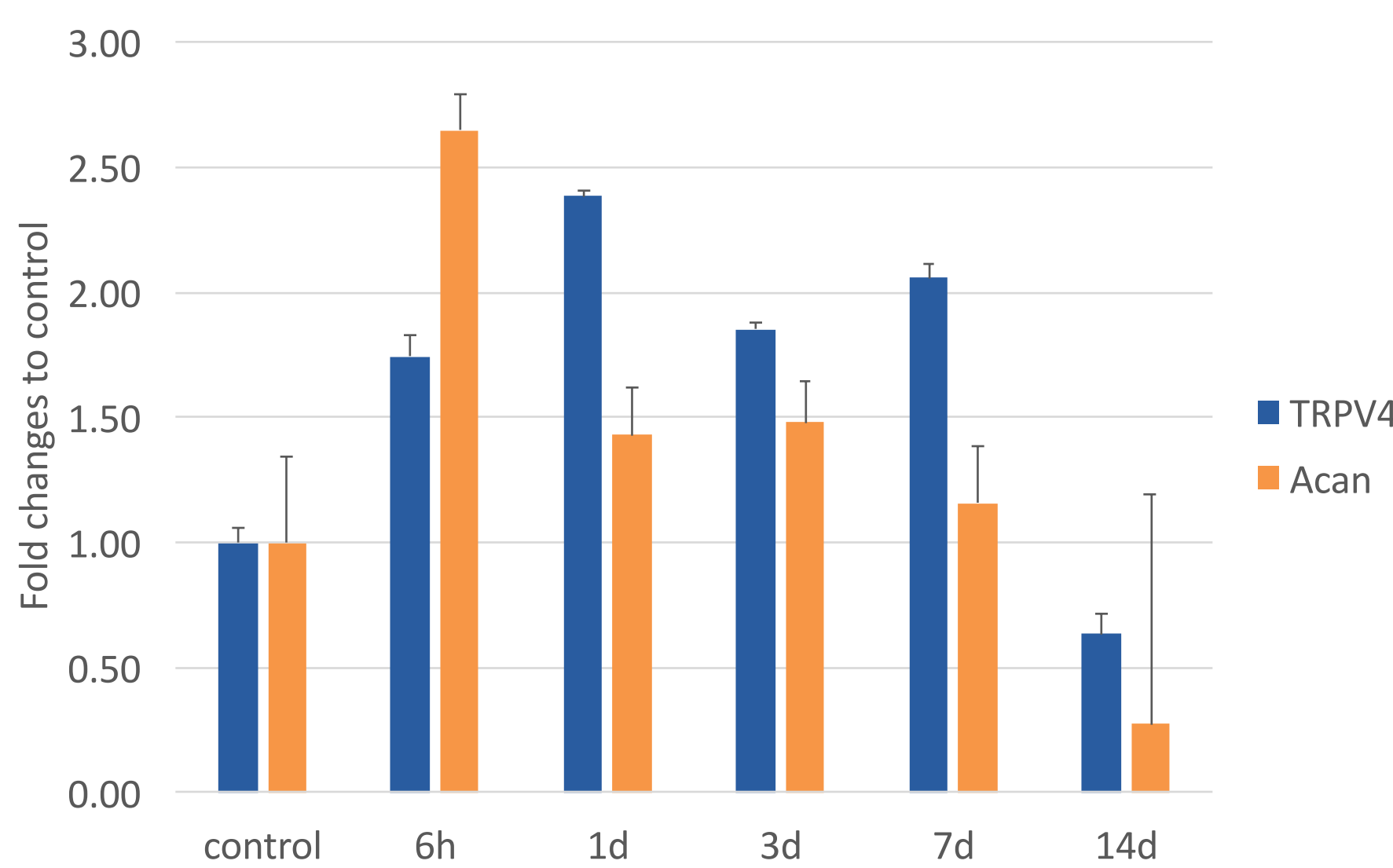
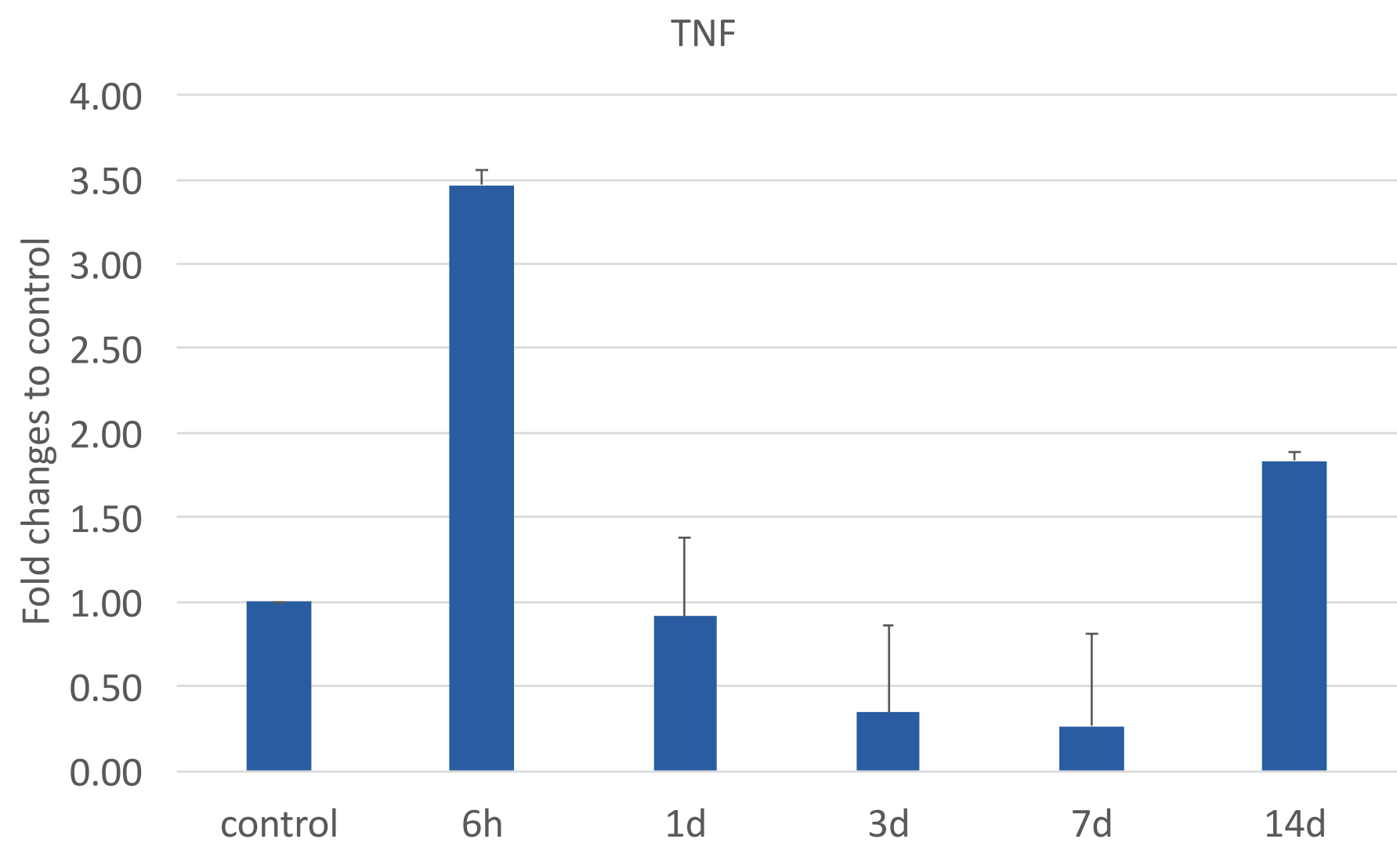
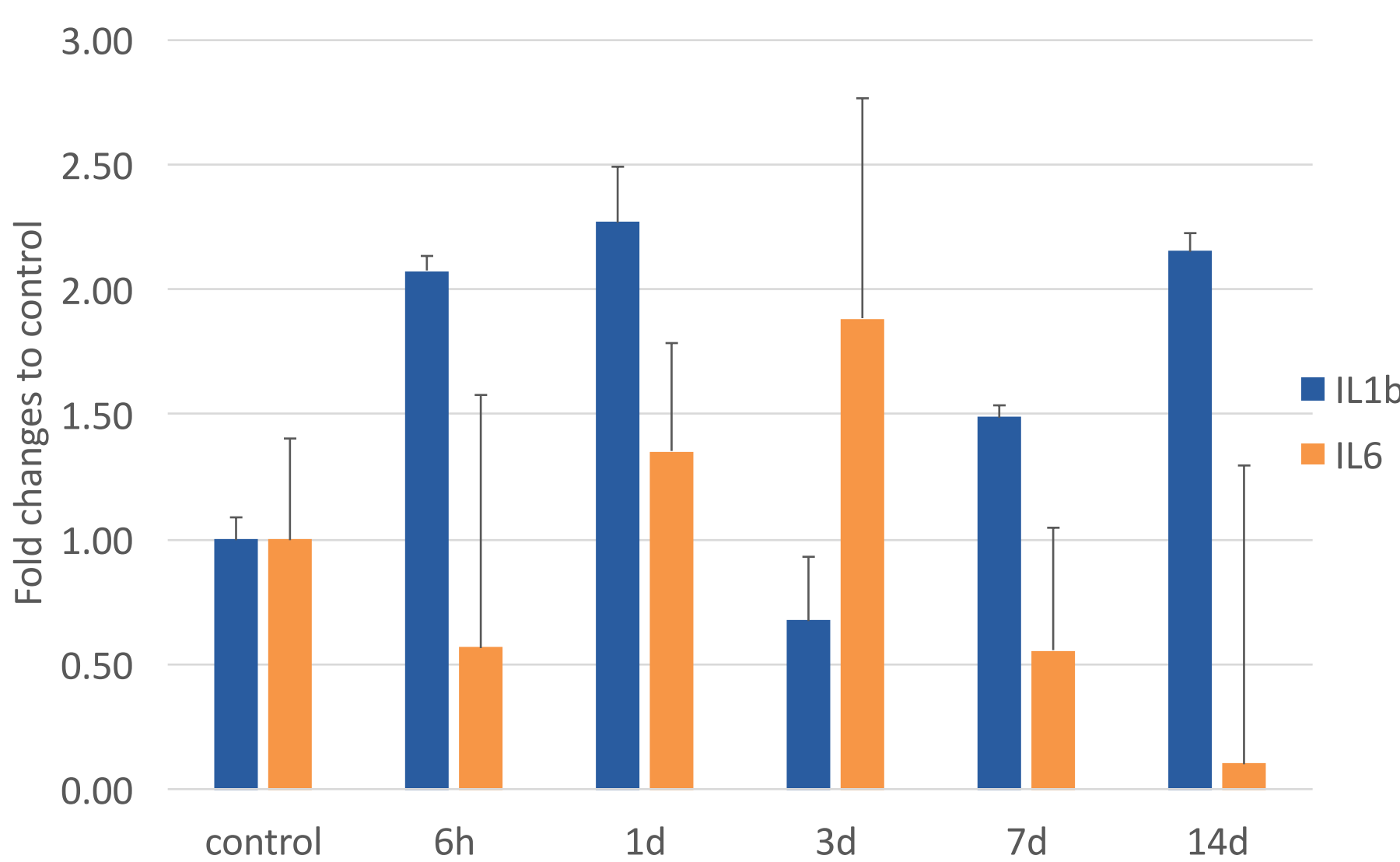
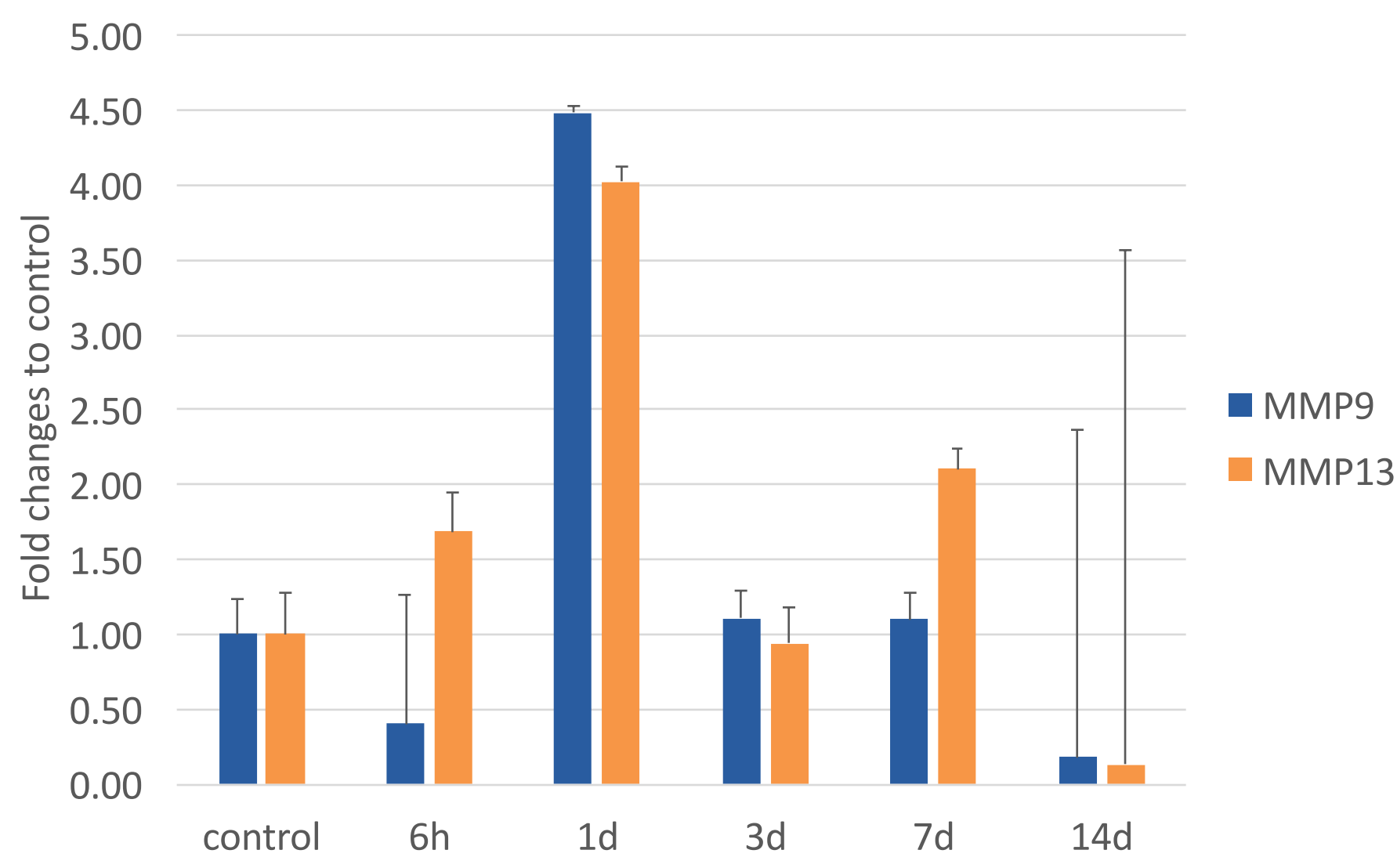
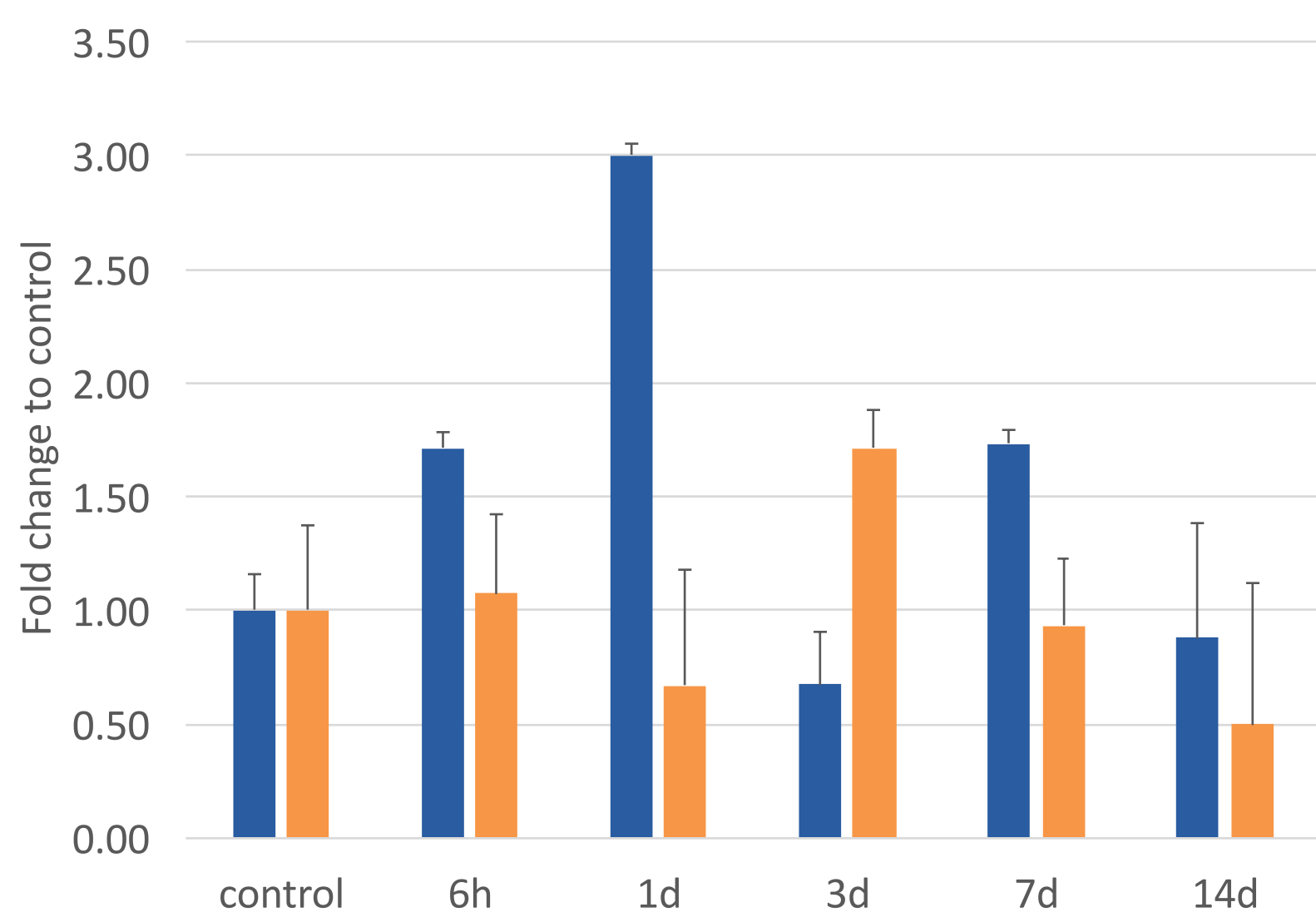
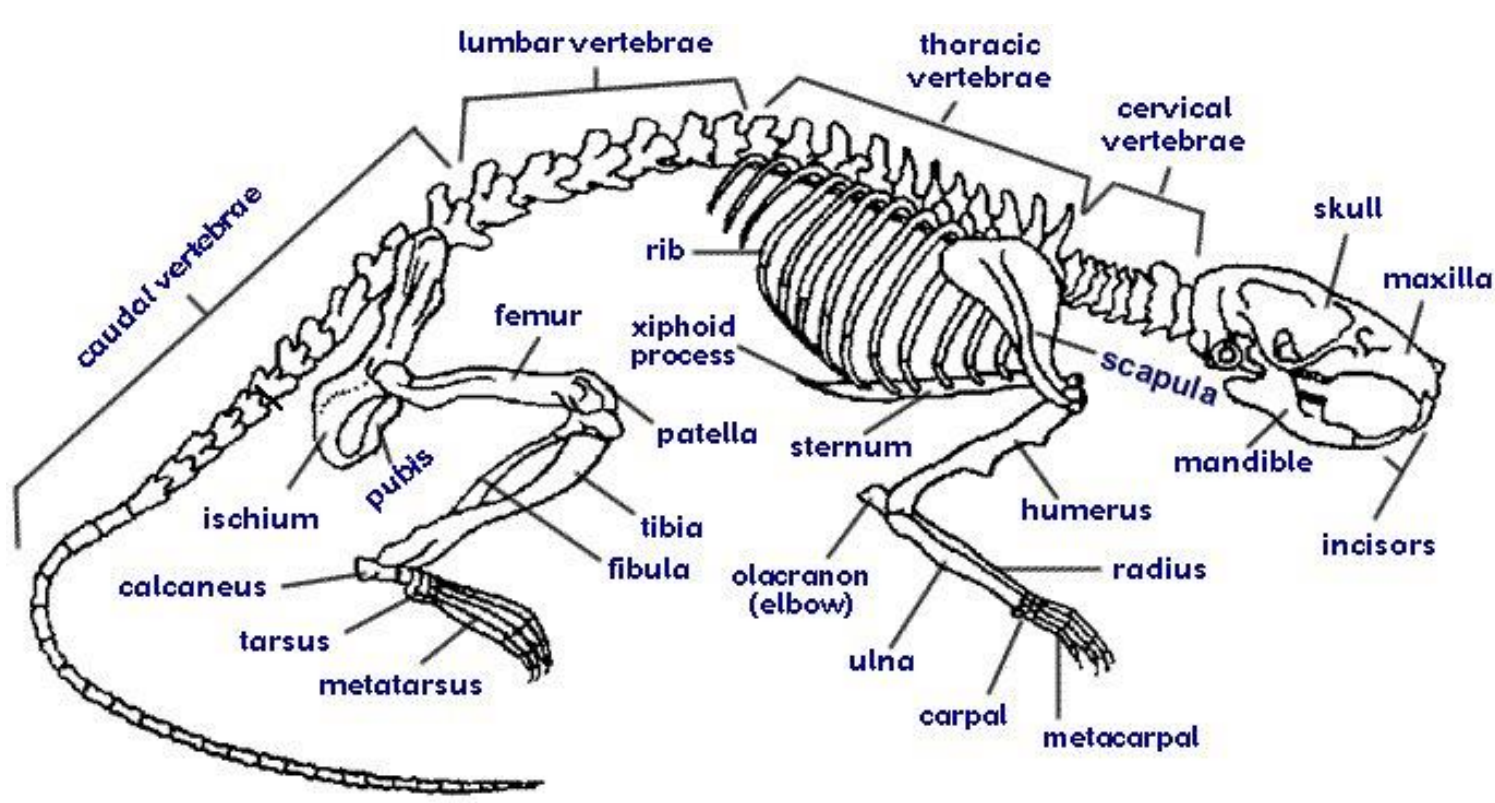
- Severe burn injury shown to cause systemic inflammatory response leading to skeletal muscle catabolism and loss of bone mass density
- Inflammatory state could lead to increased intervertebral disc degeneration, thus predisposing to disc-related spinal pathology

Purpose/Aims

- To investigate the gene expression changes after severe burn injury in rat intervertebral disc tissue and explore the possible role of inflammatory cytokines in tissue remodeling imbalance

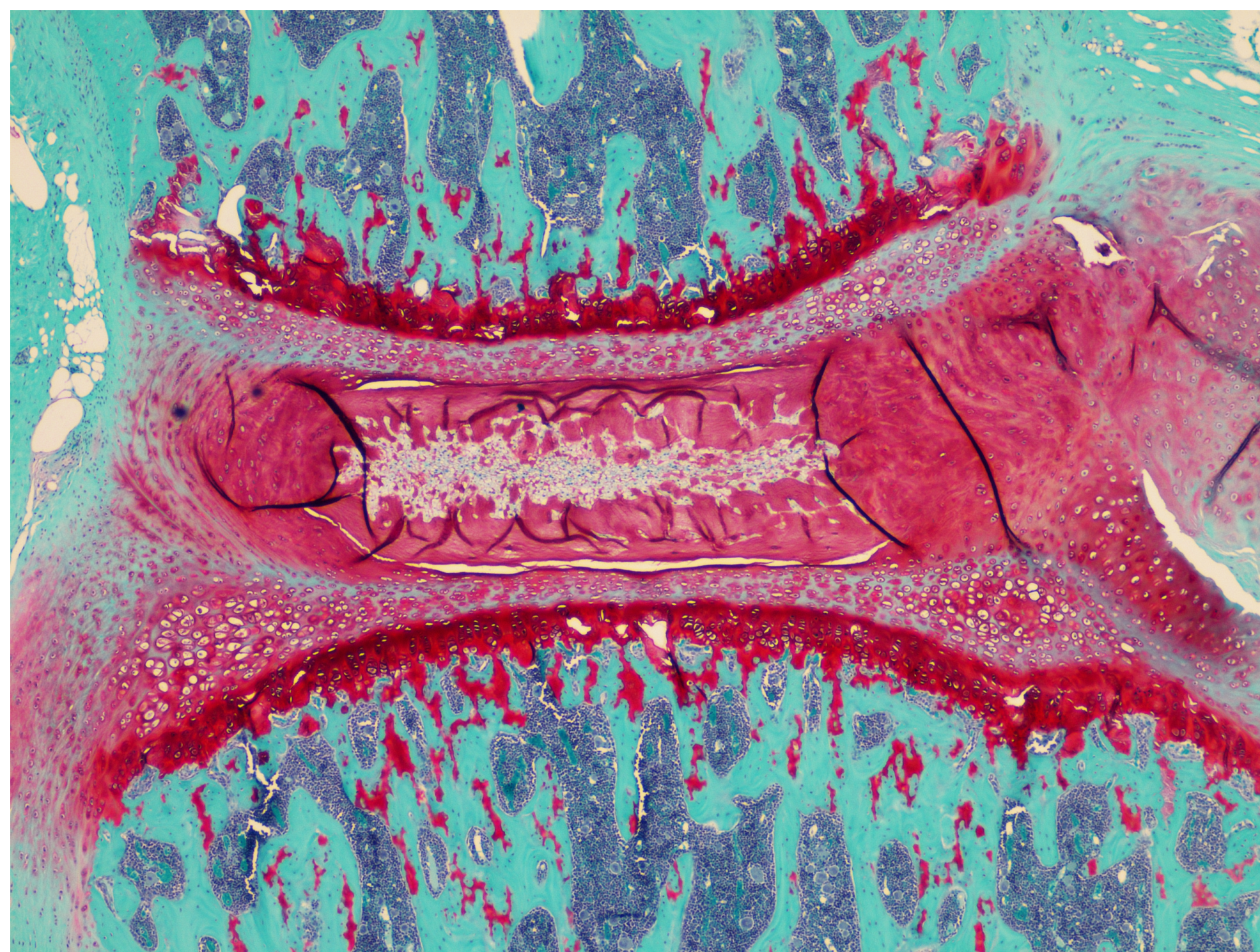
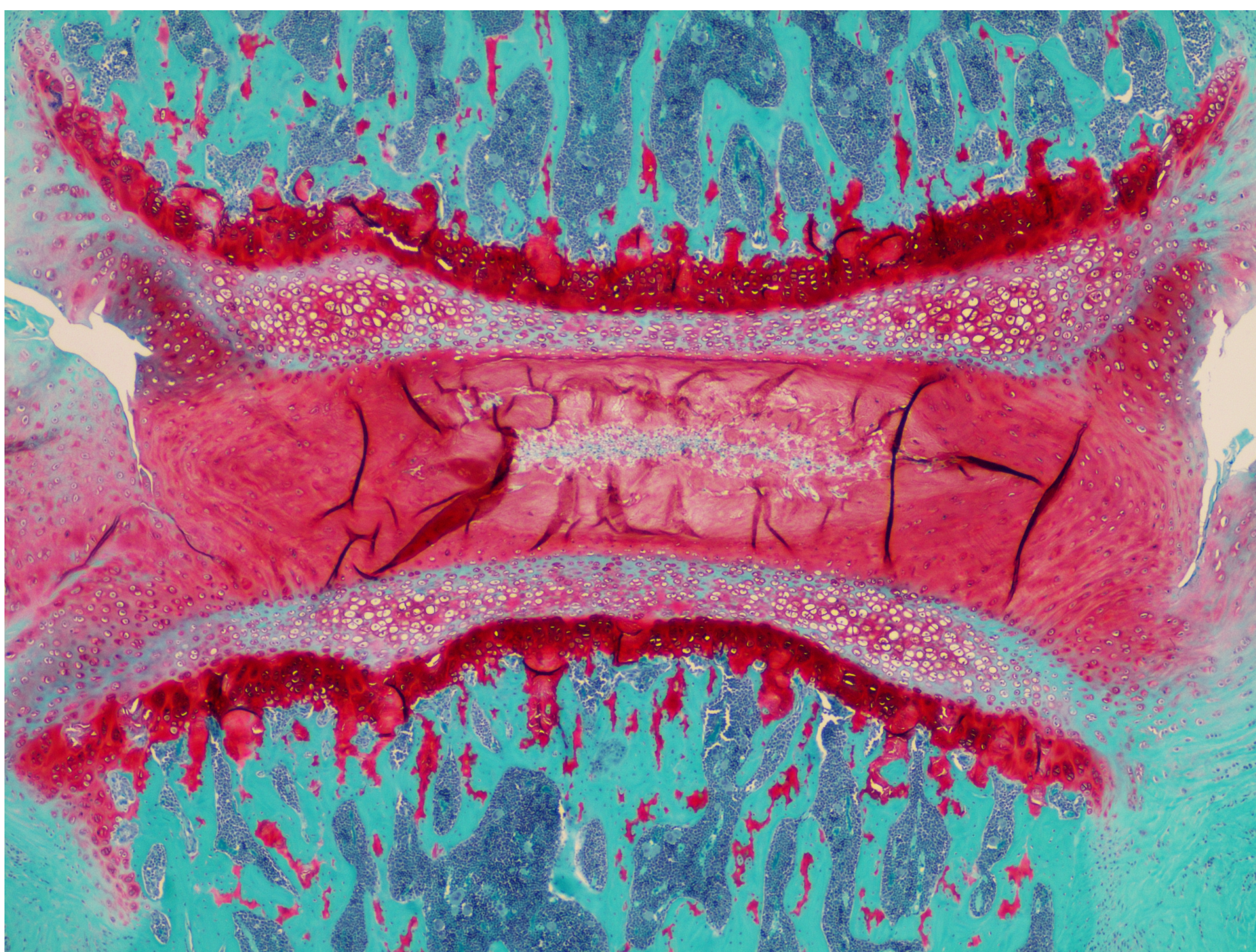
Methods

- Rats were given severe burn injury according to an established protocol: Rats were anesthetized and dipped into hot water bath to produce full thickness burn over thoracolumbar region
- Rats then sacrificed at multiple time points: 6h, 1d, 3d, 7d, and 14d post-burn. Unburned rats used for control group
- Rat intervertebral discs were dissected and removed, then used for measurement of gene expression using qPCR



Results

- Differences in gene expression of structural matrix proteins, Collagen 1 and 2, as well as matrix remodeling enzymes, MMP9 and MMP13, between time points were not statistically significant
- TNF-alpha showed increased expression at 6h that approached significance ($p=.055$), followed by statistically significant ($p<.05$) decreased expression at 1d
- This pattern of gene expression is consistent with the established process of acute inflammation and agrees with previous results seen in skeletal muscle



Conclusions

- These results indicate that in the post-burn state pro-inflammatory cytokines, such as TNF-alpha, likely play a role in the onset of degeneration of the intervertebral disc, which leads to very common and potentially debilitating spinal pathology
- These findings represent the first known characterization of inflammatory cytokines in intervertebral disc tissue after severe burn injury, and could be a model for similar response to systemic inflammation associated with other diseases, such as autoimmune diseases