

Modelling kidney fibrosis in human kidney organoids

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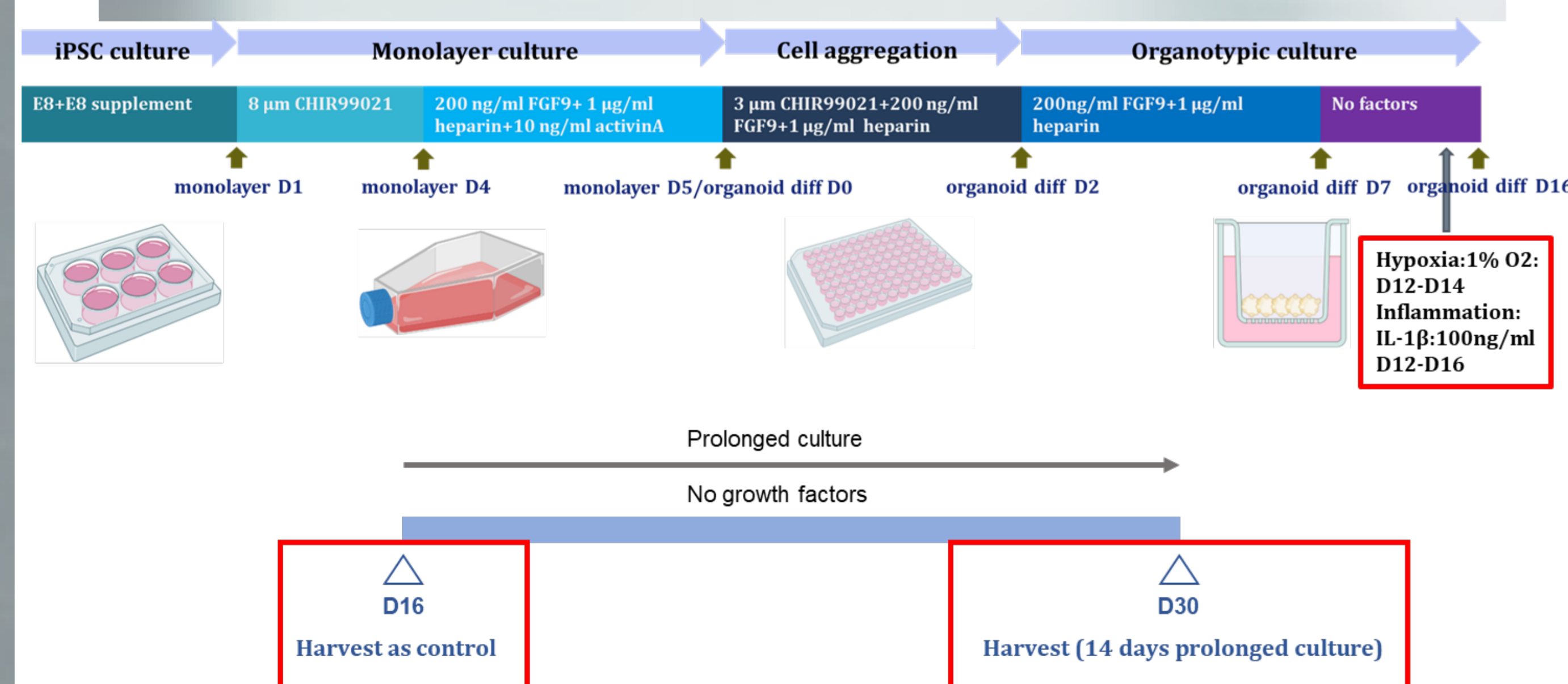
INTRODUCTION

Kidney transplantation is the treatment of choice for end-stage kidney disease. However, ischemia, inflammation during transplantation, and aging after transplantation drive transplant kidney fibrosis. The lack of experimental models for human kidney fibrosis limits the development of anti-fibrosis medicines. Kidney organoids may represent a tool for studying fibrosis.

AIM

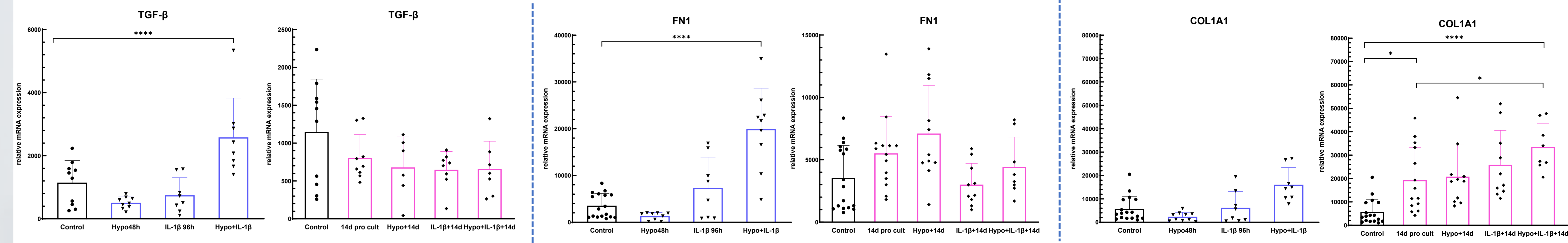
The aim of this study is to set up a kidney organoid fibrosis model and explore the degree of fibrosis and the changes in organoid phenotype.

METHOD

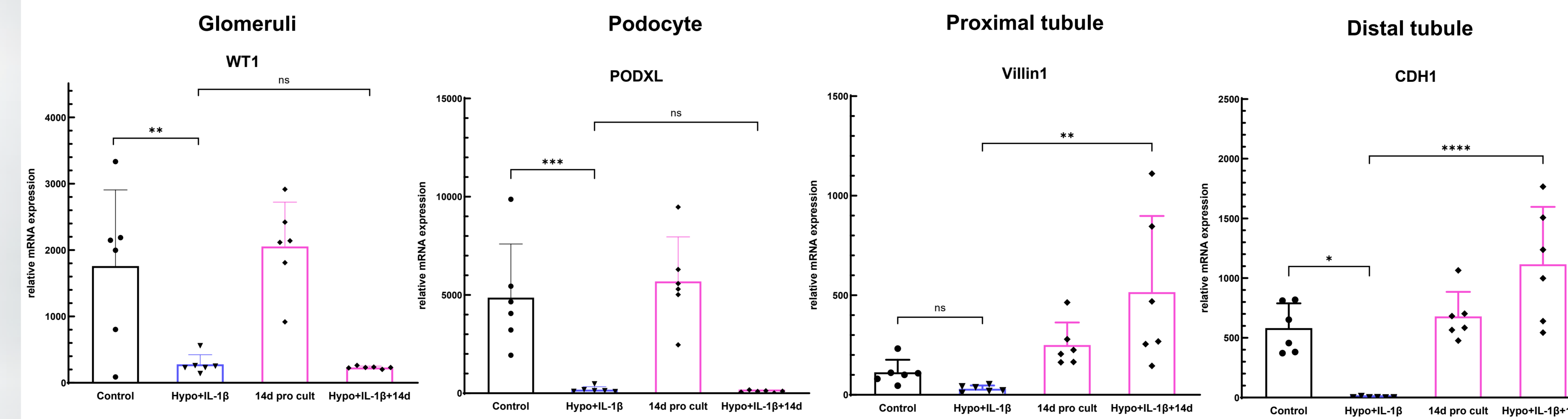


RESULTS

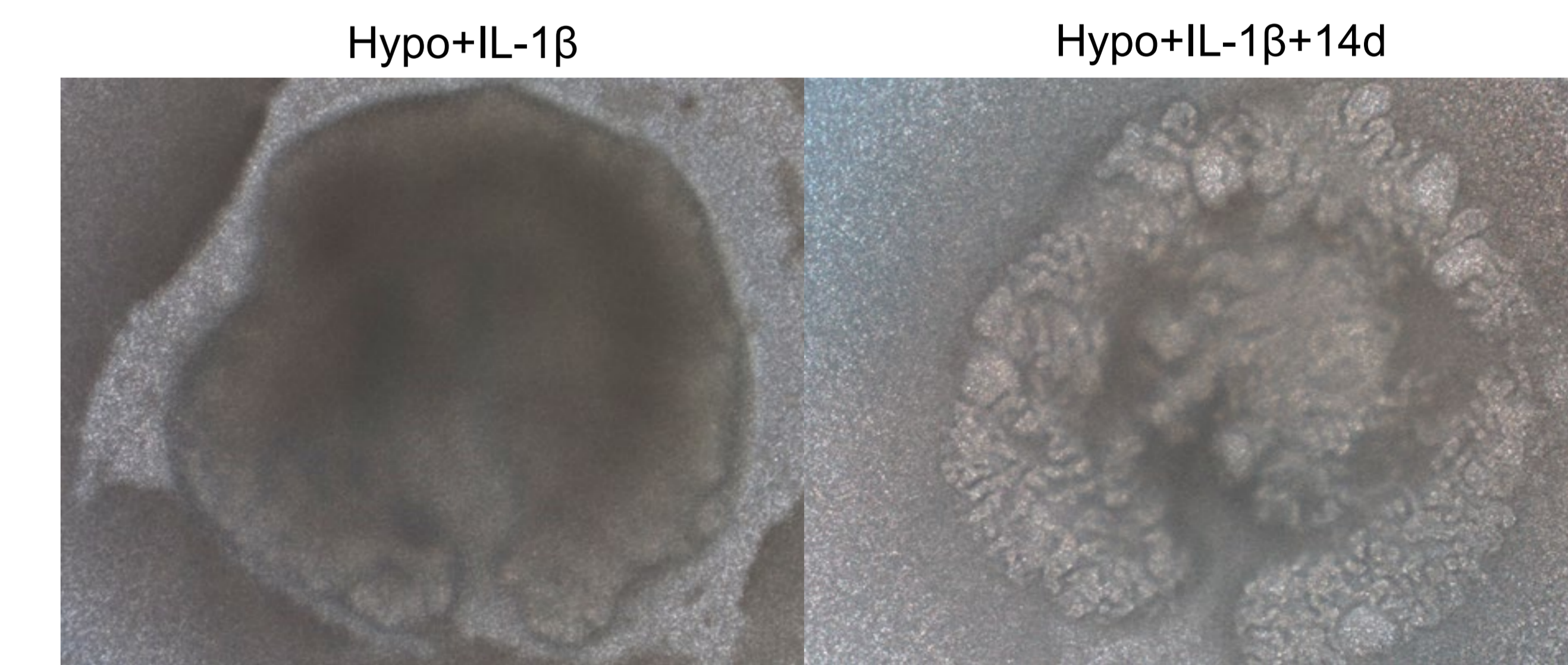
Fibrosis markers



Kidney structure markers



Organoids under microscope



CONCLUSIONS

- Early fibrosis markers were increased in group hypoxia+IL-1β, suggesting that it induces early fibrosis
- Early fibrosis transitioned to late fibrosis after combined hypoxia and IL-1β and 14 days prolonged culture, with a higher degree of fibrosis than only 14 days prolonged culture
- Fibrosis mainly affects the development of glomeruli and distal tubules
- Kidney organoid may have regenerative property

Fibrosis induction conditions:

- Hypoxia: 1% O₂ 48h
- Inflammation: 100ng/ml IL-1β 96h
- Aging: 14 days prolonged culture
- Combine hypoxia and inflammation: 1% O₂ 48h+100ng/ml IL-1β 96h
- Combine hypoxia with aging: hypoxia+14 days prolonged culture
- Combine inflammation with aging: IL-1β+14 days prolonged culture
- Combine hypoxia, inflammation with aging: hypoxia+IL-1β+14 days prolonged culture