

# 学位論文の要旨

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## 主論文の題名

The Protective Role of KANK1 in Podocyte Injury

## 主論文の要旨

Approximately 30% of steroid-resistant nephrotic syndromes are attributed to monogenic disorders that involve 27 genes. Mutations in *KANK* family members have also been linked to nephrotic syndrome; however, the precise mechanism remains elusive. To investigate this, podocyte-specific *Kank1* knockout mice were generated to examine phenotypic changes. In the initial assessment under normal conditions, *Kank1* knockout mice showed no significant differences in the urinary albumin-creatinine ratio, blood urea nitrogen, serum creatinine levels, or histological features compared to controls. However, following kidney injury with adriamycin, podocyte-specific *Kank1* knockout mice exhibited a significantly higher albumin-creatinine ratio and a significantly greater sclerotic index than control mice. Electron microscopy revealed more extensive foot process effacement in the knockout mice than in control mice. In addition, *KANK1*-deficient human podocytes showed increased detachment and apoptosis following adriamycin exposure. These findings suggest that *KANK1* may play a protective role in mitigating podocyte damage under pathological conditions.