

# Nephrin trafficking in a mouse model of experimental autoimmune nephrotic syndrome



## Introduction

Nephrin is essential for slit diaphragm integrity and glomerular filtration. **However, its trafficking in our Crb2-induced autoimmune nephrotic syndrome model remains unknown.**

## Methods

Mouse kidney / Human biopsy (anti-CRB2+)



↓  
Nephrin localization  
Endocytosis / Autophagy markers



Human podocyte

↓  
Anti-Crb2 Ab

↓  
Surface nephrin ↓ ?



## Results

### Autoimmune injury drives nephrin internalization

Nephrin shifts from membrane  
→ Rab5/Rab7 (endosomes)  
→ LC3 (autophagy)  
→ LAMP1 (lysosomal degradation)



**Reduction of Nephrin expression and decreased CRB2-Nephrin colocalization reproduced in vitro and in human MCD/FSGS.**



**Conclusions:** Nephrin is internalized and degraded via the amphisome-lysosome pathway. Membrane localization decreases in the Crb2-induced model. This suggests a mechanism of autoantibody-mediated podocyte injury.

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