

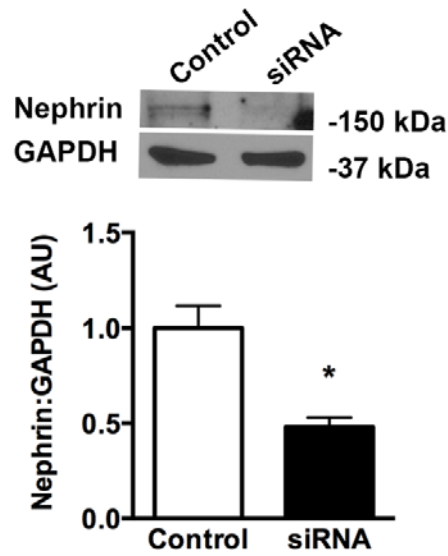
SUPPLEMENTARY DATA

Supplementary Table 1. Primer sequences used in the study.

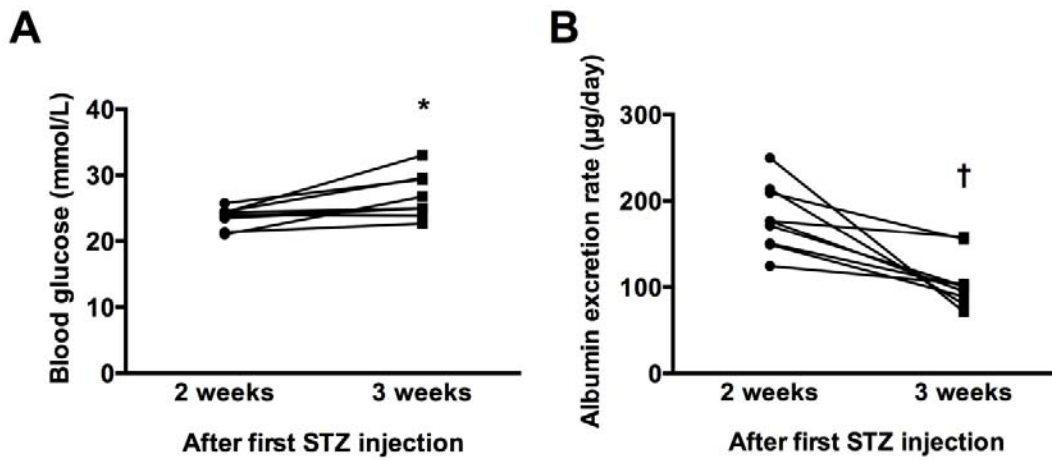
Sequences	Description
CACCAGCCCTAAGTGATCCG	Forward mouse insulin
GGCTGGGTTGAGGATAGCAA	Reverse mouse insulin
CAACATTGGTTATGGAAGCAACA	Forward human RPL32
TGACGTTGTGGACCAGGA ACT	Reverse human RPL32
GCTCTCAAGGTTGTTCTGGCTGA	Forward mouse RPL13a
AGATCTGCTTCTTCTTCCGATA	Reverse mouse RPL13a
TGGGCACTTGtttGATGAGGTAG	Forward human nephrin 1176 mutation
GGGAAGGCCATATCCTCATC	Reverse human nephrin 1176 mutation
GGGACCCCTCttcGATGAAGTGCAG	Forward human nephrin 1193 mutation
CAGGCTCCAGACGGGGGG	Reverse human nephrin 1193 mutation
GGGCTCCCAGCAGAACTCT	Human nephrin sequencing primer, forward primer 1
GCCTTCACCAAGGAGACCTTC	Human nephrin sequencing primer, forward primer 2
CCCAGGTACACGGAGCACAC	Human nephrin sequencing primer, forward primer 3
CCCAGGTACACGGAGCACAC	Human nephrin sequencing primer, forward primer 4
GGACAGGAGAGCGGGACACT	Human nephrin sequencing primer, forward primer 5

SUPPLEMENTARY DATA

Supplementary Figure 1. Effect of siRNA on nephrin knockdown in MIN6 cells. (AU) = arbitrary units. * $p < 0.05$ vs. control.

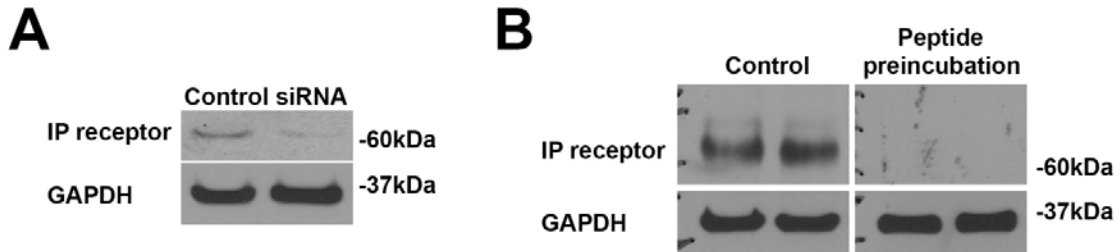


Supplementary Figure 2. Change in blood glucose (A) and urine albumin excretion (B) after two and three weeks in STZ-C57BL/6 mice treated with selexipag. * $p < 0.05$, † $p < 0.01$.

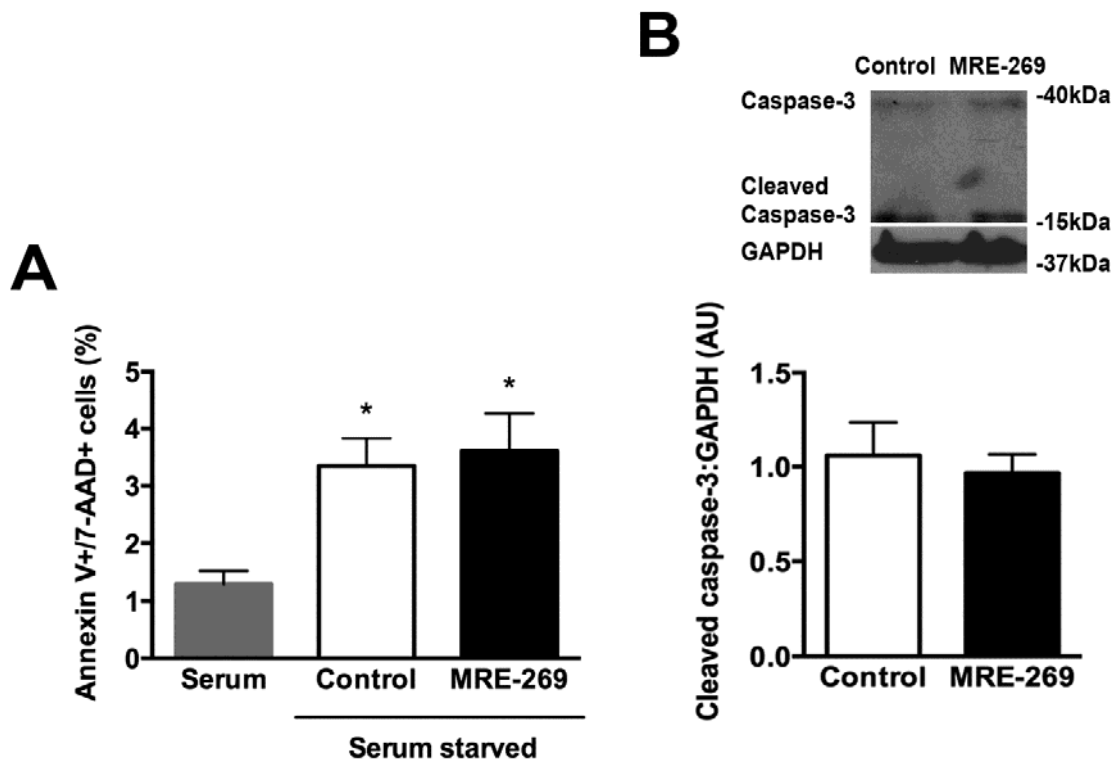


SUPPLEMENTARY DATA

Supplementary Figure 3. Controls for the IP receptor antibody in mouse podocytes. (A) Knockdown of IP receptor with siRNA. (B) Preincubation of the antibody with the immunizing peptide (sequence MADSCRNLTYVRGSVG).

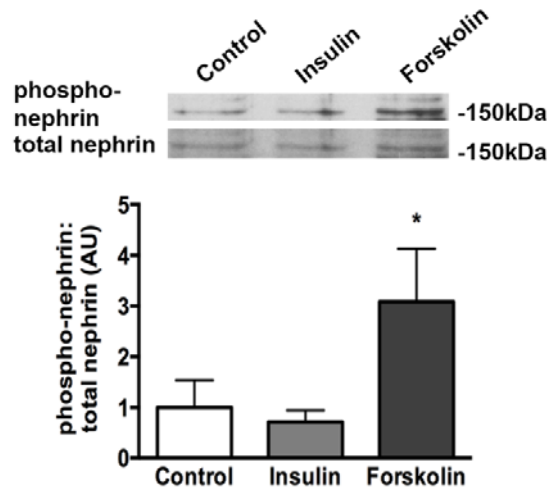


Supplementary Figure 4. Cell death in human podocytes serum starved for 48 hours under control conditions or in the presence of MRE-269 (10 μ M) for 48 h. (A) Annexin V/7-Aminoactinomycin D (7-AAD) labeling. (B) Cleaved caspase 3 levels. (AU) = arbitrary units. * p <0.05 vs. media containing 10% serum.

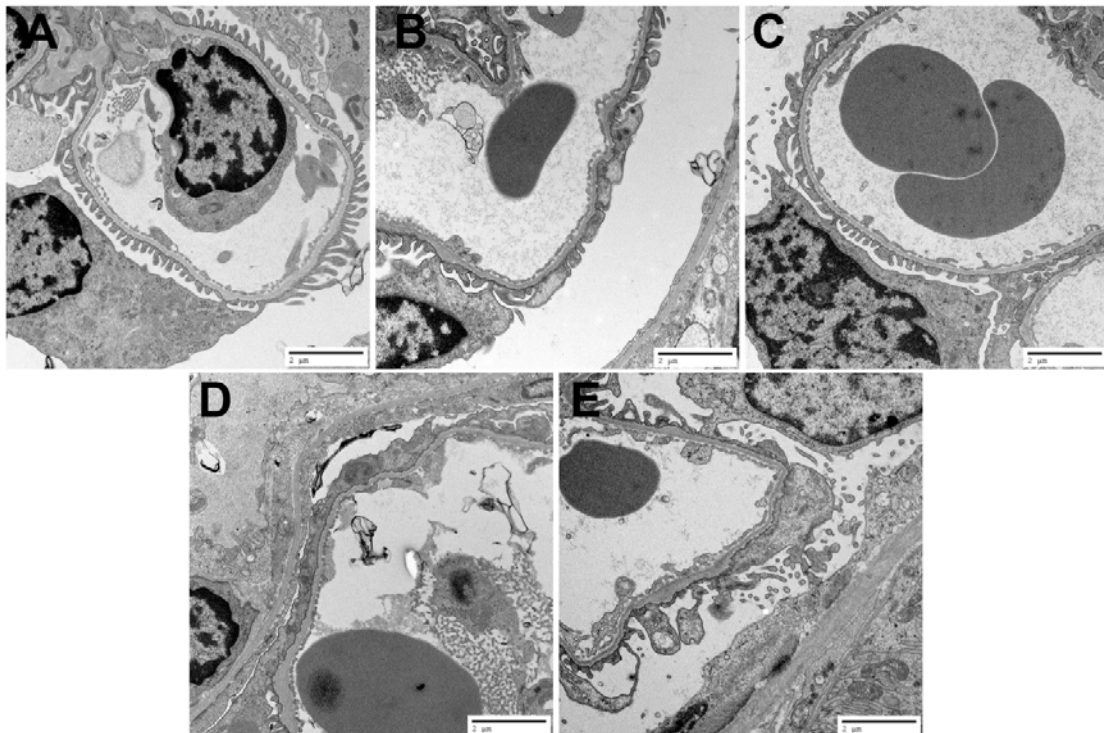


SUPPLEMENTARY DATA

Supplementary Figure 5. Effect of insulin (100nM) or forskolin (10μM) on nephrin phosphorylation in human podocytes. (AU) = arbitrary units. *p<0.05 vs. either control or insulin.



Supplementary Figure 6. Effect of selexipag on podocyte ultrastructure. Representative transmission electron micrographs from (A) C57BL/6, (B) STZ-C57BL/6, (C) eNOS^{-/-}, (D) STZ-eNOS^{-/-} and (E) STZ-eNOS^{-/-} + selexipag.



SUPPLEMENTARY DATA

Supplementary Figure 7. Effect of selexipag on glomerular CD2 associated protein (CD2AP) and Wilms tumor 1 (WT1) immunostaining. (A-E) Immunostaining for CD2AP in (A) C57BL/6, (B) STZ-C57BL/6, (C) eNOS^{-/-}, (D) STZ-eNOS^{-/-} and (E) STZ-eNOS^{-/-} + selexipag. (F) Quantitation of glomerular CD2AP. (G-K) Immunostaining for WT1 in (G) C57BL/6, (H) STZ-C57BL/6, (I) eNOS^{-/-}, (J) STZ-eNOS^{-/-} and (K) STZ-eNOS^{-/-} + selexipag. (L) Quantitation of glomerular WT1 positive nuclei. Original magnification x400.

