

Supplementary Material

Association between FNDC5 Deficiency and Compromised Mitochondrial Integrity and Biogenesis in Kidney Disease

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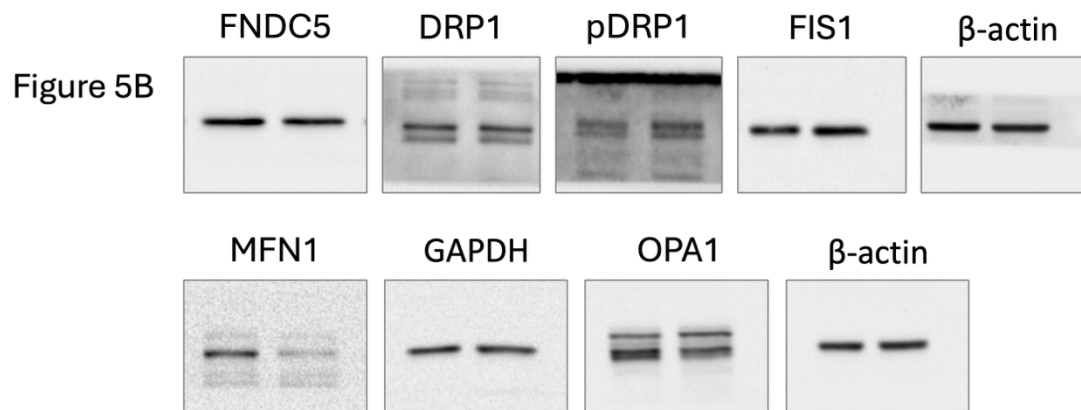
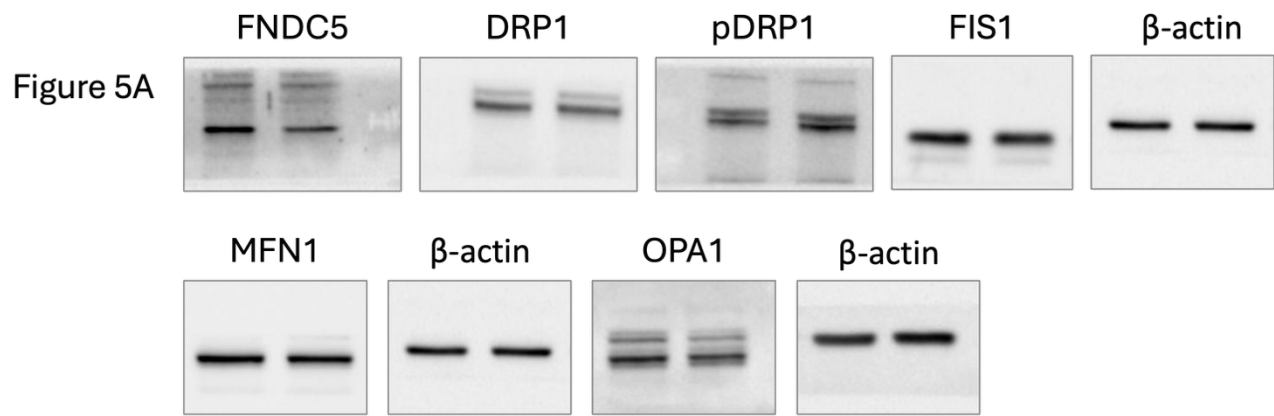
Supplementary Fig. S1. The full, uncropped, and unedited versions of the Western blots.

Supplementary Fig. S2. High glucose reduces cell viability in mouse podocytes.

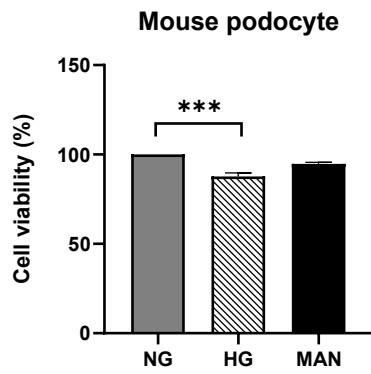
Supplementary Fig. S3. Overexpression of FNDC5 modulates mitochondrial dynamics-related proteins under HG conditions.

Supplementary Fig. S4. Recombinant irisin attenuates high-glucose-induced oxidative stress in renal cells.

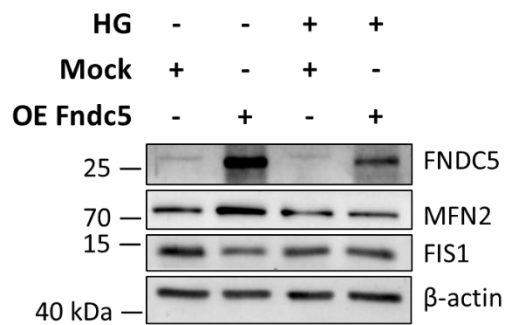
Supplementary Fig. S5. Metabolic and renal phenotypic characterization of the NIDDM mouse model.



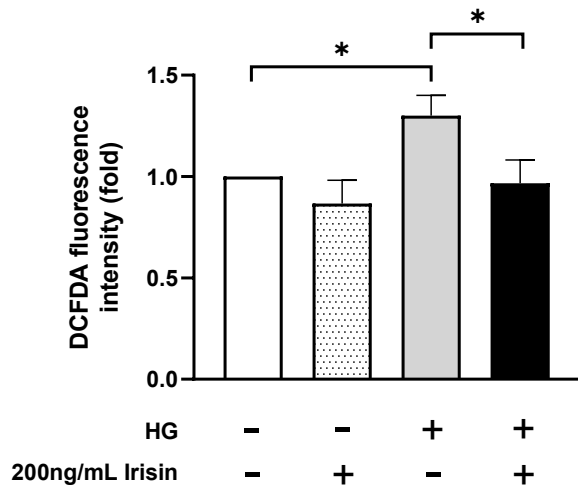
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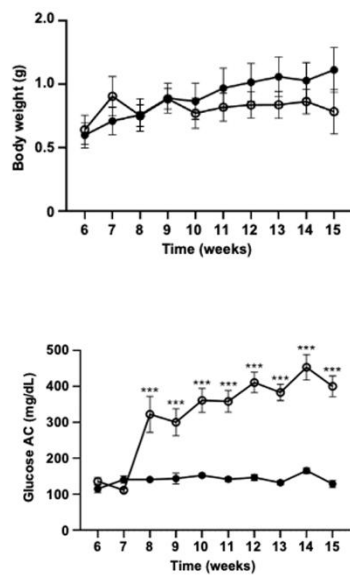
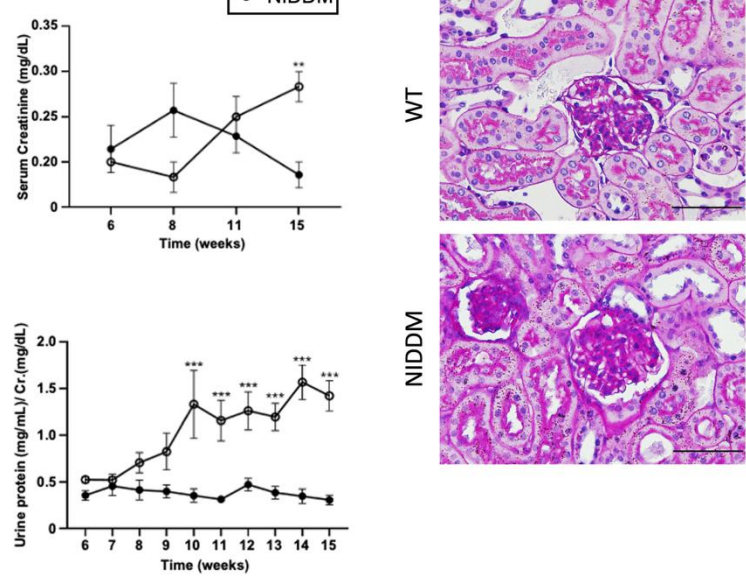
Supplementary Fig. S2. High glucose reduces cell viability in mouse podocytes. Cell viability of mouse podocytes was measured following treatment with NG (Normal Glucose, [5.5 mM]), HG (High Glucose, [33 mM]), or MAN (Mannitol, as an osmotic control). The results are expressed as a percentage relative to the NG group. Data are presented as mean $\pm$ SD/SEM. *** $P < 0.001$ indicates a significant difference between the NG and HG groups.



Supplementary Fig. S3. Overexpression of FND5 modulates mitochondrial dynamics-related proteins under HG conditions. Representative Western blot bands showing the protein expression levels of FND5, MFN2, and FIS1 in HK2 cells. Cells were treated with or without high glucose (HG) and transfected with either a mock vector or an FND5 overexpression plasmid. β -actin was used as an internal loading control to ensure equal protein loading across all lanes.



Supplementary Fig. S4. Recombinant irisin attenuates high-glucose-induced oxidative stress in renal cells. Intracellular ROS levels were measured using the DCFDA fluorescence assay. Human PTCs HK2 were treated with NG or HG in the presence or absence of recombinant Irisin (200 ng/mL) for 48 hours. The results are expressed as fold change in fluorescence intensity relative to the NG control group. Data are presented as mean $\pm$ SD (n =3). *P < 0.05 indicates statistical significance between the indicated groups.

A**B**

Supplementary Fig. S5. Metabolic and renal phenotypic characterization of the NIDDM mouse model. (A) Time course of body weight, fasting blood glucose levels, serum creatinine levels, and urinary protein-to-creatinine ratio in WT control and NIDDM mice. Data are presented as mean $\pm$ SEM ($n = 6$ for each group). (B) Glomerular basement membrane thickening and mesangial expansion were evaluated by periodic acid-Schiff staining in WT control and NIDDM mice. Scale bars = 100 μ m.