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PTPRD-Mediated Asprosin Signaling Regulates the AMPK-Sirt1-mTOR Pathway in Diabetic Kidney Disease

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Objectives : Asprosin is an adipokine associated with metabolic dysregulation. However, its role in renal energy metabolism and receptor-mediated signaling in diabetic kidney disease (DKD) remains unclear. This study investigated the role of asprosin in regulating intracellular metabolic signaling and renal injury in DKD.
Methods : Type 2 diabetic db/db mice were treated with an anti-asprosin neutralizing antibody or the AMPK activator metformin. Renal injury, metabolic parameters, and signaling pathways were evaluated. Human glomerular endothelial cells (HGECs) and renal mesangial cells (HRMCs) were cultured under high glucose and palmitic acid conditions. Cells were treated with anti-asprosin antibody, metformin, or the AMPK inhibitor Compound C. siRNA targeting protein tyrosine phosphatase receptor δ (PTPRD) was used to investigate receptor-mediated signaling.
Results : Asprosin expression was significantly increased in the serum and kidneys of db/db mice and in renal cells exposed to high glucose and palmitic acid. Neutralization of asprosin improved metabolic parameters and attenuated renal injury in diabetic mice. Anti-asprosin treatment reduced renal lipid accumulation, oxidative stress, apoptosis, and inflammatory responses while restoring autophagy. Mechanistically, asprosin inhibition activated AMPK and Sirt1 signaling while suppressing mTOR activation. PTPRD expression was markedly increased in diabetic kidneys and renal cells, and knockdown of PTPRD attenuated asprosin-induced metabolic signaling and cellular injury. Inhibition of AMPK with Compound C reversed the protective effects of asprosin inhibition.
Conclusions : Asprosin promotes renal injury in DKD by disrupting intracellular energy metabolism through PTPRD-mediated AMPK-Sirt1-mTOR signaling. Targeting asprosin signaling may represent a potential therapeutic strategy for metabolic stress–induced renal injury in diabetic kidney disease.