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Elucidating Asprosin's Role as an Intracellular Metabolism Regulator in Diabetic Kidney Disease

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Objectives : Asprosin, an adipokine with diverse functions, including inducing insulin resistance and promoting inflammation, is hypothesized to play a pivotal role in regulating cellular energy metabolism in diabetic kidney disease (DKD).
Methods : This study utilized type 2 diabetic db/db mice, dividing them into two groups: one received Asprosin neutralizing antibodies targeting the 28-amino acid peptide, while the other was treated with Metformin, an AMP-activated protein kinase (AMPK) activator for three weeks. Additionally, renal constituent cells (mesangial and glomerular endothelial cells) were cultured in high glucose and palmitic acid conditions, treated with an AMPK inhibitor (Compound C) after Asprosin expression was silenced using siRNA. We evaluated Asprosin, AMPK levels, and their downstream signaling pathways.
Results : Asprosin was found to be overexpressed in the serum and kidney tissues of db/db mice, as well as in renal cells under high glucose and palmitic acid conditions. Interfering with Asprosin resulted in reduced body and liver weight, improved glucose tolerance, and mitigated renal injury in vivo. Asprosin knockdown reduced lipid accumulation and inflammatory infiltration both in vitro and in vivo. Notably, Asprosin absence activated the AMPK/Sirts/mTOR signaling pathway. The AMPK inhibitor Compound C reversed Asprosin's effects on lipid accumulation and inflammation, confirming Asprosin's role in intracellular lipid metabolism.
Conclusions : This study reveals Asprosin's critical role in regulating intracellular energy metabolism, with potential implications for targeted therapies in DKD. Inhibiting Asprosin suppressed lipid accumulation and inflammation by activating the AMPK-associated signaling pathway, suggesting a promising therapeutic strategy for managing DKD.