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MicroRNA-5010-5p ameliorates high-glucose induced inflammation of renal tubular epithelial cells by modulating expression of PPP2R2D

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Objectives: We previously reported significantly upregulated circulating exosomal microRNAs in patients with diabetic kidney disease. However, their roles and molecular mechanisms in the kidney has not been elucidated yet. Among these miRNAs, this study focused on evaluating the impact of miR-5010-5p on renal tubular epithelial cells in a diabetic condition.

Methods: We used renal tubular epithelial cell line HK-2 and transfected it with miR-5010-5p mimic using recombinant plasmids. We identified the target gene of miR-5010-5p through a dual-luciferase assay. Expressions of mRNAs and proteins were evaluated by real-time polymerase chain reaction and western blot, respectively.

Results: High glucose (30mM) itself did not significantly affect intracellular expression of miR-5010-5p in HK-2 cells. With transfection of miR-5010-5p mimic, there was no change in cell viability. However, miR-5010-5p transfected HK-2 cells showed significantly ameliorated expressions of inflammatory cytokines increased with high-glucose stimulation, such as MCP-1, IL-1β, and TNF-α. These changes were accompanied by restored expression of phosphorylated AMP-activated protein kinase (AMPK) and decreased phosphorylation of Nuclear factor kappa B. The dual-luciferase assay showed that miR-5010-5p targeted gene expression of PPP2R2D, a subunit of protein phosphatase 2A known to modulate AMPK phosphorylation.

Conclusions: Our findings suggest that increased expression of miR-5010-5p reduces the high-glucose induced inflammatory response in renal tubular epithelial cells. This effect was mediated by regulating target gene, PPP2R2D, which modulated AMPK phosphorylation. Therefore, miR-5010-5p might be a promising therapeutic target for patients with diabetic kidney disease.