Shaping Tomorrow's Nephrology: Insight-Driven Kidney Care
KSN 2026 SEOUL, KOREA
The 46th Annual Meeting of the Korean Society of Nephrology
THE KOREAN SOCIETY OF NEPHROLOGY
Jun 11 (Thu) – 14 (Sun), 2026
COEX, Seoul, Korea

Abstract Type : Oral presentation
Abstract Submission No.: A-0727
Abstract Topic : Diabetic Kidney Disease + Metabolic Abnormality-related Kidney Disease

Pathophysiological Significance of Reduced Monocarboxylate Transporter (MCT) Expression in Diabetic Kidney Disease and a Novel Therapeutic Strategy through Its Restoration

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Objectives : Metabolic reprogramming and the subsequent accumulation of lactate in renal tubular epithelial cells are recognized as critical drivers in the progression of diabetic kidney disease (DKD). Monocarboxylate transporters (MCTs) play a vital role in maintaining intracellular metabolic homeostasis and pH by facilitating the transport of lactate. However, the specific expression patterns of MCT isoforms and their therapeutic potential in the context of DKD remain to be fully elucidated.
Methods : Human renal epithelial cells were cultured under either normal glucose or high glucose (HG, 35mM) conditions for 24 hours. To identify the dominant MCT isoform, the basal expression levels of MCT1, 2, and 4 were analyzed using Western blot. Furthermore, we evaluated the impact of HG on MCT expression and investigated whether treatment with AdipoRon, an adiponectin receptor agonist, could restore its expression.
Results : Western blot analysis revealed that MCT4 is the predominant isoform expressed in the human renal epithelial cell line used in this study. Exposure to high-glucose conditions resulted in a significant decrease in MCT4 expression compared to the control group. Notably, the administration of AdipoRon effectively restored the HG-induced reduction of MCT4 expression, suggesting a protective role for adiponectin signaling in tubular lactate transport.
Conclusions : Our findings demonstrate that high glucose impairs MCT4 expression in renal tubular cells, which may contribute to lactate-mediated cellular injury. The restoration of MCT4 by AdipoRon identifies a potential therapeutic strategy for DKD by enhancing metabolic clearance in the renal tubule.