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The role of Nuclear Receptors and Aquaporin 2 as a Mechanism for Renal water regulation by Abnormal Lipid Metabolism

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Objectives:

Lipids promote kidney disease progression by causing damage to both glomerular and tubular cells. Nuclear receptors (NR) are major regulators of adipocyte differentiation and energy storage and are also involved in the regulation of renal water and sodium homeostasis. Water channel protein (AQP) located in renal tubular epithelial cells plays a decisive role in water reabsorption and excretion. However, the underlying molecular mechanisms of NR expression changes and AQP2 expression abnormalities as a mechanism of renal water metabolism caused by abnormal lipids have not yet been clearly identified.

Methods:

After culturing M1 cells, an IMCD cell line, and treating LDL, cytotoxicity was confirmed over time through CCK8 assay, and lipid accumulation was confirmed through Oil red O staining.
Five-week-old male C57BL/6 mice were obtained from Hyochang science (Dague). For 13 weeks, mice were fed a normal diet or were fed a high-fat diet (HD, diet D12451; Rodent Diet with 45 Kcal% Fat).

Results:

When LDL concentration was treated, the expression levels of PPARγ and LXRα were significantly decreased. As a result of cell membrane damage, cytoplasmic LDLR showed a tendency to decrease unexpectedly, and lipid receptor CD36 and cholesterol efflux ABCA1 also decreased in the same tendency. As the LDL concentration increased, the expression of AQP2 decreased compared to normal, and the expression of α-SMA, Vimentin, and Fibronectin increased, leading to fibrosis in renal tubular cells.

Conclusions:

LDL-induced lipid accumulation and lipotoxicity in renal tubular epithelial cells are involved in the expression of nuclear receptors and affect the expression of water-regulating AQP2.

Therefore, it is expected that the difference in expression of nuclear receptor (NR) and water channel protein (AQP2) to abnormal lipid metabolism will be a basic study related to water metabolism in the kidney.