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Decreased of ring finger protein 20 induces abnormal lipid metabolism in renal injury model with obesity.

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Objectives: Abdominal obesity was confirmed to be a major factor that increases the mortality risk in patients with chronic kidney disease. However, the direct mechanism for exacerbation of kidney damage caused by obesity has not been clearly elucidated. Ring finger protein 20 (RNF20), an E3 ubiquitin ligase that plays various roles such as adipogenesis and DNA damage response, is known to regulate lipid metabolism in the kidney, but studies on lipid metabolism in exacerbation of chronic kidney disease have not been conducted.

Methods: Experiments were performed after treating human kidney epithelial (HK2) cells with palmitic acid (PA). In animal models, obesity and chronic kidney disease were induced through high-fat diet and nephrectomy. Expression of proteins related to renal tissue damage, lipid accumulation and lipid metabolism in cells and kidney tissues was confirmed by Western blotting.

Results: Lipid accumulation and cytotoxicity were confirmed in HK2 cells treated with fatty acids, and RNF20 expression was decreased and PPARa and SREBP1 expressions were changed. To investigate the relationship between lipids and fibrosis, as a result of treatment with TGF-beta and PA, it was confirmed that lipid accumulation increased, fatty acid oxidation decreased, and fibrosis-related proteins increased compared to the single treatment group. In animal experiments, the expression of RNF20 and PPARa was most decreased in the obesity-induced chronic kidney disease group. It was confirmed that protein expression related to this promotes intracellular lipid accumulation, not normal metabolism.

Conclusions: RNF20 is involved in fatty acid-induced lipid accumulation and lipotoxic processes in renal tubular cells. In the presence of fibrosis, RNF20 regulates proteins involved in lipogenesis and fatty acid oxidation, activating factors that promote lipid accumulation and fibrosis. Therefore, it means that RNF20 is an important factor in lipid-induced exacerbation of renal fibrosis in chronic kidney disease.