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Altered renal lipid metabolism by aging

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Objectives:

There are various genetic and environmental factors in the development of chronic kidney disease and renal aging. However, the role of lipid metabolism in kidney is still in the early stages of research. To elucidate the mechanism of aging and renal fibrosis in chronic kidney disease, we investigated the changes of lipid metabolism and abnormal metabolic pathway according to the aging process.

Methods:

Glomerular and tubular fractions were separated from renal aging models (8 weeks, 12 and 24 months) and the changes of lipid metabolites were analyzed by LC-MS / MS in each fraction.

Results: In aging model, renal function was significantly decreased in 24 months mice and glomerular hypertrophy and renal fibrosis were also increased in renal tissue.

The increase of CE was observed in the whole kidney, glomerular fraction, tubular fraction and interstitial fraction after aging and overall phospholipid were decreased by aging.

The major constituent fatty acids of CE in the kidney due to aging were mostly unsaturated fatty acids (PUFAs). CE was also confined to specific fatty acids.

The CE in the blood decreases with aging, and the change in the constituent fatty acids is also different from those in the aged kidney.

We finally isolated and analyzed the renal lipid droplets. The major change in LD following aging was the increase of CE, especially the increase of PUFA fatty acid in CE.

Conclusions:

Main feature of the altered renal lipid metabolism by aging was the accumulation of CE and this process was caused by abnormal intrarenal lipid metabolism rather than the secondary phenomenon to systemic aging.