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# Role of Lipid Accumulation in Renal Injury: Basic Study

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Lipids are essential for membrane integrity, and act as a source of energy and components of cell signaling in the kidney. However, disruption of balance between lipids load and metabolism results in ectopic lipid accumulation in the kidney, which can lead to direct and indirect renal injury. In the animal models of kidney disease, renal lipid accumulation of the kidney have been observed in both acute and chronic disease. Experimental studies on mice models of high fat diet induced obesity (DIO) and type 2 diabetes provide more direct evidence that altered lipid metabolism induces the progression of the chronic kidney disease. The affected model shows increased glomerulosclerosis and fibrosis in association with increased accumulation of triglycerides and cholesterol in the kidney, as well as increased lipid peroxidation. Several experiments using therapeutic agents targeting inflammatory and oxidative cascades in DIO and type 2 diabetic mice have shown improvement of renal injury with concurrent improvement of systemic hyperlipidemia and tissue lipid metabolism. Direct exposure to saturated free fatty acids on cultured podocytes significantly increases pro-inflammatory gene and cytokine secretion, which effects are augmented upon adding high glucose. Recent published studies have demonstrated that the mechanisms of altered lipid metabolism are linked with alteration in sterol regulatory element-binding proteins (SREBPs), decreased activity of AMPK-activated protein kinase and increased activity of acetyl-CoA carboxylase and fatty acid synthase. Dyslipidemia is a reversible and controllable risk factor in the clinical setting in patients with kidney disease. Targeting altered renal lipid metabolism may be a promising approach for providing protection against obesity and diabetes related kidney disease.

**Key words;**
Lipid accumulation
**Obesity**
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