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Hepatic Fatty Acid Metabolism in Chronic Kidney Disease: Effects of Niacin Supplementation

Kyubok Jin[1, 2], Yang Wook Kim[1], Nosratola D.Vaziri[2]

Department of Internal Medicine[1] Inje University Haeundae Paik Hospital Busan Korea
Division of Nephrology and Hypertension[2] University of California Irvine Irvine California

Background: Chronic kidney disease (CKD) results in profound dysregulation of lipid and lipoprotein metabolism which contribute to atherosclerosis and cardiovascular disease. Many studies have shown that central role of impaired clearance of triglyceride-rich lipoproteins in the pathogenesis of CKD-induced hypertriglyceridemia. We recently found niacin administration improved renal tissue lipid metabolism and renal function and structure in experimental CKD. Present study sought to determine efficacy of niacin supplementation on hepatic fatty acid metabolism in CKD.

Methods: Kidney function, lipid content, and expression of molecules involved in triglyceride and fatty acid metabolism were determined in untreated CKD (5/6 nephrectomized), niacin-treated CKD (50 mg/Kg/day in drinking water for 12 weeks) and control rats.

Results: CKD resulted in hypertension, proteinuria, hypertriglyceridemia, up-regulation of acyl-CoA cholesterol acyltransferase-1 (ACAT1), ACAT-2, carbohydrate-responsive element binding protein (ChREBP), fatty acid synthase (FAS), acyl-CoA carboxylase (ACC), and scavenger receptor class B type 1 (SR-B1) and down-regulation of sterol responsive element binding protein-1 (SREBP-1), SREBP-2, HMG-CoA reductase, PPAR-α, fatty acid binding protein (L-FABP), CPT1A, ATP synthase α, ATP synthase β, glycogen synthase, and diglyceride acyltransferase 1 (DGAT1), DGAT2. Niacin therapy attenuated hypertension, proteinuria, hypertriglyceridemia, reduced ChREBP, FAS, ACC abundance and raised SREBP-1, L-FABP, ATP synthase α, and DGAT1.

Conclusion: Niacin administration improves hepatic fatty acid metabolism preserves renal function and structure in experimental CKD.

Key Words: Fatty acid, TG, Anti-oxidants
Fatty acid, Triglyceride, Anti-inflammatory-antioxidant agents