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Niacin improves renal lipid metabolism and slows progression in chronic kidney disease.

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Abstract

BACKGROUND:

Mounting evidence points to lipid accumulation in the diseased kidney and its contribution to progression of nephropathy. We recently found heavy lipid accumulation and marked dysregulation of lipid metabolism in the remnant kidneys of rats with chronic renal failure (CRF). Present study sought to determine efficacy of niacin supplementation on renal tissue lipid metabolism in CRF.

METHODS:

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

RESULTS:

CRF resulted in hypertension, proteinuria, renal tissue lipid accumulation, up-regulation of scavenger receptor A1 (SR-A1), acyl-CoA cholesterol acyltransferase-1 (ACAT1), carbohydrate-responsive element binding protein (ChREBP), fatty acid synthase (FAS), acyl-CoA carboxylase (ACC), liver X receptor (LXR), ATP binding cassette (ABC) A-1, ABCG-1, and SR-B1 and down-regulation of sterol responsive element binding protein-1 (SREBP-1), SREBP-2, HMG-CoA reductase, PPAR-alpha, fatty acid binding protein (L-FABP), and CPT1A. **Niacin therapy attenuated hypertension, proteinuria, and tubulo-interstitial injury, reduced renal tissue lipids, CD36, ChREBP, LXR, ABCA-1, ABCG-1, and SR-B1 abundance and raised PPAR-alpha and L-FABP**.

CONCLUSIONS AND GENERAL SIGNIFICANCE:

Niacin administration improves renal tissue lipid metabolism and renal function and structure in experimental CRF.