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Omega-3 fatty acid attenuates renal fibrosis via AMPK mediated autophagy flux activation.

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Objectives: As concern renal fibrosis, the role of autophagy has been unclear through conflicting studies. The activation autophagy flux, authors hypothesize, would attenuate renal fibrosis progression. Previously authors reported that ω3-PUFAs in fat-1 mice, endogenously increased ω3-PUFAs transgenic animals, manifested autophagy activation via AMP-activated protein kinase (AMPK), and reduced the risk of I/R injury, with formation of antioxidant, UCP2 and p22phox in kidney tissue, attenuated oxidative stress.

Methods: In vitro, HK-2 cells in of TGF- β1 for 72 hours, were incubated with ω-3 PUFAs such as EPA and DHA. In vivo, BL/6 mice were divided into 4 groups: sham (n = 5), sham + ω-3 PUFA (n = 5), UUO (n = 10), and UUO + ω-3 PUFA (n = 10). For 2g/kg of ω-3 PUFA was administered daily via oral route, sacrificed UUO at 7^{th} day.

Results:) DHA/EPA reduced EMT in TGF-β treated HK2 cells on E-cadherin and α-SMA. 2) DHA/EPA induced autophagy flux in TGF-β treated HK2 cells on phosphorylation of AMPK, LC3-II and p62. 3) In vivo, ω-3 PUFA reduces renal oxidative stress and inflammation in UUO mice kidney TIS, 8-OHDG and F4/80, and MCP-1 and osteopontin 4) ω-3 PUFA reduces α-SMA, collagen IV, and fibronectin 5) ω-3 PUFA induced autophagy flux as beclin1, ATG7, LC3, ATP6E, Cathepsin D and p62, and colocalization of LC3-II and LAMP-1.

Conclusions: We demonstrated that ω3-PUFAs promote activation of autophagy and attenuate apoptosis and reduce renal fibrosis in UUO mice. It provides an insight for regulation of autophagy, suggests another therapeutic option for CKD patients.