

N-3 long-chain PUFA supplementation prevents high fat diet induced mouse liver steatosis and inflammation in relation to PPAR- α upregulation and NF- κ B DNA binding abrogation

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Scope: Dietary n-3 long-chain PUFAs (n-3 LCPUFAs) supplementation was studied in an HFD-induced (HFD is high-fat diet) steatosis and inflammation in relation to peroxisome proliferator-activated receptor alpha (PPAR- α) and nuclear factor κ B (NF- κ B) signaling. Methods and results: Male C57BL/6J mice received (i) control diet (10% fat, 20% protein, 70% carbohydrate), (ii) control diet plus n-3 LCPUFAs (daily doses of 108 mg/kg body weight of eicosapentaenoic acid plus 92 mg/kg body weight of docosahexaenoic acid), (iii) HFD (60% fat, 20% protein, 20% carbohydrate), or (iv) HFD plus n-3 LCPUFAs for 12 wk. PPAR- α , tumor necrosis factor alpha (TNF- α), and IL-1 β mRNA expression, acyl-CoA oxidase 1 (ACOX1), and carnitine-acyl-CoA transferase 1 (CAT-I) protein contents, and NF- κ B DNA binding activity were measured. HFD significantly decreased liver PPAR- α , ACOX1, and CAT-I levels with NF- κ B activation, higher TNF- α and IL-1 β expression, and steatosis development. These changes were either redu