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Eicosapentaenoic acid (EPA) prevents TNF-α-induced NF-αB and ERK 1/2 activation in 3T3-L1 adipocytes

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High levels of pro-inflammatory cytokine TNF-α in obesity have been associated with the development of insulin resistance. Eicosapentaenoic acid (EPA) is a polyunsaturated fatty acid of the omega-3 family found in fish and fish oils. Many studies have reported beneficial effects of this fatty acid on obesity and insulin resistance that might be linked to EPA's anti-inflammatory properties. Thus, the aim of the present study was to investigate the potential anti-inflamatory mechanisms of EPA in adipocytes. Seven days post-differentiation 3T3-L1 adipocytes were serum-starved overnight and then incubated with the appropriate treatment: EPA (100 and 200 μM) and/or TNF-α (1 ng/ml). NF-αB binding activity was analysed by electrophoretic mobility shift assay (EMSA) in 3T3-L1 adipocytes. Western blot was performed using antibodies for phospho-ERK 1/2 (Thr202/Tyr204) and ERK. Nuclear extracts from TNF-α-treated adipocytes showed a strong increase ($P<0.05$) in the abundance of NF-αB-DNA complexes as compared to control. EPA (100 and 200 μM) did not have any significant effect on NF-αB binding activity. However, the presence of EPA was able to partially prevent TNF-α-induced NF-αB-DNA binding. On the other hand, TNF-α treatment resulted in a positive activation of ERK 1/2 (Thr202/Tyr204) which was also prevented by EPA-treatment both in 3T3-L1 pre-adipocytes (completely) and adipocytes (partially). These findings suggest that EPA involves anti-inflammatory mechanisms in adipocytes which may contribute to explain the insulin sensitizing properties of this fatty acid.