

Abstract

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Eicosapentaenoic acid prevents and reverses insulin resistance in high-fat diet-induced obese mice via modulation of adipose tissue inflammation.

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OBJECTIVE: We investigated the effects of eicosapentaenoic acid (EPA) on prevention (P) and reversal (R) of high saturated-fat (HF) diet-induced obesity and glucose-insulin homeostasis.

METHODS: Male C57BL/6J mice were fed low-fat (LF; 10% energy from fat), HF (45% energy from fat), or a HF-EPA-P (45% energy from fat; 36 g/kg EPA) diet for 11 wk. A 4th group was initially fed HF for 6 wk followed by the HF-EPA-R diet for 5 wk.

RESULTS: As expected, mice fed the HF diet developed obesity and glucose intolerance. In contrast, mice fed the HF-EPA-P diet maintained normal glucose tolerance despite weight gain compared with the LF group.

CONCLUSIONS: Whereas the HF group developed hyperglycemia and hyperinsulinemia, both HF-EPA groups (P and R) exhibited normal glycemia and insulinemia. Further, plasma adiponectin concentration was lower in the HF group but was comparable in the LF and HF-EPA groups, suggesting a role of EPA in preventing and improving insulin resistance induced by HF feeding. Further analysis of adipose tissue adipokine levels and proteomic studies in cultured adipocytes indicated that dietary EPA supplementation of HF diets was associated with reduced adipose inflammation and lipogenesis and elevated markers of fatty acid oxidation. In C57BL/6J mice, EPA minimized saturated fat-induced insulin resistance and this is in part mediated by its effects on fatty acid oxidation and inflammation.

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