Anti-diabetic Effects of Eiosapentaenoic acid on Adiponectin receptors in the Liver
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Abstract
Objective: Type 2 diabetes (T2DM) is a metabolic disease characterized by hyperglycemia which results from insulin resistance and relative insulin secretion. Insulin inhibits hepatic glucose production, stimulates glucose uptake in cells, and inhibits lipolysis in adipose tissue [1]. When insulin resistance develops, this metabolic process is damaged which causes ectopic fat accumulation. Insulin signaling is mainly controlled by hormones and nutrient-sensitive signaling pathways and pro-inflammatory signals [2, 3]. Accumulating evidence suggests that n-3 PUFA, especially eicosapentaenoic acid (EPA), is known to increase insulin sensitivity in diabetic animal models [4, 5]. However, the molecular mechanism of omega-3 in reducing insulin resistance has not yet been fully elucidated. The aim of the present study was to evaluate for the first time the direct potential anti-diabetic effect of EPA on AdipoR1 and T-cadherin in liver.

Method: We examined the anti-diabetic effect of EPA on adiponectin receptors (T-cadherin, AdipoR1) in normal diet (ND), high-fat diet (HFD), and HFD-low dose STZ-induced T2DM rats.

Results: EPA supplementation was found to alleviate AdipoR1 mRNA level of Rat fed with ND and T2DM rats. Our results suggest that, at least in rat liver EPA supplementation ameliorated their hyperglycemia by increased activity of Adiponectin/AdipoR1 signaling pathways. We also found that T-cadherin levels were increased in response to EPA treatment in HFD fed Rats liver. This result suggests that EPA/T-cadherin balance may be an important predictor of the risk of developing HFD-induced abdominal obesity and obesity-related diseases.

Conclusion: Taken together, these observations support the hypothesis that EPA exerts an antidiabetic effect in ND-fed rats and T2DM rats, possibly through its action on Adiponectin/AdipoR1 signaling pathway. In addition to its anti-diabetic effects, EPA may contribute to improving HFD-induced obesity-related diseases by increase T-cadherin levels. These results need to be confirmed with further research.

Biography:
Zeynep Avcil is a Ph.D. student in department of clinical Biochemistry of Akdeniz University School of Medicine, Turkey. She has her expertise in diabetes and obesity.

Speaker Publications:

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