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Dietary u-3 polyunsaturated fatty acid intake modulates impact of insertion/deletion polymorphism of APOB gene on obesity risk in type 2 diabetic patients

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Objectives: The goal of the study described here was to determine whether dietary u-3 polyunsaturated fatty acid (PUFA) intake modulates the association between ApoB Ins/Del polymorphism and obesity in type 2 diabetic patients. Methods: In this cross-sectional study, 700 patients with type 2 diabetes were recruited in Tehran. Weight and waist circumference (WC) were measured, and body mass index (BMI) was calculated. Dietary intake was assessed using a validated semi quantitative food frequency questionnaire. APOB genotyping was performed with 8% polyacrylamide gel electrophoresis. Results: We observed a significant interaction between Ins/Del genotype and dietary u-3 PUFA intake with respect to BMI, WC, and obesity risk in both unadjusted ($P = 0.007$, $P = 0.001$, and $P = 0.021$, respectively) and adjusted ($P = 0.007$, $P = 0.04$, and $P = 0.002$, respectively) samples. Thus, the carriers of the

Del allele were only associated with lower BMI ($P = 0.01$) and WC ($P = 0.002$) among individuals with high u-3 PUFA intake ($\geq 0.6\%$ of energy), but not in those with low u-3 PUFA intake ($< 0.6\%$). Also, when dietary u-3 PUFA was $< 0.6\%$, general obesity risk in carriers of the Del allele was about 1.6 times higher than that of Ins/Ins homozygotes (odds ratio = 1.59, 95% confidence interval: 1.05–2.52, $P = 0.039$). But with high u-3 PUFA intake ($\geq 0.6\%$), the risk was 0.46 times lower (odds ratio ≈ 0.46 , 95% confidence interval: 0.25–0.79, $P = 0.003$). Moreover, a similar interaction was observed in central obesity only in men after adjustment for confounder variables ($P = 0.041$). Conclusions: These findings support the hypothesis that a diet high in u-3 PUFA ($\geq 0.6\%$) can decrease the obesity risk in carriers of the Del allele of ApoB gene.

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