Effects of administration of omega-3 fatty acids with or without vitamin E supplementation on adiponectin gene expression in PBMCs and serum adiponectin and adipocyte fatty acid-binding protein levels in male patients with CAD

To the Editor,

We read the article written by Ramezani et al. (1) published in Anatol J Cardiol 2015; 15: 981-9 entitled "Effects of administration of omega-3 fatty acids with or without vitamin E supplementation on adiponectin gene expression in PBMCs and serum adiponectin and adipocyte fatty acid-binding protein levels in male patients with CAD" with great interest. The authors found that n-3 PUFAs with or without vitamin E affected adiponectin levels. Moreover, additive effect on plasma adiponectin was suggested.

An interesting aspect of this study was the study population, which may be described as "normal-weight obese." Although the inclusion criterion was body mass index (BMI) ≤30 kg/m^2, waist circumference (WC) in each study subgroup exceeded the threshold values of abdominal obesity (>94 cm for men and >80 cm for women). Also, waist to hip ratio (WHR) in each subgroup was >0.8 for women and >0.9 for men, indicating excess abdominal fat distribution. The authors of the current study diagnosed obesity on the basis of World Health Organization BMI criteria. There is some data indicating that while BMI is not a sufficiently specific or sensitive tool to assess obesity and metabolic status, additional methods such as WC and WHR measurement at least provide additional, valuable information (2).

Since adiponectin is perceived as cardioprotective substance, multiple studies have focused on the possibilities of increasing its concentration. Our study showed improvement of adipose tissue endocrine activity and increase of adiponectin level after 1 month of n-3 PUFAs supplementation in patients with stable CAD who underwent percutaneous coronary intervention (3). In that study, independent predictors of changes in adiponectin concentration after 1 month of n-3 PUFAs supplementation were n-3 PUFAs treatment, baseline platelet count, and total cholesterol. It would be interesting to see what the predictors of changes in adiponectin levels during combined n-3 PUFAs and vitamin E supplementation were in the current study.

We wonder why the authors chose a "non-obese" study population. It is not common practice to exclude obese patients from research on endocrine activity of fat tissue. However, n-3 PUFAs have been found to incorporate into lipid fractions of the adipocytes (4). One of the potential mechanisms in which n-3 PUFAs increase adiponectin secretion is peroxisome proliferator-activated receptor-gamma stimulation, expression of which in adipose tissue is much greater than in other tissues (4). Therefore, it is possible that the amount of fat tissue affects impact of n-3 PUFAs on hormones. Puglisi et al. (5) suggested that n-3 PUFAs may downregulate leptin, another fat tissue hormone, in association with reduced adiposity or upregulate its level in association with increased adiposity. Thus, it cannot be excluded that impact of n-3 PUFAs on adiponectin is also dependent on amount of fat tissue. In our opinion, since the authors of the current study did not observe any influence of n-3 PUFAs on adiponectin gene expression in subjects with BMI <30 kg/m^2, it would be advisable to investigate this relationship in obese population, which potentially may benefit the most from this intervention.

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