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Fish oil supplementation has beneficial effects on numbers of circulating endothelial progenitor cells and microparticles independent of eNOS genotype

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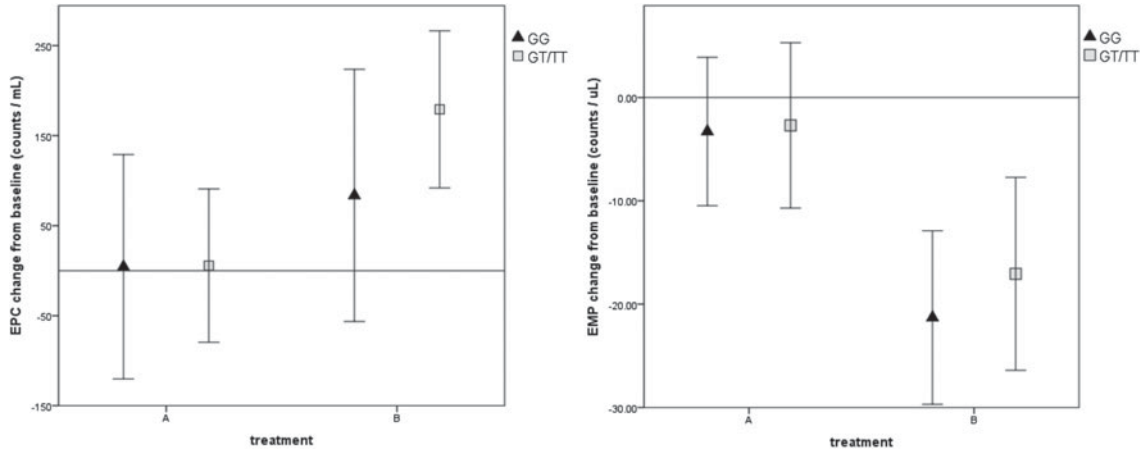
A growing body of evidence suggests that endothelial dysfunction plays a crucial role in all stages of atherosclerosis. Numbers of circulating endothelial progenitor cells (EPC) and endothelial microparticles (EMP) are emerging as novel markers of endothelial dysfunction and CVD risk; low numbers of EPCs indicate limited repair of the endothelium, and high numbers of EMPs indicate endothelial damage.[1,2] In addition, the Glu298Asp (or 894G→T) polymorphism in the eNOS gene has been associated with CVD and reduced endothelial function, although it is not clear whether this affects EPC or EMP numbers[3]. However, recent data suggests that supplementation of *n*-3 PUFA significantly improves endothelial function[4]. Therefore, the aim of the present study was to investigate the effect of fish oil supplementation on the EPC and EMP levels in individuals prospectively genotyped for the Glu298Asp polymorphism.

A double-blinded, randomized, placebo-controlled, cross-over trial was performed in 91 non-smoking healthy adults (21–65 y), at moderate risk for CVD, who were genotyped for the eNOS Glu298Asp polymorphism and assigned to a GG ($n = 46$) or GT/TT ($n = 45$) group. Subjects were randomized onto either fish oil (1.8 g/d *n*-3 PUFA) or placebo (corn oil) for 8 weeks, followed by an 8-week washout and cross-over to the other treatment for another 8 weeks. Plasma EPC and EMP levels were measured by flow cytometry at the beginning and end of each supplementation phase. EPC and EMP were defined as $CD34^+KDR^+$ and $CD42b^-CD31^+$, respectively, and were expressed as absolute numbers using TruCOUNT tubes.



Change in EPC (left) and EMP (right) counts after 8-week treatment with placebo (A) or fish oil (B).

Supplementation with fish oil significantly increased numbers of circulating EPC ($P = 0.019$), but decreased numbers of EMP ($P < 0.001$) compared with the control period. However, there was no influence of eNOS genotype ($P = 0.49$ and 0.32 for EMP and EPC, respectively; linear mixed model). The current study therefore indicates that *n*-3 LC-PUFA might improve endothelial function by modulating endothelial damage and repair, regardless of the eNOS genotype of the individual.

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