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Antioxidant effects of vitamin E in hyperlipoproteinemias

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Of human plasma lipoproteins, LDL (low density lipoproteins) are most susceptible to oxidation [1]. LDL inhibit prostacyclin (PGI_2) biosynthesis by endothelial cells [2, 3] and this inhibition is due to LDL-lipid peroxides formed during centrifugation, dialysis and storage of LDL fractions [4]. Similarly, peroxidation of LDL *in vitro* results in their cytotoxicity toward endothelial cells, an effect which can be prevented by antioxidants [5]. Finally, modification of LDL by malondialdehyde which is formed during peroxidation, may be a prerequisite to the accumulation of cholesteryl esters within the cells of atherosclerotic lesions. All these findings indicate that peroxidation of LDL leads to its atherogeneity. Spontaneous oxidation of LDL *in vivo* might promote development of atherosclerosis.

In a placebo-controlled trial 27 healthy volunteers and 58 patients with hyperlipoproteinemias types II and IV received orally vitamin E at doses of 300 mg or 600 mg daily for 14 days. Serum tocopherol levels increased by 2–2.5 fold in healthy subjects and in patients with hyperlipoproteinemias. Doubling of the dose of vitamin E (600 mg) did not lead to its further serum accumulation. Serum concentrations of total lipids, cholesterol, triglycerides, ceruloplasmin and transferrin remained unchanged. In patients with hyperlipoproteinemias, vitamin E effectively depressed elevated concentrations of plasma lipid peroxides (estimated by a modification of the thiobarbituric acid procedure) down to levels of healthy subjects. This effect of vitamin E was most pronounced in patients with high levels of plasma lipid peroxides whose serum antioxidant activity was weakened. In these patients vitamin E also increased serum antioxidant activity. In patients, a mild platelet suppressant effect of vitamin E (600 mg) was observed, as expressed by depressed aggregatory responsiveness to arachidonic acid and ADP, but not to collagen. These results are in agreement with a previous study [1] on the effects of vitamin E on experimental atherosclerosis induced by high-fat diet. They indicate that in hyperlipoproteinemias vitamin E corrects certain abnormalities of lipid metabolism which might predispose to atherosclerosis.

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