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Letters to the Editor

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Letters to the Editor

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Effects of Vitamin E on the Expression of the Adhesion Molecule P-Selectin

To the editors:

A number of studies provides evidence that the antioxidant vitamin E may be a prevention factor in the development of atherosclerosis which can reduce the risk of coronary artery disease (CAD). In 1996, the Cambridge Heart Antioxidant Study (CHAOS) [1] gave distinct advice that the risk of infarction for patients with CAD decreases under treatment with vitamin E (400 respectively 800 IU daily). In January 2000, the Heart Outcomes Prevention Evaluation (HOPE) Study Investigators [2] found at variance with this that the treatment with vitamin E (400 IU daily) has no apparent effect on cardiovascular outcomes. This topic continues to be matter for discussion, and results about effects of vitamin E supplementation on parameters in development of atherosclerosis are going on to be of interest.

High P-selectin values indicate endothelial cell injury, activation of platelets and the risk of acute cardiac events in patients with CAD [3, 4]. It is known that P-selectin mediated vascular adhesion is an important factor in the development of early atherosclerotic lesions and thrombus formation [5].

In a double-blind randomised study following the GCP-guidelines (good clinical practise) we selected 60 patients 53.9 $\pm$ 7.9 years of age with disturbed lipid metabolism (LDL-cholesterol > 160 mg/dl) and a mean body mass index of 27.9 $\pm$ 3.1. One group was treated with 3 x 400 IU vitamin E (RR-alpha tocopherol from natural sources; etocomed, Richter Pharma, Austria) + pravastatin 20 mg (pravachol, Bristol-Myers Squibb, Germany), the second group with placebo + 20 mg pravastatin daily for 3 weeks. Serum levels of the adhesion molecule granule membrane protein-140 (GMP-140) were determined using a solid phase enzyme immunoassay (human soluble P-Selectin, R&D Systems, UK/USA).

After treatment with vitamin E we found significantly decreased serum levels (mean values changed from 95 to 82 ng/ml) of the P-selectin GMP-140 ($p < 0.03$), whilst the placebo group showed no significant changes (Fig. 1). Regarding the end points after 3 weeks, the difference of the mean values between both groups was significant ($p < 0.02$).

We found no additional effect of vitamin E on serum LDL-cholesterol levels. The beneficial lowering of the LDL-cholesterol in both groups can be associated with the effects of the pravastatin treatment. Median LDL values changed from 194 to 137 in the vitamin E + pravastatin group ($p < 0.00001$) and from 196 to 129 in the placebo + pravastatin group ($p < 0.00001$).

To our knowledge, the results give first indications that therapy with vitamin E in high dosage – more than vitamin E concentrations in the above mentioned studies – can influence the expression of GMP-140, a marker of endothelial cell injury and platelet activation which are primary events in the pathogenesis of coronary artery diseases.

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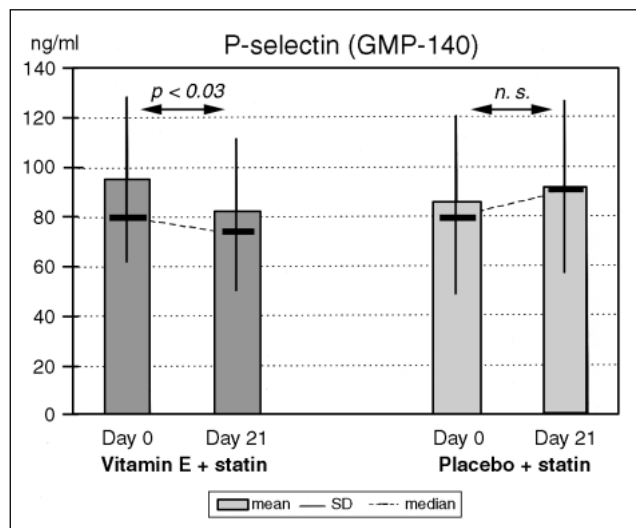


Figure 1. Changes of the p-selectin GMP-140 in serum of 60 patients with dyslipidaemia; 3 weeks treatment with vitamin E + statin compared to placebo + statin

Lack of Changes in Serum-Homocysteine Levels in Patients with Acute Myocardial Infarction Treated with Thrombolytic Therapy

To the editors:

Homocysteine is a sulphur containing amino acid that plays an important role in methionine and folate metabolism [1]. Since the early study of Wilcken and Wilcken [2] many case control studies have shown that homocysteine is an independent risk factor for atherosclerotic vascular disease and for venous thrombosis. Several factors influence total plasma homocysteine (tHcy) metabolism and variation in fasting tHcy in patients with acute occlusive vascular events with lower concentrations during the first hours [3]. It remains uncertain whether the event really depresses tHcy or triggers a subsequent increase in tHcy due to a cascade of pathophysiological changes [4]. The effect of fibrinolytic drugs for treatment of acute myocardial infarction on tHcy is uncertain.

We assessed tHcy in seven consecutive patients with acute transmural myocardial infarction and systemic thrombolytic therapy with alteplase, streptokinase or lanoteplase, aged between 37 and 83 years (mean 61.7). Serial measurements of tHcy were performed immediately after hospital admission, after application of the thrombolytic agent, four and 24 hours later. The measurement of the first three samples was done after fasting from time of admission, the last sample was assessed after fasting for at least 12 hours [5].

We found no significant variation in tHcy concentrations during and shortly after treatment of acute transmural myocardial infarction with fibrinolytic drugs. The mean plasma tHcy concentrations were 13.87 $\mu\text{mol/l}$ (SD 5.15) on admission, 13.53 (5.03), 12.78 (4.15) and 12.18 (4.05), respectively (figure 1; p for trend across time of tHcy-measurement = 0.42).

Our study suggests no apparent effect of systemic fibrinolytic therapy for acute myocardial infarction on tHcy concen-

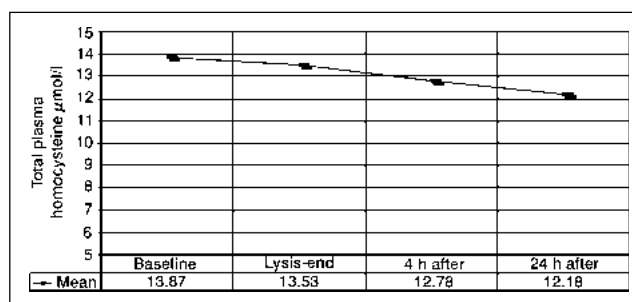


Figure 1. Mean total plasma homocysteine (tHcy) concentrations during serial measurements immediately after hospital admission, after application of the thrombolytic agent, four and 24 hours later (p for trend across time of tHcy-measurement = 0.42).

tration and, in agreement with Egerton and colleagues [3], no significant variation in tHcy during the first 24 hours after an acute occlusive coronary event.

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