healthy study participants were analyzed by whole-blood aggregometry. Platelet aggregation was determined by the increase in impedance across paired electrodes in response to the agonists adenosine diphosphate (ADP) and adrenaline, respectively. For each participant, changes in platelet aggregation were calculated using the response before and after administration. Blood samples from healthy study participants were studied after addition of ACE inhibitors in different dosage titrations. Results: The extent of change in aggregation was determined by comparing the area under the curve (AUC) for each participant before and after administration of ACE inhibitors. An increase in AUC suggests a decrease in platelet aggregation. The results were divided into two groups: those with a decrease in AUC of 10% or greater and those with a decrease of less than 10%.

Conclusion: This study found that ACE inhibitors had a significant effect on platelet aggregation in healthy study participants. The results suggest a potential role for ACE inhibitors in the prevention of thrombosis and myocardial infarction.

1132-120 Effects of Vascular Endothelial Growth Factor on Proliferation and Differentiation of Embryonic Stem Cells

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Our previous study showed that compared to transplantation of embryonic stem cells (ESCs) alone in infarcted hearts, improvement of cardiac function was significantly greater in post-infarcted mice transplanted with ESCs expressing vascular endothelial growth factor (VEGF). Accumulated evidence demonstrates that VEGF affects embryonic development and stimulates vascular growth. In the present study, we investigated the effects of VEGF on ESC proliferation and differentiation in the presence of leukemia inhibitory factor (LIF). ESC proliferation was measured by the MIT method (Cell proliferation kit I, sigma). Compared to the control group, 20 ng/ml VEGF did not affect proliferation of ESCs cultured in the presence of LIF (1000 units/ml) at 6 and 11 days. However, in the presence of LIF, 20 ng/ml VEGF significantly enhanced ESC proliferation. The number of ESC colonies was increased from 80 ± 0 to 170 ± 7. Treatment with 20 ng/ml VEGF increased the number of ESCs at 8 days by 51 ± 5% (n = 7) and by 70 ± 7% (n = 7) at 11 days in culture. Similar results were observed in the experiments with ESCs transfected with VEGF cDNA (pVEGF165). In addition, the hanging drops method was used to evaluate differentiation of ESCs cultured in the absence of LIF. The portion of ESCs that differentiated into cardiac myocytes was significantly increased in the presence of VEGF.

1132-121 Angiotensin Receptor Type 1 (AT1) Independent Growth Effects of Intracellular Angiotensin II (Ang II) in Cardiac Myocytes

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Background: Cardiac remodeling and growth can be elicited through autocrine and paracrine actions of Ang II, via binding to the AT1, plasma membrane receptor. An intracellular role for Ang II has also been elucidated; however, this role is greatly obscured by a very rapid breakdown of Ang II by plasma membrane or nuclear receptor.

Methods: We have previously demonstrated the anti-atherosclerotic effects of fish oil. Uptake of oxidized low-density lipoprotein (ox-LDL) by endothelial cells is an early step in atherogenesis. Ox-LDL upregulates expression of adhesion molecules, such as P-selectin and intracellular adhesion molecule-1 (ICAM-1). We hypothesized that fish oil may reduce expression of these adhesion molecules.

Results: Ox-LDL markedly increased the expression of P-selectin and ICAM-1 (both protein and mRNA) in HCAECs. Enhanced activity of myocytes to the cultured endothelial cells, and inhibited the activity of PBK. Both EPA and DHA decreased ox-LDL-induced upregulation of P-selectin and ICAM-1 expression and adherance of monocytes, and increased the activity of PBK (P<0.05 vs. ox-LDL alone, n=6). The effects of 50 μM concentration were more pronounced than the effects of 10 μM (P<0.05).

Conclusion: Our data suggest that EPA and DHA may be effective in reducing atherosclerosis by reducing the expression of adhesion molecules and the adherence of monocytes to the endothelial cells. These effects of EPA/DHA may underlie the anti-atherosclerotic effects of fish oil.