The mechanism of action of α-tocopherol at a cellular level (Grant 268)

Angelo Azzi, Institut für Biochemie und Molekularbiologie, Universität Bern, 3012, Bern, Switzerland.

The effects of α-tocopherol has been studied in rat and human aortic smooth muscle cells. α-Tocopherol inhibits smooth muscle cell proliferation and protein kinase C activity in a dose-dependent manner, at concentrations ranging from 10 to 50 μM. The effect of α-tocopherol appears not to be shared by the analogue β- tocopherol, provided with similar radical-scavenging properties. The data are interpreted in terms of α-tocopherol specifically causing activation of the phosphatase PP2A, followed by a diminution of protein kinase C phosphorylation and a decreased enzyme specific activity. The diminution of protein kinase C is causal in the inhibition of cell proliferation.

We have shown that a protein (TAP) is present in most tissues. It binds α-tocopherol and distinguishes it from very similar molecules. Mediated by this protein therefore, α-tocopherol may exert the function of regulating the transcription of a number of genes such as CD36 (scavenger receptor, acid collagenase and α-tropomyosin.

The newly established molecular basis of α-tocopherol action may explain some of the intriguing epidemiological results showing protection by data against atherosclerosis and some types of cancer.