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Changes in Superoxide Dismutase Activity and mRNA Levels in Vivo after Short-term Supplementation with Vitamin E in Rats

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Key Words
α–Tocopherol (vitamin E), Superoxide dismutase (SOD), mRNA, Wistar rats.

ABSTRACT

Vitamin E (α-tocopherol) is a lipophilic naturally existing antioxidant that prevents cell membranes from lipid peroxidation by scavenging oxygen free radicals. Superoxide dismutase (SOD), catalase (CAT), and glutathione peroxidase (GPX) are endogenous enzyme systems which can protect cells from free radical damage by catalyzing superoxide anions. The effect of exogenous antioxidant supplementation on SOD of vital organs is still unclear. This study was undertaken to evaluate the effect of short-term vitamin E treatment on gene expression and SOD activity. Two-month-old male Wistar rats were injected intraperitoneally with vitamin E (200 mg/ml/kg) or normal saline (1 ml/kg) daily for 3 consecutive days. Then, whole organs including liver, kidney, and brain were used to carry out assays of SOD activity and mRNA levels. After vitamin E supplementation, the SOD-mRNA level and activity had increased about 50% compared to the control groups ($p < 0.01$), and this increase was more significant in liver ($p < 0.001$). As for catalase (CAT) and glutathione peroxidase (GPX), the effect of vitamin E on changes of enzyme activity was parallel to that of SOD. In conclusion, a short-term optimal dose of vitamin E supplementation is effective in increasing SOD activity and mRNA gene expression in the liver and other vital organs. Whether this effect is beneficial in a clinical setting needs further evaluation. (N. Taipei J. Med. 2001; 3:182-186)

INTRODUCTION

α-Tocopherol, a major component of vitamin E, is well known to represent the last possibility of preventing membrane peroxidation by scavenging lipid peroxyl ($LOO^{\bullet}$) radicals involved in peroxidation chains.[1] Vitamin E has an OH phenolic group responsible for its antioxidant activity and a phytyl side chain ($C_{16}H_{33}$) which favors its insertion into the lipid bilayer region.

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