# Effect of vitamin E supplementation on adriamycin induced changes in oxido-reductive status of mouse red blood cells

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## ABSTRACT

The effect of oral vitamin E supplementation on the oxido-reductive status of red blood cells in normal mice and those subject to oxidative stress by chronic administration of the anti-tumour drug Adriamycin was investigated. Mice were randomly separated into three groups of 20 animals each and maintained on diets identical in all respects except for vitamin E content. Group 1 received a low vitamin E died that provided 10mg vitamin E/kg body weight/day, Group 2 received a normal mice chow died (45mg vitamin E/kg body weight/day) while group 3 received a high vitamin E died (200 mg Vitamin E/kg body weight/day)

In comparison with the normal mice in group 1, their counterparts in groups 2 and 3 exhibited significantly higher ($P<0.001$) activities of superoxide dismutase (SOD) in red blood cells (79.4% higher in group 2 and 114.2% higher in group 3, respectively) and produced lower concentrations of malondialdehyde (MDA) (22.9% less in group 2 and 51.2% less in group 3, respectively), with little difference in the glutathione peroxidase (GPX) activity. In Adriamycin-treated animals on the low vitamin E died (group 1) the red blood cell SOD activity and MDA production were 46.2% and 200.7% higher ($P<0.001$), respectively, and the GPX activity was 39.1% lower than in the red blood cells of untreated (normal) animals in the same group. The Adriamycin-induced changes were significantly less in animals receiving higher doses of vitamin E (groups 2 and 3). Thus, in the group maintained on the high vitamin E died (group 3), Adriamycin administration resulted in only a 38.9% incease in the MDA production above that generated by red blood cells of normal mice in the same group, with no significant change in the SOD or GPX activities. Thus, in normal conditions as well as in condition of oxidative stress, high doses of vitamin E appear to be able to protect the oxido-reductive status of red blood cells by modulating the extent of lipid peroxidation as the activities of antioxidant enzymes.