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Protective Effect of Vitamin E in Stagnant Hypoxia of the Brain

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Summary

The author demonstrated the protective effect of intraperitoneally administered vitamin E on the resistance of the laboratory rat to stagnant hypoxia induced by 5*xg* positive radial acceleration. A dose of 300 mg/kg body weight proved to be the most effective.

Key words
Hypoxia of the brain – Vitamin E – 5*xg* positive radial acceleration

Introduction

The reaction of biological metabolites with oxygen is accompanied by the formation of reactive intermediate products, including free radicals, superoxides and hydroxyl and hydroxyperoxide radicals. The resultant oxidations form a basis for the toxic action of oxygen radicals and if the peroxidation of lipids, the oxidation of thiols and enzymatic inactivations overcome the physiological anti-oxidative capacity of the tissues, they give rise to oxidation stress (Müller-Peddinghaus 1981). The radical (HO_2), which has no charge, is particularly toxic because it is lipophilic. It is most dangerous for membranes and since reactive hyperaemia occurs in the brain during the post-hypoxic phase (Trojan and Kapitola 1989), the danger of oxygen having a toxic effect is much greater. We therefore studied the effect of vitamin E on the resistance of laboratory rats of different ages to stagnant hypoxia induced by 5*xg* positive radial acceleration.

Material and Methods

Experiments were carried out with 306 3-month-old laboratory rats (Wistar strain, our own breed) reared under standard conditions (Velaz diet, environmental temperature 23±1 °C, a regular light regime). Stagnant hypoxia of the CNS was induced by positive radial acceleration of 5*xg* intensity on a special centrifuge (Trojan and Jílek 1967). The criterion of survival was irreversible respiratory arrest. Vitamin E was administered intraperitoneally as tocopherol aceticum (EREVIT, Biotika), 1 h before exposure to 5*xg* positive radial acceleration in doses of 30, 150, 300 and 450 mg/kg. The results were evaluated as LD 50 after Behrens and Schlosse (1957).

Results

The results are given in Fig. 1. In a large dose, vitamin E increased the resistance of rats to oligaemia induced by 5*xg* positive radial acceleration. No differences were found between the two sexes.

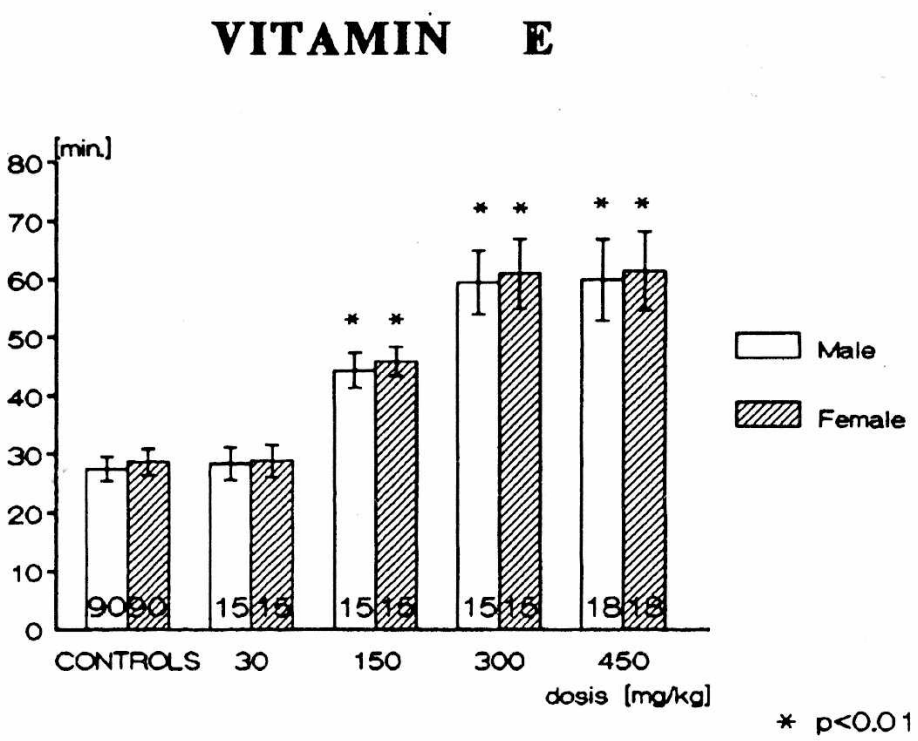

Fig. 1
Changes in the resistance of laboratory rats of both sexes to stagnant hypoxia (5*xg* positive radial acceleration) after the administration of vitamin E, expressed as LD 50. The doses of vitamin E are given on the abscissa and resistance (respiratory arrest) in minutes on the ordinate. The figures in the columns denote the number of animals in the various groups.

Discussion

Positive radial acceleration of 5*xg* intensity leads to oligaemia of the CNS by limiting venous return (Trojan 1978). During oligaemia, the concentration of oxygen radicals rises (Přibyl and Lochman 1984). Thanks to their enzymes (e.g. superoxide dismutase, glutathione peroxidase, catalase), aerobic organisms are normally able to decompose radicals promptly as they are formed (Trojan and Šťastný 1988). If the enzymes are unable to cope, non-enzymic anti-oxidants – e.g. vitamins C and E – are effective (Burton *et al.* 1983, Nagaoka *et al.* 1990). We attribute the protective effect of large doses of vitamin E on the resistance of the respiratory center to stagnant hypoxia both to its anti-oxidant properties (Chiswick *et al.* 1983) and to its stabilizing effect on cell membranes (Imaizumi *et al.* 1988). The finding that the largest dose of this vitamin used here did not raise resistance any further indicates that vitamin E has a strong supportive effect on the oligaemic nervous tissue, but that it is not able to prevent further, irreversible changes.

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