

Antioxidant Properties of Rimantadine in Influenza Virus Infected Mice and in Some Model Systems

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Z. Naturforsch. **55c**, 824–829 (2000); received April 13/June 15, 2000

Rimantadine, Lipid Peroxidation, Scavenger

Influenza virus infection is associated with development of oxidative stress in lung and blood plasma, viz. increase of primary and secondary lipid peroxidation products. It was established that rimantadine treatment led to a decrease of the products of lipid peroxidation in tissues of mice experimentally infected with influenza virus A/Aichi/2/68 (H3N2). The effect is strongest in blood plasma (a decrease of about 50%) and weaker in the lung (about 20%). To elucidate the mechanism of this action of rimantadine, experiments were carried out with some model systems. The capability of rimantadine to scavenge superoxide radicals (scavenging properties) was studied in a system of xanthine-xanthine oxidase to generate superoxide. The amount of superoxide was measured spectrophotometrically by the NBT-test and chemiluminescence. Rimantadine does not show scavenging properties and its antioxidant effect observed *in vivo*, is not a result of its direct action on the processes of lipid peroxidation and/or interaction with antioxidant enzymes. The antioxidant properties of rimantadine were investigated by measurement of induced lipid peroxidation in a Fe²⁺ and (Fe²⁺ – EDTA) system with an egg liposomal suspension. Our findings with model systems do not prove an antioxidant or prooxidant effect of the drug on the processes of lipid peroxidation. Apparently, the observed antioxidant effect of rimantadine *in vivo* is not connected directly with free radical processes in the organism.