ACTA BIOLOGIAE ET MEDICINAE EXPERIMENTALIS

CONFERENCE
of the
Serbian Physiological Society

December 20-23, 2001
Zlatibor, Yugoslavia

VOLUME 27
NUMBER 1
SUPPLEMENT
2002

YU ISSN0350-5901

PUBLISHED BY THE UNIVERSITY OF PRIŠTINA,
SOCIETY FOR BIOLOGY AND EXPERIMENTAL MEDICINE APKM &
THE SERBIAN PHYSIOLOGICAL SOCIETY

A32.
EFFECTS OF COENZYME Q10 AND VITAMIN E ON ANTIOXIDANT DEFENSE SYSTEM IN THE BLOOD OF RATS ACUTELY EXPOSED TO CADMIUM

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Cadmium induced lipid peroxidation (LPO) and altering the antioxidant system after the administration of a single dose of $CdCl_2$ (0.4 mg /kg body wt, i.p., 24 hours before the sacrificing) was studied in the blood of rats. The protective role of coenzyme Q (20 mg CoQ_{10}/kg body wt, i.m., 48 hours before the sacrificing) and vitamin E (20 iu Vit E/kg body wt, i.m., 48 hours before the sacrificing) was also studied in the blood of rats after the acute intoxication with cadmium. The treatment with Cd increased LPO in the blood while animals pretreated with coenzyme Q and/or vitamin E prior to Cd treatment showed decreased LPO as compared with animals given Cd alone. Activities of superoxid dismutase (SOD), catalase (CAT), glutathione peroxidase (GSH-Px) and glutathione reductase (GR) in erythrocytes and activities glutathione S-transferase (GST) as well as concentrations ascorbic acid (AsA) and vitamin E (Vit E) in the plasma were significantly increased of Cd and CoQ_{10} treated rats ($p<0.05$ or less). The pretreatment with coenzyme Q and/or vitamin E prior to Cd administration partially reversed such changes. In animals treatment with antioxidants prior to Cd treatment decreased significantly activities of SOD, CAT and GR in erythrocytes, and activity GST and concentrations AsA in the plasma and were the same as controls values. However, the pretreatment with coenzyme Q and/or vitamin E prior to Cd administration showed increased erythrocyte GSH-Px activity and concentrations vitamin E in the plasma as compared with animals given Cd alone. The results sugges that Cd intoxication induces oxidative stress and alters the antioxidant system, resulting in increased lipid peroxidation to rat erythrocytes. Coenzyme Q and vitamin E are the powerful liposoluble antioxidants which in cells quenched free oxygen radicals, inhibit lipid peroxidation and thus prevent free-radical mediated oxidant injuries. It is known that coenzyme Q and vitamin E may act sinergically as antioxidanrs. The pretreatment with coenzyme Q and/or vitamin E prior to Cd treatment showed protective effect against toxic influence of Cd on peroxidation of membrane lipids and altering the antioxidant system in the blood of rats.

A33.
NITRIC OXIDE (NO) DONORS-INDUCED OXIDATIVE STRESS IN RAT RETICULOCYTES

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Oxidative stress was defined as a disturbance in the prooxidant-antioxidant balance in favor of the former. Nitric oxide (NO), as a free oxygen radical, in high doses induced many harmful effects in cells. The aim of this study was to evaluate the role of nitroglycerin (NTG) and sodium nitroprusside (SNP), a NO donors, on oxidative stress parameters in rat reticulocytes. Rat reticulocyte rich red blood cell suspensions containing 60-90 % of reticulocytes, were aerobically incubated (a) without (control) or in the presence of different concentrations of (b) NTG and SNP (0.1, 0.25, 0.5, 1.0 and 1.5 mmol/l). After two hours of incubation the next parameters were followed in cell suspensions: (1) concentration of superoxide anion ($O_2^{\cdot-}$) and nitrite (NO_2^-, a stable metabolic product of nitric oxide oxidation), (2) methemoglobin (MetHb) and Heinz body levels and (3) lipid peroxide (TBARS) levels. Results of this study showed that $O_2^{\cdot-}$ decreased in the presence of low doses of NTG and SNP ($p < 0.5$), while high concentrations of NO donors elevated superoxide anion levels to control values. Nitrite concentration followed the same characteristics, indicating NO inactivation by $O_2^{\cdot-}$ to form peroxynitrite. MetHb increased for 192 and 109 % ($p < 0.05$), respectively, in the presence of NTG and SNP. However, Heinz bodies' increase is not significantly ($p > 0.05$). Control level of TBARS amount to 6.40±0.60 nmol/ml cells. NTG and SNP dose-dependently increased lipid peroxidation, even for 75 and 91 % ($p < 0.05$), respectively, in the presence of 1.5 mmol/l NTG and SNP. Our study showed that NO donors in experimental concentrations increased oxidative damage in rat reticulocytes.