

Antioxidant steroids and the expression of the gene of superoxide dismutase enzyme

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According to our earlier data most steroid end hormones and some intermediate metabolites of the steroidogenesis are able to inhibit the production of free radical superoxide anion.

In our study we intended to investigate, whether gene-expression of the important antioxidant enzyme superoxide dismutase (SOD) changes after incubation with steroidal compounds.

Peripheral blood samples were collected from healthy volunteers (men and women, aged 20-30 years). After the neutrophil cell separation four different steroid treatments (oestradiol, progesterone, testosterone and cortisol; all in 10^{-8} M concentration, for 2 hours and at 37°C) were performed on 5 million cells. Total RNA was isolated from the treated and control cells, then reverse transcription and real time polymerase chain reaction (RT PCR) were performed on each sample. SYBR Green assays were used for the relative quantification. The SOD₂ gene expression was compared to GAPDH housekeeping gene expression level (incubation with steroidal compounds mentioned above did not alter the expression of this gene in our pilot study).

Upregulated SOD₂ gene expression levels were found after treatment with each steroidal compounds. In case of estradiol 14.1fold, progesterone and cortisol 11.3fold increase in average was detected. The largest change (almost twenty –19.7fold rise) was caused by testosterone. The standard deviations of the ddCT values were within one in each treatment.

Based on these data the antioxidant effect of steroid endhormones might be caused at least in part by the enhancement of the SOD gene expression. These results may have innovative pharmacological importance in connection with free radical mediated disorders.

Metal elements, transmethylation ability and redox homeostasis in tumourous processes

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The receptors, kinases, and nuclear transcription factors affected by metals and metal-induced oxidative stress are associated with cancer growth and spreading. The formaldehyde is in connection with redox homeostasis. HCHO can be formed in transmethylation reactions. Data show the important role of HCHO in proliferative, as well as in apoptotic processes.

Therefore we were interested in studying erythrocyte metal element status, redox homeostasis and transmethylation ability of randomly chosen, operated, middle aged 68 colon and/or prostate tumourous patients and 46 healthy volunteers in both genders.

Tumour markers (CEA, CA 19-9, AFP, PSA) and routine laboratory parameters in sera, redox parameters (scavenger- and reducing ability, SOD, GSHPx) in plasma and erythrocytes, bounded HCHO, HbA1c, protoporphyrin and metal element concentrations in erythrocytes were measured.

We found significant differences in the metal and redox homeostasis between control and operated patients. Significant changes in erythrocyte function can be observed in transmethylation ability and protoporphyrin concentrations as well. The bounded HCHO concentrations were significantly lower in tumourous patients than in healthy controls.

These changes of erythrocytes were similar in operated colon and prostate tumourous patients. We hypothesize that there are similar changes in all other tumour types.

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