**REGULATION OF REDOX-HOMEOSTASIS BY METABOLIC DRUG ELTACIN IN THERAPY OF AGING PATIENTS WITH ISCHEMIC HEART DISEASE**

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*Objectives:* Intracellular reduction-oxidation imbalance, called oxidative stress, can subsequently contribute to the development and/or progression of cardiovascular diseases such as atherosclerosis, ischemia-reperfusion injury, chronic ischemic heart disease. The aim of this investigation was to determine the effect of metabolic drug eltacin contained amino acids (glutamate, cysteine, glycine) on cellular redox homeostasis state in old patients with ishemic heart disease.

*Methods*: The use of eltacin (220 mg x 3 times per day) in addition with traditional therapy (β- adrenoblockers, aspirin, Ca-antagonists, nitrates, diuretics) of aging patients (69 ± 2.7 years old) with ischemic heart disease, angina pectoris functional class II-III was estimated. Before and 21 days after the therapy ECG-monitoring, EchoCG data were examined. Activities of antioxidant enzymes, reduced (GSH) and oxidized (GSSG) glutathione maintenance in erythrocytes, malonyl dialdehyde (MDA) level in plasma have been tested.

*Results*: The use of eltacin in therapy of patients resulted in an increase of glutathione (GSH) maintenance, GSH/GSSG ratio and activity of GSH-related enzymes (glutathione peroxidase, glutathione transferase) as well as glutaredoxin and thioredoxin, Cu,Zn-superoxide dismutase, catalase in erythrocytes up to control values depressed until the treatment. The increase of antioxidant state of erythrocytes was accompanied by the decrease of lipid peroxidation and depression of ROS production. Extent of the development of antioxidant response was time-related and correlated with positive alteration of patient states: a rise of exercise tolerance, reduction of myocardial power consumption, antiarrhythmical effect.

*Conclusions*: It may be concluded that eltacin has ability to reduce oxidative stress by improving cellular redox state that give it perspective for the use in therapy of ischemic heart disease.