**ACTOVEGIN DECREASES THE ROS LEVEL IN BLOOD SAMPLES OF HEART FAILURE PATIENTS AND DIMINISHES NECROSIS OF SK-N-SH HUMAN NEUROBLASTOMA CELLS**

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Aim: To study the effects of Actovegin on ROS generated by blood phagocytes and isolated PMN of Heart Failure (HF) patients and necrosis of SK-N-SH cells induced by H2O2.
Material and methods: Using blood samples (BS) and isolated PMN of 17 patients with heart failure (II-III NYHA classes) the ROS generation were examined with "Biotox-7" chemiluminometer. Bacterial formyl-peptide (fMLP, 3 μM) and phorbol ester (PMA, 1 μM) were used as stimulators of phagocytes. The generation of O2−• were assessed as counts per second. SK-N-SH cell viability was determined by fluorescence microscopy using propidium iodide by means microscope (Keyence BZ8100, Japan). .
Results: In BS and PMN of HF patients "spontaneous" production of O2−• increased with time. PMA and fMLP increased the ROS production. The initial exposure of BS to actovegin (1-10 mg/ml) decreased spontaneous generation of ROS and subsequent response to fMLP or PMA. ROS generation, first induced by fMLP or PMA was decreased by actovegin. H2O2 have cytotoxic effects on neurons. Viability of SK-N-SH cells in the presence of H2O2 (100 μM) in vitro was studied. Adding of actovegin (1-10 mg/ml) led to decrease in neuron death.
Conclusion: These protective effects of actovegin may be due to decrease in ROS generation.