ABSTRACT

Name of thesis: Impairment of cellular metabolism as common pathophysiological mechanism of CNS diseases

Problem definition: Every human cell needs energy for living. If the production of ATP (as an universal energy carrier) is broken, cell restricts its activity first and during longterm depletion of ATP, dies. It was found, that cellular metabolism is broken in most pathologies in CNS. Disorder of respiratory chain by free radicals is the best known at Parkinson's disease, epilepsy, brain ischemia etc. Mitochondria, where respiratory chain is situated, is not only the aim of free radicals, but it is their major producer. The activity of respiratory chain decreases during the life and this phenomenan is called aging.

Aim of thesis: To determine whether there is increased production of free radicals in mitochondria of rat (LE Wistar) hippocampus during the epileptic seizure.

Method: Thesis involves experiment which was done with acute rat hippocampal slices. To induce epileptic seizure it was used 4-aminopyridine model. It was used fluorescence imaging as imaging method. Changes of superoxide production was detected with MitoSOX. Electrophysiological record was taken by programme Spike 2 with stimulation and recording electrode inside the slice.

Results: There was no significant difference between superoxide production with or without addition of 4-aminopyridine.

Key words: cellular metabolism, mitochondria, free radicals, CNS.