balance but also leads to depolarization of mitochondrial membrane and decreasing of mitochondrial potential. In our experiment, laser irradiation at 1265 nm causes a decrease of mitochondrial potential. In the presence of DIDS, mitochondrial potential demonstrates a decrease stronger than under laser irradiation without DIDS. A similar effect has been produced by the laser on mitochondrial mass. Surprisingly, in our experiments the concentrations of ROS and GSH increase simultaneously. In the presence of DIDS, these effects are substantially potentiated. This effect could be considered as a specific feature of mitochondrial metabolism in cancer cells that still requires its explanation. LLLR at 1265 nm causes a significant damage of mitochondrial DNA and has no effect on nuclear DNA. Similar results have been obtained for laser treatment with DIDS.

The experimental results have brought us to the conclusion that the 1265 nm laser irradiation affects intracellular processes through interaction with mitochondrial photoactive molecules. Inhibition of VDAC enhances the damage LLLR effect.

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Use of fluorescence spectroscopy for assessment of brain metabolism parameters in the rat model

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Modern optical technologies show considerable advantages in application for drug discovery in small laboratory animals during efficiency and toxicity trials. In this study, using the developed fibre-optic probe, the fluorescence of the anterior brain regions was evaluated and analysed in a Wistar rat model. The probe made it possible to simultaneously record both fluorescence intensity and blood content parameters of the studied tissue site. To modulate the metabolic activity of brain tissues, zinc compound solution (in the form of sulphate) of four different concentrations were administrated to the animals from treatment groups for 1 month as drinking water. After that, the fluorescence signals were measured on the open brain. Excitation at wavelengths of 365 nm, 450 nm, 532 nm and 637 nm was used to record the fluorescence signals. The processing of the obtained experimental spectra made it possible to reveal a direct relationship between the fluorescence intensity in the spectral region of NADH emission and the concentration of the orally administrated zinc. Optical measurements in vivo were supplemented by histochemical measurements of zinc ions in brain sections both in the treatment and control groups. The obtained results are in agreement with the statement that high concentrations of zinc ions are capable of inhibiting the mitochondrial complexes I, II and IV. The effect leads to inhibited cellular respiration in the cells of the nervous tissue.