



## **The 7th Congress of Biophysicists of Russia - conference proceedings**

### **Abstracts**

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assessment of tissue repair, the use of the multimodal optical coherence tomography method is proposed.

The purpose of this study was to search for optical criteria for the effectiveness of PDT and early recurrence of lichen sclerosus.

**Materials and methods.** Total 12 patients diagnosed with VLS and 10 patients without vulvar pathology were studied. PDT was performed with a medical laser "Lakhta-Milon" 662 nm at a dose of 0.16 W/cm<sup>2</sup> with intravenous administration of Photoditazine (0.7 mg/kg). The study was carried out using MM OCT developed at the Institute of Applied Physics Russian Academy of Sciences (Nizhny Novgorod). A 3.4 x 3.4 x 1.25 mm<sup>3</sup> 3D dataset was acquired within 26 s, from which tissue structure and microcirculation information was extracted. Dynamic observation was carried out before PDT, immediately after PDT, after 24 hours, 1-4 months and 6 months after PDT. Histological examination was performed before PDT and 4 months post PDT. Results. Multimodal OCT in VLS demonstrates a change in the scattering and polarization properties of the vulvar tissue due to atrophy of the epidermis, hyperkeratosis, edema, and formation of a sclerotic dermis, as well as a decrease in blood and lymphatic vessels relative to the norm. After PDT, a decrease in the density of the vascular network was observed, up to their complete disappearance after 24 hours. Starting from 1 month after PDT, the density of blood and lymphatic vessels gradually recovered, reaching a maximum level 2-3 months after PDT, but at the same time not reaching the density values characteristic of normal tissue. The restoration of scattering and polarization properties simultaneously with the restoration of the layered structure on OCT images was observed on the 2nd month after PDT. A biopsy at 4 months after PDT shows a normotrophic scar formation.

**Conclusions.** Multimodal OCT is a promising in vivo method for monitoring the dynamics of both vulvar vascular component and the connective tissue collagen fibers recovery. This may allow assessing the effectiveness of treatment and fixing the occurrence of a recurrence of VLS.

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#### S9.663. NO production intensity and rat myocardial contractility during hypokinesia

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Deficiency of movements in human life is an important medical and social problem caused by lifestyle, professional activities, prolonged bed rest, etc. There is a decrease in the load on the muscular apparatus, which leads to changes in functional and morphological changes to pathological conditions, depending on the duration and degree of hypokinesia.

Nitric oxide (NO) is an important biological mediator involved in many physiological and pathophysiological processes. The NO synthase system is widely represented in different heart structures. NO is able to exert both an activating and an inhibitory effect on various metabolic processes occurring in the body of mammals and humans. It has also

been shown that the NO system plays an important role in the adaptation of the body to various changes in the external environment and external conditions, for example, during significant physical exertion. Prolonged restriction of motor activity causes changes in contractile function and weakening of the heart muscle, as well as weakening of venous and arterial vessels.

An EPR study of the intensity of nitric oxide (NO) production was carried out in the simulation of movement deficit in rats by analyzing the amount of NO-containing paramagnetic complexes in the tissues of the heart. Also, a study was made of the force of contraction of the isolated heart of rats according to Langendorff. Experimental animals were in conditions of deficit of movements, starting from the age of 3 weeks: the first two days, the time of hypokinesia was 1 hour, and then increased by 2 hours every 2 days. By the 25th day of hypokinesia, the residence time of the animals in the cages reached 23 hours, and remained constant until the end of the experiment.

The amount of NO was estimated from the intensity of the characteristic EPR signal belonging to the (DETC)<sub>2</sub>-Fe<sup>2+</sup>-NO complex. The contractile (or inotropic) function of the heart was assessed by the pressure developed by the left ventricle using the Langendorff Power-Lab 8/35 device (ADInstruments, Australia) using the LabChart Pro program (Australia).

It has been established that the presence of rats in conditions of deficit of movements for 2 months leads to an increase in the content of NO in the tissues of the heart by 2 times. With a deficit of movements, a decrease in the force of contraction of the heart was recorded, which is possibly associated with a change in the level of NO. Since excessive formation of NO can significantly reduce the tone of smooth muscle cells, impair endothelial function and directly suppress myocardial contractility.

#### S9.664. Neuroprotective potential of intracellular acidification in a toxic model of Parkinson's disease

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Parkinson's disease (PD) is a progressive neurodegenerative disorder caused by the loss of midbrain dopaminergic neurons. The mechanism of neurodegeneration is associated with the accumulation of pathological protein aggregates, oxidative stress, and mitochondrial dysfunction, including impaired mitophagy. It is assumed that the activation of mitophagy avoids cell death and returns them to normal functioning. We have previously demonstrated that lactate and pyruvate are able to restore mitochondrial function by inducing mitophagy through a decrease in intracellular pH. The concentration of lactate in the body can increase dramatically during physical activity, while the brain absorbs lactate in proportion to the concentration in the arterial blood. Based on this, we hypothesized that physical exercise could induce mitophagy in midbrain neurons. In addition, an increase in the concentration of carbon dioxide (5-20%) in the inhaled air causes a reversible acidification of brain cells. Therefore, it may promote mitophagy. Thus, we hypothesized that forced inhalation of a CO<sub>2</sub>-rich gas-air mixture could activate mitophagy and promote neuronal survival.

In this study, we aimed to test two experimental therapeutic approaches: forced moderate physical activity and high CO<sub>2</sub> inhalation for the treatment of PD in a rodent model treated with the model toxicant rotenone. 12-month-old CD-1 mice were used in the experiments. Rotenone dissolved in olive oil was administered intraperitoneally 5 times a week for 6 weeks at a dose of 2 mg/kg of body weight. Treatment was carried out starting from the third week of toxic modeling. Moderate physical activity consisted of forced running on a treadmill; for forced inhalation, the animals were placed in a closed glass cylinder filled with 20%