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Dynamics of ultrastructural changes in the isolated neuron under photodynamic impact

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Photodynamic therapy (PDT) is a strong inducer of singlet oxygen and following oxidative stress. To explore the mechanism of photodynamic injury of neuronal cells we studied PDT effect of alumophthalocyanine Photosens (10^{-7} M) and laser diode light (670 nm, 0.4 W/cm^2) on a single mechanoreceptor neuron. After 5-min treatment that only slightly changed neuron activity, the initial injury (alteration of some mitochondria, vacuolization of the cytoplasm) was observed in parallel with compensatory changes (chromatin decondensation, elongation and aggregation of mitochondria, formation of lysosomes and autophagosomes). Longer photosensitization (30 min) abolished firing, destroyed mitochondria and Golgi apparatus, depleted energy sources (glycogen granules), impaired granular endoplasmic reticulum and polysomes involved in protein synthesis. Therefore, mitochondria and Golgi apparatus were the primary targets for Photosens-mediated PDT in a single neuron. Their alteration might underlie functional shifts. These structural changes continued to develop after loss of neuronal activity and led to necrosis.

The work was supported by RFBR grants 05-04-48440, 05 – 04 – 96754 and 08-04-01322.