Laserové centrum HiLASE Vás zve na seminář v anglickém jazyce:

Photoactivation of Mitochondria by High Fluence Low-Power Laser Irradiation

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Low-power laser irradiation can cause cell proliferation, differentiation, or death; however, the cellular mechanisms of these effects of low-power laser irradiation, at high or low fluences, are not well known. Moreover, deep understanding of the molecular mechanisms behind laser-induced cellular effects remains a significant challenge. Here, we investigated mechanisms involved in the death process in human hepatic cell line Huh7 at a laser irradiation. We decoupled distinct cell death pathways targeted by laser irradiations of different fluences. Out data demonstrate that high dose laser irradiation exhibited the highest levels of total reactive oxygen species production leading to cyclophilin D-related necrosis via the mitochondrial permeability transition. On the contrary, low dose laser irradiation resulted in nuclear accumulation of superoxide and apoptosis execution. Our findings offer a novel insight into laser-induced cellular responses, and reveal distinct cell death pathways triggered by laser irradiation. The observed link between mitochondria depolarization and triggering ROS could be a fundamental phenomenon in laser-induced cellular responses. Furthermore, we showed that ROS scavenger N-acetyl-L-cysteine reduced cytotoxicity caused by ROS production induced by laser irradiation indicating that dietary supplementation with antioxidants might be a suitable strategy to reduce oxidative damage. Hence, these results imply that the cytotoxic effects of red laser light require more intensive study, which should be considered when such lasers are intended for use in biomedical applications.

Kdy: v pondělí, **16. 04. 2018 od 14:00**

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