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Live-cell imaging study of mitochondrial morphology in mammalian cells exposed to X-rays

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Recent reports suggest that extranuclear targets in cytoplasm may have a role in mediating radiation effects in mammalian cells exposed to ionizing radiation (Tartier et al., 2007, Maeda et al., 2010). Mitochondria, a kind of major organelles, are distributed throughout cytoplasm. They contain their own genome, and mediate essential cell functions, such as generation of ATP and regulation of cell death. Mitochondria generate reactive oxygen species (ROS) as by-product of respiration for ATP production. If cells fail to reduce ROS, it might give the cells oxidative stress. In addition, dysfunctions of mitochondria have been known to be involved in a wide variety of diseases. Radiation effect on mitochondrial functions, however, remains to be fully elucidated. As the first step to understand the cytoplasmic effects of radiation, we have examined mitochondrial morphology in mammalian cells exposed to X-rays.
Mitochondria are continuously fusing or dividing during mitosis, or in response to environmental condition changes. It is also known that mitochondrial morphology dynamically change with cell cycle progression. In this study, after irradiation of X-rays (150kVp) to human or mouse cells, we labeled mitochondria by Mitotracker Red and analyzed kinetics of mitochondrial morphology by a live-cell imaging technique using a fluorescence microscope (KEYENCE, BZ-9000) equipped with a time lapse imaging system. Cell cycle stages were identified by simultaneously staining cell nuclei with Hoechst33342. Mitochondrial images were captured every 15 or 30 minutes for 6 days after irradiation. We report the relation between the morphological change of mitochondria and radiation induced cell cycle arrest.

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