5 - 65 Study on the Function of CPT1A Transporter in Radiation-induced Fatty Acids Entry into Mitochondria

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After experiencing severe radiation damage, tumor cells must overproduce energy to counteract radiation stress. Fatty acid oxidation as the most highly efficient way of energy supply, fatty acids are oxidized in mitochondria to release energy for tumor cells to utilize[1]. However, the mechanism of radiation-induced fatty acids transport is unknown. Carnitine palmitoyl transterase 1A (CPT1A) is a key protein in the transport of fatty acids to mitochondria. Further studies showed that X-ray upregulates CPT1A expression in pancreatic cancer cells PANC-1 and SW1990 cells, while knockdown of CPT1A no longer causes changes in CPT1A expression after radiation (Fig. 1(a)). As shown in Fig. 1(b), using Mito-tracker labeled mitochondria and BODIPY C12 labeled fatty acids, it was found that radiation significantly increased the co-localization of mitochondria with fatty acids, while knockdown of CPT1A weakened the co-localization and fatty acids aggregated outside of mitochondria, indicating that the CPT1A transporter is a key protein for X-ray induced fatty acids transport to mitochondria. Our study has illustrated the mechanism by which CPT1A regulates the transport of fatty acids to mitochondria after X-ray irradiation, which has positive implications for gaining insight into the energy metabolism of tumors.

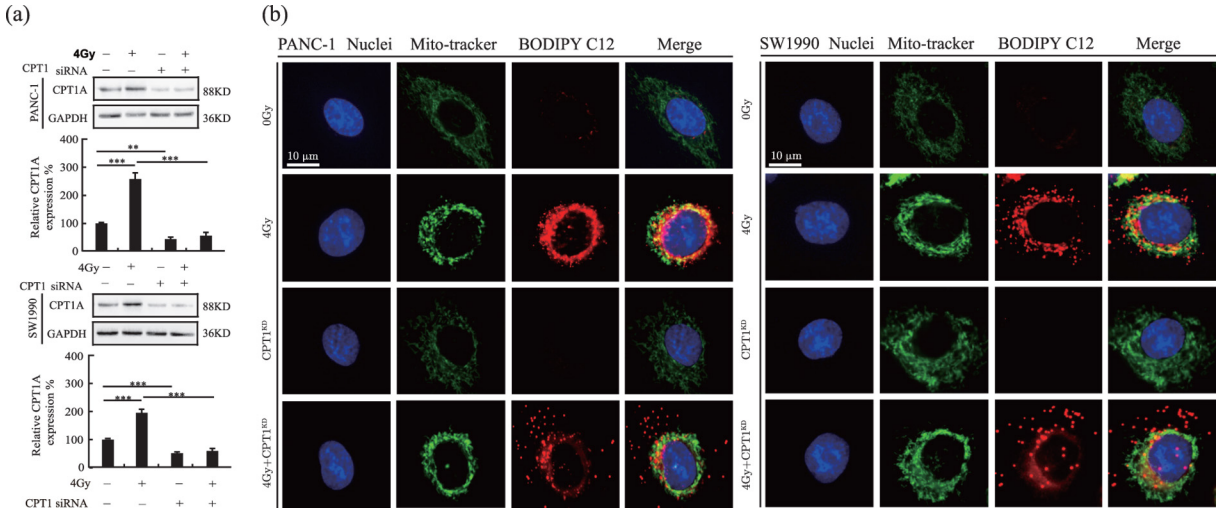

Fig. 1 (color online) X-ray induces fatty acids entry into mitochondria via CPT1A transporter. (a) The protein expression of CPT1A in PANC-1 and SW1990 cells after knockdown of CPT1A by X-ray irradiation, (b) Co-localization of mitochondria and fatty acids after combined X-ray irradiation with knockdown of CPT1A in PANC-1 and SW1990 cells, Hoechst 33342 (blue) for the nucleiMito-tracker (green) for mitochondria and BODIPY C12 (red) for fatty acids.

Reference

[1] I. R. Schlaepfer, M. Joshi, Endocrinology, 161, 2(2020)bqz046.