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Inhibitory Effect of Proton Beam Irradiation on Metastatic Cancer Metabolism of Human Colorectal Adenocarcinoma Cells

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Proton beam therapy has recently been used to improve local control of tumor growth and reduce side effects by decreasing the global dose to normal tissue. However, the regulatory mechanisms underlying the physiological role of proton beam radiation are not well understood, and many studies are still being conducted regarding these mechanisms. To determine the effects of proton beams on mitochondrial biogenesis, we investigated: mitochondrial DNA (mtDNA) mass; the gene expression of mitochondrial transcription factors, functional regulators, and dynamic-related regulators; and the phosphorylation of the signaling molecules that participate in mitochondrial biogenesis. Both the mitochondrial DNA/nuclear DNA ratio and the mitochondria staining assays showed that proton beam irradiation increases mitochondrial biogenesis in 12-O-tetradecanoylphorbol-13-acetate-induced aggressive HT-29 cells. Simultaneously, proton beam irradiation increases the gene expression of the mitochondrial transcription factors PGC-1α, NRF1, ERRα, and mtTFA, the dynamic regulators DRP1, OPA1, TIMM44, and TOM40, and the functional regulators CytC, ATP5B, and CPT1-α. Furthermore, proton beam irradiation increases the phosphorylation of AMPK, an important molecule involved in mitochondrial biogenesis that is an energy sensor and is regulated by the AMP/ATP ratio. Based on these findings, we suggest that proton beam irradiation inhibits metastatic potential by increasing mitochondrial biogenesis and function in TPA-induced human colorectal adenocarcinoma cells.

Paper submission Plan

No

Best Presentation

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Contribution track

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