3 - 65 Influence of Dithiothreitol on HeLa Cells Apoptosis Induced by Carbon-ion Irradiation

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Dithiothreitol is a strong reducing agent. It can restore the protein disulfide bond and cause the accumulation of misfolded proteins and induce endoplasmic ER Stress. In response to ER stress, cells activate a set of tightly controlled regulatory programs to restore the normal function of the ER. If the protective mechanisms are not sufficient to restore normal ER function, apoptosis will occur to remove the stressed cells[1]. A variety of anticancer therapies have been linked to the induction of ER stress in cancer cells, suggesting that strategies devised to stimulate its prodeath function or block its prosurvival function, could improve their antitumor effect.

In order to study the influence of dithiothreitol on HeLa cells apoptosis induced by carbon-ion irradiation, the cell samples were divided into two groups, one group samples were exposed to carbon ions (LET was 20 keV/μm) at the dose range of 0.5 to 5 Gy directly, the other samples were treated with 2.5 mM dithiothreitol 3 h and then exposed to carbon ions with the same dose. After irradiation, all samples were cultured normally for 24 h, and then replaced the medium with new medium without dithiothreitol and culture for another 24 h. Apoptosis was analyzed by acridine orange/ethidium bromide staining.

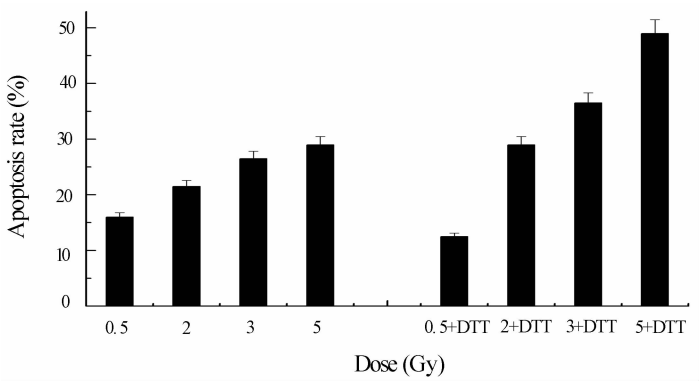

Fig. 1 Hela cells apoptosis induced by carbon-ion radiation combined dithiothreitol treatment or by carbon-ion radiation alone.

The results were showed in Fig. 1. HeLa cells apoptosis rates in both group samples increased with the radiation dose increasing from 0.5 to 5 Gy, and they were lower in carbon-ion radiation combined dithiothreitol treatment effects group than those in carbon-ion radiation alone.

The experiment shows that 2.5 mM dithiothreitol can increase the radiation resistance, and it may influence HeLa cells radiosensitivity through apoptosis pathway.

Reference

[1] D. J. Todd, A. H. Lee and L. H. Glimcher, Nature Reviews Immunology. 8, 9(2008)663.