3 - 69 Effects of Carbon-ion Beam Irradiation on MSH2 Expression in HepG2 Cells*

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Primary liver cancer (namely hepatocellular carcinoma, HCC) represents the third most frequent cause of cancer death. National Comprehensive Cancer Network guidelines suggest that radiotherapy (RT) can be considered as an important role for unresectable HCC[1]. Heavy ion radiotherapy (*e.g.* carbon ion) has showed a strong role for killing malignancies especially for radioresistant malignancies that are insensitive to conventional radiotherapy, such as X-ray.

The mismatch repair (MMR) is a highly conserved DNA repair pathway that recognizes and repairs base-pairing errors during DNA replication or recombination. When the genes (mainly including *MutS* and *MutL* families) that mediate MMR are mutated or epigenetically silenced, the predisposition to cancer is significantly increased. MMR deficiency can be inherited, as in the case of Lynch syndrome also known as hereditary non-polyposis colorectal carcinoma (HNPCC). MSH2 protein also can modulate the homologous recombination that is a prominent double-strand break (DSB) repair pathway.

Although the role of DSB repair pathways in the survival response to IR has been extensively studied, DNA mismatch repair (MMR) has received less attention. The role of this pathway has been largely studied in the DNA damage response to chemotherapeutic agents, but the activation of the MMR system after IR remains controversial. And so far little information is available on the relationship between $MSH2$ and high-LET radiation.

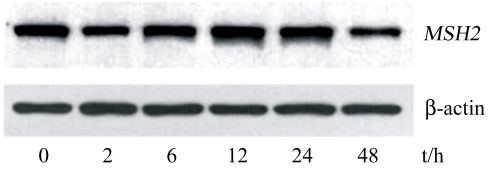

Fig. 1 (color online) Western blot detected $MSH2$ protein expression at different times of heavy ion irradiation.

We used western blot and RT-PCR assay to analyze $MSH2$ expression in human HepG2 carcinoma cells after low or high-LET radiations (Fig. 1). The $MSH2$ gene activated by carbon ion irradiation suggests that DNA mismatch repair gene $MSH2$ should be involved in DNA repair pathways.

Reference

[1] I. J. Lee, J. W. Kim, K. H. Han, et al., Yonsei Med. J, 55(2014)1489.

3 - 70 Capsazepine, a TRPV1 Antagonist, Enhances Radiation Sensitivity in Human Hepatocellular Carcinoma HepG2 Cells*

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Transient receptor potential vanilloid 1(TRPVl) that is known as capsaicin receptor is a non-selective cation ion channel[1]. TRPV1 can regulate Ca^{2+} influx, participate in a variety of physiological and pathological process of tumor[2]. One such agent is Capsazepine (CPZ) that is now widely used as a selective vanilloid type 1 receptor (TRPV1) antagonist. CPZ can be directly actived on the capsaicin receptor, blocked its biological effects, and abolished osteosarcoma-induced hyperalgesia when administered subcutaneously at doses ranging from 3 to 10 mg/kg, blocked calcium channels[3]. However, the mechanisms underlying the anticancer effects of CPZ have not fully been understood. Whether CPZ can induce apoptosis of human hepatocellular carcinoma cell line HepG2 is not known. Therefore, the objective of the study reported here was to determine whether CPZ can enhance radiation sensitivity in HepG2 cell, and impact on cell proliferation.

* Foundation item: Key Program of National Natural Science Foundation of China (U1432248), National Natural Science Foundation of China (11175222, 11205219) , Western Talent Program of Chinese Academy of Sciences (Y260230XB0).