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Research article

A12B4C3, an inhibitor of polynucleotide kinase/phosphatase enhances radio-sensitivity in PC-3 cells exposed to carbon ion beam

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Abstract

Cancer recurrence is a major problem of current radiotherapies like gamma radiation or High LET (Linear Energy Transfer) radiation. This problem can be overcome by the use of radio-modulator which will enhance cell killing and/or prevent cell regeneration after the treatment. Polynucleotide kinase / phosphatase (PNKP) plays an important role in DNA repair pathway. Accordingly, its inhibition can hamper the DNA damage repair processes and ultimately can suppress the recurrence of cancer. In this context, we have reported that following 24 hr ^{12}Carbon ion beam combined with PNKPi (PNKP inhibitor) as radio-modulator enhanced the radio-sensitivity. Present study will elucidate the effect after 48 hr of ^{12}Carbon ion beam in combination with PNKPi. We have checked the PC-3 cell viability through MTT assay, cell death mode by DAPI staining and cell cycle analysis through flow cytometry after 48 hr of irradiation (^{12}Carbon ion beam having energy 62 MeV; equivalent to 5.16 MeV/nucleon with entrance LET 287 kev/μm) and in combination with PNKPi as radiomodulator. It was observed that PC-3 cell viability decreased following apoptosis when carbon ion beam was combined with PNKPi. Further, the cell cycle arrest was enhanced at S phase in combined treatment group compared to only irradiated cells. These findings suggest that, after 48 hr of carbon ion irradiation PNKP inhibition could enhance cellular radiosensitivity in prostate cancer cell line PC-3 by suppressing DNA damage repair pathway. The synergistic effect of PNKPi and carbon ion irradiation may be a promising method to avoid recurrence of cancerous cells following High LET carbon-ion radiation therapy.