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Effect of irradiation and DNA synthesis and repair inhibitors treatment on DSBs formation and repair in normal and cancer cells

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The immunocytochemical staining and fluorescence microscopy was used to investigate the effects of radiosensitization by the cytosine arabinoside AraC on the induction and repair of DNA double-strand breaks (DSBs) in normal human dermal fibroblasts and human glioblastoma U87. Cells were irradiated with 1.25 Gy of Bragg peak protons (LET = 2 –25 keV/μm) and accelerated nitrogen ions with doses of 0.57 Gy (LET = 81 keV/μm) and 1.25 Gy (LET = 180 keV/μm). The most pronounced modifying effect of the AraC inhibitor on human fibroblast and glioblastoma cells was observed after proton irradiation. By contrast, the action of accelerated 15N ions with high LET value reduces the radiosensitizing effect of AraC in human fibroblast and glioblastoma cells. These results may reflect the changes in ratio of different types of induced DNA damage: direct and enzymatic double-strand breaks. Under the action of radiation with increasing LET values, the frequency of direct DNA DSBs formation increases and the yield of DNA single-strand breaks and modified bases decreases, which are the main substrates for the formation of enzymatic DNA DSBs under the influence of AraC.

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