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## Effect of repair inhibitors on the formation DNA double-strand breaks under the action of ionizing radiation with different physical characteristics.

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Nucleoside analogues are a class of chemotherapeutic drugs that are used to increase tumor sensitivity to ionizing radiation (IR). In this work, we study the effect of the arabinoside-cytosine inhibitor AraC on the induction and elimination of DNA double-strand breaks (DSB) under the action of IR with different physical characteristics. Normal human fibroblasts and human tumor cells (glioblastoma U87) were irradiated with accelerated 15N ions (LET = 181 keV/ $\mu$ m) and extended Bragg's peak protons (LET = 2–100 keV/ $\mu$ m) at a dose of 1.25 Gy under the action of the repair inhibitor AraC. Radiation-induced colocalized yH2AX/53BP1 foci of proteins involved in DNA DSB repair were visualized by immunocytochemical staining. A quantitative analysis of the obtained images showed that the most pronounced effect of AraC exposure is observed in both types of cell cultures under proton irradiation at the Bragg's peak. The output of damage in fibroblasts increases 5 times compared to the control level, and in glioblastoma cells, the increase occurs more than 2 times. A decrease in the efficiency of DNA DSB repair under the action of accelerated 15N ions in the presence of the inhibitor was found in U87 glioblastoma cells.

Summary

**Presenter:** SHAMINA, Daria (Dmitrievna) **Session Classification:** Sectional talks