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Induction and repair of DNA double strand breaks in melanoma B16 cells in the presence of repair inhibitors under the action of X-rays in vitro

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The search for new approaches aimed to change the radiosensitivity of cells is one of the priority tasks of radiobiology. Promising studies are of agents' modifying effect that affect the DNA repair associated both with the transformation of long-term unrepaired DNA single-strand breaks into enzymatic double-strand breaks (DSB) - 1-β-D-arabinofuranosylcytosine (AraC), and inhibition of enzymes involved in non-homologous end joining - SCR7 pyrazine (SCR7). This is a new approach based on the mechanisms that lead to the formation of additional lethal DNA damage and can also lead to increase the effectiveness of ionizing radiation. DNA comet assay method was used for comparative analysis of the induction and repair of DNA DSB in melanoma B16 cells under the action of X-rays under the influence of repair inhibitors AraC and SCR7. The dose rate was about 1.6 Gy/min. With the help of this method, the dose dependences of DNA DSB formation were obtained. It was found that under the combined action of AraC/SCR7, the maximum amount of DNA DSBs is formed, exceeding the amount of DNA DSBs in the control level by 1.6 times, and 1.15 times under the influence of AraC. The repair kinetics of DNA DSB was studied under the influence of radio modifiers up to 24 hours of post-radiation incubation. It was shown that the repair kinetics is complex. In contrast with the control, the DNA DSB yield increases up to 6 hours post-irradiation, and then decreases. Moreover, under the influence of the combined action of radiomodifiers the number of unrepaired lesions exceeds the control level by 10.3 times and by 1.76 times under the action of AraC alone.

Summary

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