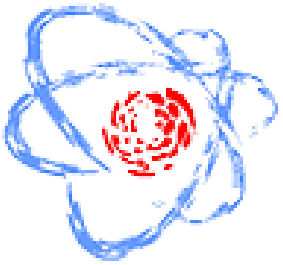

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The repair inhibitors effect on DNA damage in mice after exposure to 60Co γ-rays

Monday 6 June 2022 13:20 (10 minutes)

Research devoted to the study of modifying agents that increase the yield of DNA damage in the cells of the central nervous system appear to be promising. We have previously shown that under the influence of inhibitors of DNA repair - 1-β-D-arabinofuranosyl cytosine (AraC) and hydroxyurea (HU), the yield of DNA double-strand breaks (DSB) increases upon γ-irradiation of cells of various types in vitro. The mechanism of this process is based on the transformation of long-term non-repairable single-strand breaks (SSB) of DNA into enzymatic DSB. In this case, the magnitude of the modifying effect of these agents depends on the quality of the radiation. In this work, we performed a comparative analysis of the induction and repair of DNA SSB and DSB in neuronal cells of mice (hippocampus and cerebellum) under γ-irradiation after intraperitoneal administration of AraC and AraC/HU combination in vivo. DNA comet assay method was used to study the regularities in the formation of DNA damage in cells from hippocampus and cerebellum of mice after exposure to γ-rays in vivo. It was found that for all types of cells used, there is a linear character in the yield of DNA lesions. It has been shown that the amount of DNA SSBs and DSBs formed during irradiation under the influence of AraC significantly increases. An additional increase in the yield of DNA SSBs and DSBs is observed under the combined action of AraC and HU. In addition, the repair kinetics of DNA damage under the influence of DNA repair inhibitors was studied. It has been shown that the kinetics of DNA SSB and DSB repair both in hippocampal and cerebellar cells is complex. Under the conditions of the influence of AraC alone and the combined influence of AraC/HU, an increase in the total amount of damage is observed during the entire post-irradiation period observed in the experiment.

Summary

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