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γ-ray induction and repair of DNA double-strand breaks in rat hippocampus and cerebellum cells.

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Radiation protection both on Earth and in space is based on the principles of risk justification and limitation. The contemporary concept of the radiation risk of manned interplanetary flights incorporates the influence of space radiation on the CNS functions. It is expected that possible space radiation risks for the CNS, like altered cognitive functions (including in short-term memory disorders), reduced motor functions, and behavioral changes are connected with damage on the molecular and cellular levels. These CNS changes can also originate in the radiation-induced DNA damage arising in neuronal and non-neuronal cells of specific brain areas. DNA double-strand break (DSB) is the most harmful damage for the cell. To protect the DNA molecule from such changes there are mechanisms of DNA repair which maintain genome integrity in an intact state. Investigation of DNA DSB induction and repair is of special interest in cells of the hippocampus and cerebellum. The hippocampus is the brain area performs the function of the production of new neurons by neurogenesis and has been associated with radiation-induced cognitive decline in spatial learning and memory. The cerebellum is involved in the coordination of voluntary motor movement and contains roughly half of the brain's neurons. For the analysis of the induction and repair of DNA DSBs, Spraque Dawley rat heads were irradiated with 1, 3, and 5 Gy 60Co γ-rays. DNA DSBs were visualized by immunohistochemical staining of paraffin-embedded rat brain tissues. Tissue slices were processed by the standard procedure of immunostaining with fluorescent antibodies for the phosphorylated histone H2AX (γ-H2AX) and repair protein 53BP1 –DSBs markers. Quantification of colocalized γH2AX/53BP1 foci allows evaluation of the quantity and quality of DNA DSB and their repair in hippocampus and cerebellum cells. Fluorescent images hippocampus sections with visualized γH2AX foci in were obtained.

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