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Detection of clustered DNA damage in mammalian neuronal cells induced by ionizing radiation with different physical characteristics

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Ionizing radiation with different physical characteristics induces a wide spectrum of DNA damage. The structural DNA damage, such as single-strand breaks (SSB), double-strand breaks (DSB) and AP sites (abasic sites missing either a pyrimidine or purine nucleotide), as well as cluster damage, which are the set of all these damage, is the main factor which could lead to the formation of various mutations, chromosome aberrations, and further cell death. In this regard, research of DNA damage induction and repair under the action of ionizing radiation is the most important goal in radiation genetics, radiotherapy and space radiobiology.

It is commonly known that a substantial amount of enzymes implicated in the identification, localization and damage repair formed during the action of various physical, chemical or biological factors is involved in the repair process. The use of DNA repair enzymes involved in the base excision repair, such as DNA endonuclease III (Endo III) and formamidopyrimidine glycosylase (FPG) expanded the spectrum of experimentally-detectable clustered DNA damage.

The total number of lesions in the DNA involving AP sites, DSBs and SSBs can be detected by using the enzymatic comet assay in neutral and alkaline conditions respectively. The induction and repair regularities of DNA damage (DSB, SSB, AP sites) in mammalian neuronal cells induced by ionizing radiation with different physical characteristics: 60Co γ-rays and accelerated 15N ions (LET = 85 keV/μm) were investigated. It was shown that the yield of DNA SSB and DSB increases in the presence of repair enzymes after exposure to both types of ionizing radiation. An amount increase of DNA DSB under the influence of EndoIII and FPG indicates the great contribution of modified bases to the cluster DNA damage formation. The quantity of modified purine bases (mPur, upon treatment with FPG) is slightly higher than the quantity of modified pyrimidine bases (mPyr, upon treatment with Endo III) in Sprague-Dawley rat hippocampal cells and in human U87 glioblastoma cells after exposure to accelerated 15N ions *in vitro*. These results can be explained by the fact that using of DNA repair enzymes (Endo III and FPG) which convert damaged bases and AP sites into DNA lesions allow us to identify that the number of arising DNA AP sites is large enough after exposure to different types of ionizing radiation.

The effect of DNA synthesis inhibitors – cytosine arabinoside (AraC) under the normal conditions and after repair enzymes treatment on the cluster DNA damage in rat hippocampal cells after exposure to 60Co γ-rays *in vivo* was studied. It has been shown that under normal conditions the number of DNA damage increases up until 4 h of and after that the processes of DNA damage repair were complete within 24 h of post-radiation incubation. In the presence of AraC an increase in the total amount of DNA damage is observed during the entire post-radiation incubation. In this case, the maximum yield of DNA SSB is shifted by 1 h of post-radiation incubation.

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