MODELLING OF LOW-DOSE HYPER-RADIOSENSITIVITY

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One of the most important aims of radiation protection research is to improve the determination of health risks caused by low-dose ionising radiations (<100 mGy), since the most common exposures fall into this range. In vitro studies might help us understand the impact of these radiations at the level of the organism. It was found that followed by low-dose irradiation the survival fraction of cells first decreased sharply and after reaching a local minimum started to increase, i.e. the number of cells capable of proliferation increased by radiation dose. Thus, overall a larger number of cells died after low-dose irradiation than would have been expected based on the extrapolation from high doses. This phenomenon is called low-dose hyper-radiosensitivity (HRS). According to our hypothesis, HRS is a result of a regulatory process aiming at the long-term survival of the organism by destroying damaged cells, thus minimising the level of DNA damage and cancer risk. In order to test our hypothesis, simulations have been implemented on a mathematical model, in which mutagenic damage results from ionising radiation and cell division. The damage caused by ionising radiation is proportional to the irradiation dose and follows Poisson distribution, while the spontaneous mutation rate related to division is constant. Cells with the highest level of damage are eliminated, and their places are occupied by successors of cells with the lowest level of damage, while the total number of cells remains constant. If the strategy is the minimisation of the total level of damage, the simulations provide survival curves which show hyper-radiosensitivity similarly to experimental data.

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