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Radiation-induced adaptive response in fruit flies with mutations in DNA repair genes

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Low doses of ionizing radiation induce a wide range of biological effects including hormesis, adaptive response and hypersensitivity that modulate lifespan. However, the molecular mechanisms underlying the lifespan effects of low-dose irradiation are poorly understood. DNA repair is one of the key mechanisms of cell response to different types of stress including ionizing radiation. The purpose of this work is to investigate the role of genes of base and nucleotide excision repair (PCNA, XPC, XPF, D-Gadd45 homologues) and genes of DNA double-strand breaks repair (BLM, Rad50, Rad51, Rad54 homologues) in the radiation-induced adaptive response in Drosophila melanogaster. The lifespan of flies with Canton-S wild-type and w1118 genotypes and flies with mutations in DNA repair genes were estimated under different irradiation conditions: 1) without irradiation; 2) chronic exposure at 40 cGy dose rate induced by Ra226 source during preimaginal developmental stages (10 days); 3) acute exposure at 30 Gy dose rate induced by Co60 source after imago eclosion (30 minutes); 4) successive irradiation by both doses. It was found that the chronic low-dose irradiation induced the radioadaptive response to the acute exposure in Canton-S wild-type flies. The negative effect of the acute irradiation on the lifespan declined 2 times after pre-irradiation with low-doses. Radioadaptive response was persisted but was less expressed in flies with heterozygous mutations in PCNA, XPC, Rad51, Rad54 homologues, and was absented in flies with homozygous and heterozygous mutations in XPF, D-Gadd45, BLM homologues. Obtained results demonstrate that investigated DNA repair genes play a significant role in radiation-induced adaptive response at an organism level in vivo.

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