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Age-related alterations in stress-response gene expression induced by chronic low-dose gamma-irradiation in Drosophila melanogaster

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The purpose of this research is to investigate the role of genes of DNA breaks recognition (ATM, ATR homologues), base and nucleotide excision repair (PCNA, XPC, XPF, Gadd45 homologues) and double-strand breaks repair (BLM, Rad50, Rad51, Rad54 homologues), and apoptosis (p53, Gadd45) in formation of specific long-term effects of chronic low-dose gamma-radiation in Drosophila melanogaster. The relative expression levels were estimated in imago of Canton-S wild-type flies divided into two groups: 1) without irradiation; 2) chronically exposed by gamma-radiation at 40 cGy dose rate induced by Ra226 source during preimaginal developmental stages (10 days). The levels of expression of ATM, ATR, PCNA, XPC, XPF, BLM, Rad50, Rad51, Rad54 homologues (but not p53 and Gadd45) were 1.5-2.6-fold increased after chronic low-dose irradiation. The increase of stress-response gene expression is a possible source of cellular and organism resistance mechanisms activation via more efficient functional recognition and utilization DNA damages, one of the factors of biological effects formation under low-dose gamma-radiation exposure such as hormesis and radiation-induced response. The effect was persisted in ATM, PCNA, XPF, BLM genes during Drosophila lifetime. It is possible that higher expression levels of these genes provide the long-term effects of chronic irradiation.

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