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Caenorhabditis Elegans as a Model of Radio adaptive Response and Radiation-Induced Bystander Effect

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Abbreviations: RIBE: Radiation Induced Bystander Effect; DDR: DNA Damage Response; ROS: Reactive Oxygen Species; NHEJ: Non Homologous End Joining

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Radio adaptive response and radiation-induced bystander effect (RIBE) are two contradictory biological effects induced by low-doses radiation. Radio adaptive response refers to the resistance to the subsequent damage induced by relatively high-dose radiation after the experimental subjects accept pretreatment with low-dose radiation. RIBE refers to the phenomenon that non-irradiated cells or tissues present the biological response like that in the irradiated cells due to the factors released by irradiated cells or tissues. *Caenorhabditis Elegans* (*C.Elegans*) is an excellent in vivo model to study those two effects because of its small body size, transparent body that allows to be well observed and allows for precise microbeam irradiation, abundant mutant as well as high radio sensitivity of germ cells.

Sugimoto T, et al. first found that DNA damage-induced cell cycle arrest and apoptosis were observed in locally microbeam irradiated regions as well as in the gonad [1]. When exposed to 50 or 70 protons site specific microbeam irradiation, a strong stress response in the posterior intestine was observed in the posterior intestine after 24 hours [2]. After quantitative irradiation of the posterior pharynx bulb and the vulva of *C.Elegans* by the carbon ion source or the single particle microbeam, non-targeted radiation stimulated the cellular DNA damage and the resulting apoptosis in the distant germ line. Different tissues exhibited different sensitivities to radiation. Irradiation to vulva, rather than posterior pharynx bulb, induce more serious injury. There is a system-specific radiation sensitivity in the RIBE induction and more severe damage and genomic instability were observed in intra-system RIBE. More important, RIBE can transmit the damage to the offspring and increase the instability of the progeny genes. However, DNA damage response (DDR) pathway is involved in both intra- and inter-system RIBE. Irradiation induced the production of reactive oxygen species (ROS) in the radiation site, then, ROS delivered all over the body and activate the DDR pathway in the distant sites, especially the germ cells. After that, the cells of failure to repair damaged timely or mutation will be removed by apoptosis pathways to maintain the stability of gene [3]. But HUS-1 and MRT-2, damage checkpoint proteins, function in inter-system RIBE while not in intra-system RIBE. Besides, the deficiency of ceramide synthase could inhibit the apoptosis induced by RIBE, which indicated that ceramide biosynthesis pathway may be involved in this process[4], but the specific mechanism is unknown.

After irradiation, *C. Elegans* somatic cells increased the level of DNA damage in by stand germ cells through MAPK signaling pathways including ERK, JNK and p38 MAPK, leading to apoptosis of non-radiated germ cells. Multiple parallel signaling pathways, such as CEP-1/p53 dependent signaling pathways, may also involve in the induction of apoptosis by RIBE [5]. After exposure to ultraviolet radiation or ionizing radiation, CPR-4, a human cathepsin B lysosomal protease homologue, is the major RIBE factor, whether intra- or inter-animal RIBE. Irradiation activates the p53 homologue *cep-1*, which increases the transcription of cpr-4, leading to synthesis of more *CPR-4* proteins and increasing secretion of CPR-4, the latter appears to act through DAF-2, insulin-like growth factor receptor, and its downstream signal complex PDK-1 kinase, but not transcription factor DAF-16,to exert RIBE [6].

Compared with mammals, *C. Elegans* need a higher dose of radiation to induce radio adaptive response, 5 Gy with gamma-ray radiation, which does not harm to the growth of *C. Elegans*. The radio adaptive response of C. elegans germ cells induced by low doses of radiation increase the radio resistance of germ

cells, instead of affecting cell cycle [7]. With an adaptive gamma-radiation, it took *C. Elegans* vulva cells 1.75 h to develop radio resistance, and this resistance may Persis for only 2-4 h. DNA damage checkpoints and non-homologous end-joining (NHEJ) were involved in radio adaptive response in a cellular non-autonomous manner. NHEJ pathway, one of DNA damage repair pathway, plays an important role inradioadaptiveresponse, while HR pathway, another DNA damage repair pathway, were involved in carbon ion source radiation-induced DNA damage, but not in radio adaptive response [7,8]. ATM-1, but not HUS-1, were involved in germ lines radio adaptive response induced by microbeam [7]. Radio adaptive response and RIBE are the hot top of recent studies, but the specific mechanism needs to be further clarified. However, there is a certain relationship between radio adaptive response and RIBE. Bystander cells activate DNA damage checkpoints by RIBE, then produce radio adaptive response to subsequent high-dose irradiation [9].

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