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Radiation Fields, Effects and Risks in Human Space Missions (5)

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INTERPRETATION OF RADIATION-INDUCED CELLULAR PREMATURE SENESCENCE

Abstract

Radiation can induce lethality, mutation and premature senescence in cells. Identification of these detrimental effects and their underlying mechanisims are very important for the risk estimation during the long-term manned flight in space. Cellular senescence was involved in aging by irreversible loss of proliferative potential. It causes inhibition of cell growth and reduction of cellular function. The deepening interpretation of premature senescence following exposure to ionizing radiation will provide useful data for the risk estimation of radiation in space. Here, we found that both X-rays and carbon ions could induce premature senescence in a human uveal melanoma cell line, 92-1 cells. The results of senescence associated-β-galactosidase (SA-β-gal) positive cells showed that carbon ions were more efficient to induce premature senescence than X-rays under the same dose (3Gy), implying the high relative biological effectiveness of heavy ions. Furthermore, we revealed that the un-repairable DNA damage including double-strand-breaks, single-strand-breaks and base damage contributed to cellular premature senescence detecting by immunofluorescence hybrid technology. We also found that premature senescence was related to the long-term G2-arrest cells induced by radiation. Besides senescent diploid cells, significant proportion of senescent tretraploid cells was observed after irradiation. Since the genes essential for G2-M transition such as Cyclin B1Plk1 and Aurora were prematurely downregulated at both transcriptional and translational levels, it was deduced that the long-term G2-arrested cells slipped into G1 phase directly, and underwent premature senescence along with the long-term G1-arrested cells together. These findings not only give new insights into the link of DNA damage and aging, but also enrich the knowledge of traditional radiobiology.