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133 The Effects of Inhibitors of Intracellular Signal Transduction on Radio-adaptive Response Induced by Low-dose X-irradiation

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To elucidate the induction mechanism of radio-adaptive response in normal cells, we examined the induction of radio-adaptive response with or without inhibitors of several kinds of protein kinase. The major results obtained were as follows; (1)Addition of either inhibitor of protein-tyrosine kinase or protein kinase C to the cell culture medium during the low-dose X-irradiation inhibited the induction of radio-adaptive response. However, the addition of inhibitor of cAMP-dependent protein kinase, cGMP-dependent protein kinase, or Ca^{2+}-calmodulin kinase II failed to inhibit the induction of radio-adaptive response. (2) These results suggest that the signal induced in cells by low-dose X-irradiation was transduced from protein-tyrosine kinase to protein kinase C via either pathway of phosphatidylinositol 3-kinase or splitting of profilin binding phosphatidylinositol 4,5-bisphosphate.

134 THE DECREASE OF POLYAMINE LEVELS IN THE HELA CELLS PRIOR TO THE DEPRESSION OF CELL GROWTH BY RADIATION

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The polyamines (putrescine, spermidine and spermine) regulate cell cycle progression by mediating DNA and protein synthesis. In addition polyamines protect, in part, cell components from damage resulting from oxidation. It is tempting to speculate that polyamines affect to fix the cellular damages. In this atudy we examined the relative polyamine levels in HeLa cells after x-ray irradiation, C-ion beam irradiation or H_2O_2 treatment.

The growth curve for irradiated cells is very similar to that for non-irradiated population until the end of second doubling. In the stage the putrescine level decreased for every treatment. The lower level of the putrescine might cause the following depression of proliferation. Interestingly, ODC mRNA increased at the decreased putrescine level.

135 Flowcytometric analysis of MUC1 mucin production on the cell surface in human carcinoma cell lines

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We have examined the effects of X-rays (200 kVp) and carbon ion beam (^{12}C) produced at HIMAC (290 MeV/u, 80 keV/mm, SOBP, 3 Gy/min) on the MUC1 in human carcinoma cell lines Capan I (pancreatic), MKN-45 (gastric), HT29 (colon). Flowcytometric analysis using an anti-MUC1 monoclonal antibody, M.Y.1E12 showed that the level of cell surface MUC1 mucin was significantly increased in HT29 at 4 days in a dose dependent manner after irradiation with X-rays or ^{12}C. However, no significant change was seen in Capan I and MKN-45 that produce MUC1 constitutively. These results indicate that MUC1 mucin is a radiation-inducible glycoprotein in HT29, and that the induction of this mucin may be refrect radiation damage.

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