

Radio-response and signal transduction (131-146)

131 The effect of adaptive response on radiation-induced genetic instability
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There is accumulated evidence that genetic instability may play an important role during radiation-induced carcinogenesis. To study the mechanism of radiation-induced genetic instability, we investigated delayed reproductive death, chromosome aberrations and mutation frequency in surviving colonies of mouse m5S cells after X-irradiation. The delayed reproductive death and the number of colonies with giant cells increased in a dose-dependent manner in the surviving cells. Also, most of the surviving cells showed increased chromosome aberrations and mutation frequencies, indicating that genetic instability occurred even at more than 30 cell divisions after X-irradiation. We examined the effect of low dose radiation on genetic instability and found that pre-irradiation of 0.02Gy of X-rays with the interval of 5h suppressed the X-ray-induced genetic instability.

132 Radioadaptive Response: Improved Repair of Neocarzinostatin-induced
DNA Strand Breaks in Adapted Cells
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It has been suggested that radioadaptive response may involve the induction of an efficient repair mechanism for chromosomal DNA damage through de novo protein synthesis by activation of repair genes. Chinese hamster V79 cells adapted by 5 cGy pre-exposure and 4 h incubation were challenged by neocarzinostatin, and the yield of DNA double-strand breaks were measured by a comet assay. The initial yield of DNA double-strand breaks was at the same level in adapted cells and non-adapted cells. In adapted cells, the rejoining of DNA double-strand breaks was monitored over 120 min after neocarzinostatin treatment. The rate of rejoining of DNA double-strand breaks was higher in adapted cells than in non-adapted cells, and the amount of residual damage was lower in adapted cells than in non-adapted cells, as observed for radiation-induced DNA strand breaks. These results suggest that small radiation dose-induced improved repair might cross-react to DNA damage produced by other agents.