MODIFICATION OF X-RAY- AND U. V.-INDUCED KILLING OF CHINESE HAMSTER V79 CELLS BY 2'-CHLORO-THYMIDINE

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The effects of 2'-chloro-thymidine (2'-Cl-TdR) on cell killing induced by X- and U. V.-irradiation were investigated. Two types of cell lines, parent Chinese hamster V79 cells (TK^+ cells) and thymidine kinase deficient cells (TK^- cells), which were isolated from TK^+ cells by repeated BUdR treatments, were used. After irradiation the cells were incubated with medium containing 3.65 mM 2'-Cl-TdR for 24 hours. The medium was then changed to normal medium, and the cells were incubated for six to nine days for colony formation.

In the case of TK^+ cells, 2'-Cl-TdR enhanced the killing efficiency of both X- and U. V.-irradiation. The effect of 2'-Cl-TdR on U. V.-irradiated cells was more efficient than that on X-irradiated cells. From this result it was suggested that this drug interrupted the excision repair systems which could diminish the U. V.-induced lesions and a part of the X-ray-induced lesions. In the case of TK^- cells, 2'-Cl-TdR also enhanced the killing efficiency of both X- and U. V.-irradiation. However, when the effects of normal thymidine on cell killing were examined, thymidine showed an appreciable enhancement in killing efficiency of X- and U. V.-irradiation on TK^+ cells, and no effects on TK^- cells. These results suggested that the enhancement of cell killing by 2'-Cl-TdR might be explained by a mechanism independent of phosphorylation by thymidine kinase, while the phosphorylation of thymidine by thymidine kinase was responsible for the expression of cell killing.