Radiation-induced effects in PC-3 and DU-145 human prostate cancer cells

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Background: Prostate cancer is the first as an incidence and the second as a cause of the oncologic mortality in the male population. There is a broad range of possibilities in the prostate cancer therapy. However, there is also much controversy on the most appropriate treatment in the various stages of the disease. Advanced disease is mostly treated by radiation therapy, sometimes in combination with hormone or chemotherapy. Irradiation induces damage of cell biomolecules, which can lead to the arrest in cell division, or to apoptotic or necrotic cell death. The aim of this study was to determine the dose dependence of radiation-induced cell death in two human prostate cancer cell lines, and to define the form of death of these cells.

Methods: Human prostate cancer cell lines PC-3 and DU-145 were irradiated with 2-3 Gy from 60Co γ-source, at the dose rate of 20 Gy/h. The effect of irradiation on cell viability, morphology and DNA structure were followed 24 - 72 hours after treatment. Cells were analyzed by trypan blue exclusion assay, flow cytometry and DNA gel electrophoresis. Simultaneous staining of cells with Annexin V-FITC and propidium iodide enabled distinction of early apoptosis from late apoptosis and/or necrosis.

Results: The results of trypan blue staining indicated that radiation-induced cell death was both time and dose dependent process. According to flow-cytometry and DNA fragmentation assay, necrosis was the prevailing form of the radiation-induced cell death in both PC-3 and DU-145 cells. The apoptosis occurred in insignificant number of cells, probably due to the mutant p53 gene present in both cell lines. The cell necrosis was dose dependent and was most pronounced 72 hours post treatment.

Conclusion: The prevailing form of radiation-induced PC-3 and DU-145 cell death is necrosis. Both PC-3 and DU-145 are rather radioresistant cell lines, as the dose necessary to induce 50% decrease in viable cell number is about 10 Gy.