Biodosimetry of radiation-induced effects on DNA damage and cell cycle.

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Molecular biological markers of radiation response are thought to be of potential use to monitor the progress of radiation therapy and also to predict at an early stage the outcome of a radiotherapeutical treatment. They might also be a tool to monitor the populations potentially exposed after a radiological accident or a "dirty bomb" incident but also to monitor astronauts during a spaceflight during which they are submitted to cosmic radiations. The bone marrow and the blood are the most radiation sensitive tissues of the human body. Therefore, the specific study of the radiation effects on mononuclear cells is of particular importance to find radiation-induced biological markers. The study of radiation biomarkers includes DNA mutation, chromosome aberrations, apoptosis as well as protein, gene expression by the array technologies and cell cycle after propidium iodide staining. However, additional studies are needed to validate candidate biomarkers (molecular and/or proteic) for applied biological dosimetry applications and that could provide early and rapid information after exposure to radiation. In human mononuclear cells, we studied the effects of X- and gamma rays at low doses (from 0.015625, 0.03125, 0.0625, 0.125 to 0.25 Gy) to high doses (0.25, 0.5, 1, 2, 4, 6, 10, 15 and 20 Gy) on DNA damage and cell cycle. DNA damage was monitored by the 8-oxyDNA assay and the apoptosis by cell cycle analysis after Propidium Iodide staining and size reduction. The results show that X or gamma radiations induced a dose-dependent increase of DNA damage in mononuclear cells in comparison with the control samples. Moreover, cell cycle and the size reduction showed a higher number of cells in the sub-G1 phase (caracterising the apoptotic cells) in irradiated cells in comparison with the control. However, the pattern of the induction of the radiation-induced effects is different in function of the protocol applied (DNA damage, the cell cycle and the size reduction). This can be explained by the different radiosensitivities of the subpopulations amongst mononuclear cells and also the variation in intrinsic radiation sensitivities between individuals.

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