Contribution ID: **182** Type: **poster preferred**

Ionizing radiation induced genomic instability in human fibroblasts and modulating effects of telomerase activities in different processes of DNA DSB repair

Tuesday, 16 October 2012 16:39 (1 minute)

There have been suggestions that the telomerase system is involved in radiation induced genomic instability.

The aim of this study was to investigate the involvement of telomerase in the development of chromosomal damage and its repair at different stages of the cell cycle following an exposure of human fibroblasts to ionizing radiation.

We examine the relationship between several response criteria, including cell survival, chromosomal damage (using micronucleus), G2 induced chromatid aberrations (using conventional G2-assay as well as chemically induced premature chromosome condensation assay), and double strand breaks of the DNA (using γ-H2AX and Rad 51) in two cell lines, BJ human foreskin fibroblasts and BJ1-hTERT- a telomerase-immortalised BJ cell line.
To distinguish cells in G1, S and G2 phase, cells were co-immunostained for CENP-F, a protein whose expression is tightly cell cycle-regulated. Cells were also immunostained for the protein Cyclin A and the DSB marker 53BP1.

Following X-ray-irradiation (doses in the range of 0.1-6 Gy), for cell survival and micronuclei, where the overall effect is dominated by the cells in G1 and S phase no difference was found between the two cell types; in contrast, when radiation sensitivity at G2-stage of cell cycle was analysed, a significantly higher sensitivity was observed for the BJ cells in comparison to the BJ1-hTERT cells.
Therefore, it can be concluded that telomerase appears to be involved in DNA double strand break repair processes namely in G2 phase.

The data, taken overall, reinforce the idea that hTERT or other elements of the telomere/telomerase system may defend the chromosomes integrity by influencing the repair at G2 phase of cell cycle in human fibroblasts.

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Session Classification: Poster Session 1

Track Classification: DNA Damage and Repair