

Summary

The *RAD51* gene encodes a recombinase that is crucial for the repair of DNA double strand breaks by homologous recombination. We have found a significant increase of *RAD51* transcript level in Diffuse Large B-Cell Lymphoma (DLBCL) tissues compared to normal lymph node samples (LK). To study the pathological consequences that over-expression of *RAD51* may trigger in DLBCL, we used two cell-line models that have been established at the department of pathology. These tumor cell-line models can over-express *RAD51* ectopically by treatment with doxycycline. One of the two cell lines has 2 point mutations in the region encoding the DNA binding domain of P53 while the other cell line carries the wild-type *TP53* sequence. Using flow cytometric analysis, we found that ectopic over-expression of *RAD51* in induced wild-type *TP53* cell-line: i) triggered an arrest of the cell cycle in the G2/M phase, ii) increased the content of DNA within cells ($> 4N$), iii) and increased the fraction of apoptotic cells. These effects were absent in the mutant *TP53* cell-line model. We used karyotyping to visualize the effect of the over-expression of *RAD51* on chromosomes segregations, and we found that the induced wild-type *TP53* cell-line had an abnormally high number of chromosomes. This suggests that the combined action of P53 and *RAD51* is important for the regulation of mitotic phase and the genomic stability.