Chromosome- and Chromatid-type Aberrations after Irradiation of Various HRR or NHEJ Deficient Mutant Chinese Hamster Cells and Their Wild-type Counterparts in G0/G1 or LateS/G2 Cell Cycle Phases.

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Recently, we reported results of experiments examining the contributions of homologous recombination repair (HRR) and non-homologous end joining (NHEJ) systems involved in the processing of ionizing radiation damage leading to the formation of chromatid aberrations following exposure of cells in G2 or late S phases of the cell cycle(*1*). The current study extended these studies to examine aberrations after irradiation of cells in G0/G1. The approach in both cases was to compare aberration yields in Chinese hamster wild-type *vs* mutant cells deficient in HRR or NHEJ. We found that for cells irradiated in late S or G2, a deficiency in NHEJ led to a large increase in the yield of chromatid deletions but a very minimal or no increase in chromatid exchanges. This would be in accord with the idea that to form exchanges a rejoining would be necessary, so a lack of rejoining would then produce more deletions and less exchanges. For HRR deficient cells irradiated in S or G2 there was an increase in both deletions and exchanges. This also seems to fit with the above idea, because the HRR deficient cells are proficient in NHEJ but lack contributions of a repair system that only operates on a large scale “post replication”. The result for irradiation of these cell lines in G0/G1 was that for NHEJ deficient cells relative to wild-types there was an increase in both chromosome breaks and exchanges. In absolute terms for the same doses the yield of chromatid breaks per cell was much higher after S or G2 than chromosome breaks after G0/G1 irradiation, likely because there would be more total repair time for the latter The effect here was similar to what had been reported by us and others on previous occasions [reviewed in(*2*)]. For the HRR mutant cells irradiated in G0/G1, there was very little increase in either breaks or exchanges in the irs-1 (Xrcc2 deficient) or irs-3 (Rad51C deficient) cells, but there was a significant increase in both breaks and exchanges in the irs-1SF cells. However, the increase in breaks and exchanges in these G0/G1 irradiated irs-1SF cells which are deficient in *Xrcc3* (but proficient in NHEJ) is almost entirely due to increased levels of chromatid-type breaks and exchanges.

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References

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