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**Frequent Loss of Heterozygosity in Radiation-Induced Lymphomas of Mice**

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We have analyzed the loss of heterozygosity (LOH) at polymorphic microsatellite loci and cytogenetic status in radiation-induced lymphomas of (BALB/cHeA x STS/A)F1 hybrid mice. Highly frequent LOH was observed in the regions on chromosome 4, 12 and 19, although the frequencies of LOH were very low (0-6%) in other regions examined so far. A region containing D19Mit11 locus (31 cM from centromere) on chromosome 19 showed 51% of LOH (37/73 lymphomas), while regions containing D4Mit7 locus (32 cM) on chromosome 4 and D12Mit17 locus (57 cM) on chromosome 12 showed 30% (14/47) and 62% (29/47) of LOH, respectively. In only the region of LOH on chromosome 4, alleles derived from parental STS/A mice, a resistant strain to radiation lymphomagenesis, were lost significantly more than those of the other sensitive parents, BALB/cHeA mice. Cytogenetical analysis of six lymphomas with both wide range of LOH on chromosome 4 and on chromosomes 12 or 19 showed obvious deletion of the chromosomes. These results suggest the generation of wide range of uniparental disomy for these regions.

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**Involvement of Ubiquitin-Conjugating System in DNA Repair in Mammalian Cells**

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We reported previously the increased sensitivity and defective induced mutagenicity of ts85 cells, a temperature-sensitive mutant of ubiquitin-activating enzyme E1, to ultraviolet light (UV) than its parental mouse cell line FM3A. The similar phenotypes have been reported for some yeast DNA repair-defective mutants which are also deficient in the ubiquitin system, like as *Saccharomyces cerevisiae* rad6 mutants. However, the mechanism of the mutagenic deficiency observed there is still unknown. Now we tried an analysis on this mechanism and found a significant elongation of the expression time for the mutation induction in ts85.

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**Radioprotective effects of L-ascorbic acid**

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We have previously reported that post-irradiation treatment by L-ascorbic acid (AsA) efficiently reduced mutation frequency in X-irradiated human embryo cells. We also found that AsA scavenged long-lived organic radicals (LL radicals) (T<sub>1/2</sub>=20 hours) raised in gamma-rays-irradiated albumin solution rather than short-lived radicals, such as OH radicals. Therefore, we speculated that LL radicals might be related to mutation induction in human cells. To make clear this speculation, in this study we treated cells with AsA 20 hours after X-irradiation. We could not find any difference in surviving fraction between cells treated with AsA soon after irradiation and cells treated 20 hours after irradiation, but mutation frequency was remarkably suppressed in both cells. These results suggest that the LL radicals still remain in cells 20 hours after irradiation, and they may play more important role than short-lived radicals for mutation induction in X-irradiated cells.