154 The genetic complementation assay of Nijmegen breakage syndrome with ataxia telangiectasia on the basis of radiation sensitivity to cell killing.

Hideki TAKAMI[1,2], Shinya MATSUURA[1], Hiroshi TAUCHI[1], Satoru ENDO[1], Seiji KODAMA[3], C. WEEMAES[4], Kenshi KOMATSU[1]([1]Dept. Rad. Biol., Res. Inst. Rad. Biol. & Med., [2]Pharmaceut. Sci., Hiroshima Univ., Hiroshima 734. [3]Pharmaceut. Sci. Nagasaki Univ. [4]Nijmegen Univ.)

Based on the radiation sensitivity of an immortal cell line derived from Nijmegen breakage syndrome(NBS), the genetic complementation of this syndrome by classical ataxia telangiectasia(AT) was examined using somatic cell fusion. When the newly established NBS cells were fused with At cells, the radiation sensitivity was significantly reduced, supporting the genetic heterogeneity of both disease. This was confirmed by the introduction of a normal human chromosome 11(which includes the gene for AT)into NBS cells via microcell-mediated chromosome transfer. Our result indicated that the underlying gene for Nijmegen breakage syndrome is distinct from that of AT.

155 Genetic heterogeneity of ataxia-telangiectasia-like hamster mutant irs2 with Nijmegen breakage syndrome.

Shuichi SAKAMOTO[1,2], Kenshi KOMATSU[1], Hiroshi TAUCHI[1], Shinya MATSUURA[1], Satoru ENDO[1], J. THACKER[3]([1]Dept. Rad. Biol., Res. Inst. Rad. Biol. & Med., [2]Pharmaceutical Sci., Hiroshima Univ., Hiroshima 734. [3]MRC, England)

The genetic complementation of hamster mutants irs2, belonging to the XRCC6, was examined using somatic cell fusion with cells derived from Nijmegen breakage syndrom (NBS). When irs2 cells were fused with an immortal NBS cells, GM7166VA7, the radiation sensitivity was significantly reduced, implying the genetic heterogeneity of both mutants. To confirm this result, several kinds of a human chromosome were introduced into irs2 cell via microcell-mediated chromosome transfer. None of the microcell hybrid presently obtained showed the restoration of radiation resistance and study on searching the human chromosome including irs2 gene is going on.

156 Generation of Somatic Cell Hybrids Carrying Human Chromosome 11q23 Fragments and Their Use for the Analysis of AT Locus

*Yosuke EJIMA, **Mitsuo OSHIMURA and *Masao S. SASAKI; *Radiat. Biol. Center, Kyoto Univ., Kyoto 606, **Sch. Med., Tottori Univ., Yonago 683

A panel of somatic cell hybrids were generated that carry human chromosome fragments spanning various region of 11q23 close to ataxia telangiectasia (AT) locus. A mouse A9(3552)-2 cell strain that contains a human X/11 chromosome with a translocation breakpoint at 11q23 was used as a source to generate a set of radiation hybrids that preferentially retain human 11q23 fragments. PCR assay was used to determine the presence or absence of chromosome 11 loci in each hybrid. Among a total of 23 hybrids generated, 6 clones retained the whole 11q23 region while 3 clones showed a total deletion of that region. The breakpoints in the remaining 14 hybrids were mapped within 11q23, while they were not randomly distributed: 8 clones had breakpoints in *NCAM*~*D11S351* interval and 6 clones in *GRIA4*~*D11S927* interval. Of particular interest is that the latter 3Mb region where the breakpoints of 6 clones had been clusterd was mostly identical to the candidate region for group A/C AT gene locus. Two of the hybrids, MH12/2 and MH12/5, had breakpoints that is mapped either proximal or distal to *D11S535* locus, where the candidate AT(*ATM*)gene is located, implicating their potential use to examine the phenotypic complementation of *ATM*-containing genomic region.