Numerical changes of chromosome 17p (p53locus) detected by FISH in gastric cancer

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Numerical changes of chromosome 17p (p53locus) detected by FISH in gastric cancer

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Human pathology

Research Institution

Kanazawa University

Principal Investigator

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Research Abstract

To understand better the role of p53 deletion in gastric adenocarcinoma, 42 cases were examined by dual-color fluorescence in situ hybridization (FISH).
Probes for centromere 17 and the p53 locus (17p13.1) were hybridized simultaneously to interphase cancer cells to analyze p53 and chromosome 17 copy numbers on a cell by cell basis. The result was compared with the loss of heterozygosity (LOH) for probe YNZ22 at 17p13.3 detected by restriction fragment length polymorphism, and nuclear overexpression of p53 proteins examined immunohistochemically with anti-p53 protein antibody (RSP53). Delection was difined as when the fraction with decreased number for p53 gene signals compared with centromeric signals including monosomy fractions exceeded 60% of cell nuclei counted. Eleven of 15 cases showing deletion also showed LOH and nuclear overexpression of p53 protein. In these cases allelic loss was thought to be caused by physical deletion of 17p13.1 and point mutation of the

remaining gene was highly suggestive. Five cases with neither deletion nor LOH had large population of cancer cells over-expressing p53 protein. This suggested mutant form of the p53 protein functioned in a dominant negative fashion in these cases. Other 10 cases without deletion showed LOH in spite of being disomic for both the centromeric and p53 probes. Remaining 12 cases showed neither deletion, LOH nor overexpression of p53 protein, thus it was very likely that p53 gene were not implicated in malignant progression in these cases. It was concluded that FISH is a useful tool to analyze aberrations of p53 gene in gastric adenocarcinoma.

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