F115

Genetic Polymorphisms of a Short Tandem Repeat Locus Human Coagulation Factor XIII A Subunit gene and B Subunit Gene in Koreans

이정용*, 이정주[1]
서울대 분자생물학과, [1]생물학과

Human Coagulation Factor XIII A Subunit gene (F13A01) and B Subunit gene (F13B) are short tandem repeat loci. Each individual has a pair of varying numbers of repeat sequences. We studied the polymorphism of F13A01 among 292 unrelated Korean individuals and that of F13B among 295 individuals. For F13A01, sixteen genotypes comprised of seven alleles (alleles 3.2, 4, 5, 6, 7, 12, 15) were identified. Allele 15 of Asians had not been reported before. Deviation from Hardy-Weinberg expectation (HWE) was observed. For F13B, seven genotypes comprised of four alleles (alleles 8, 9, 10, 11) were identified. No deviation from HWE was observed for F13B.

F116

Type II Protein Kinase A Up-Regulation Is Sufficient to Induce Growth Inhibition in SK-N-SH Human Neuroblastoma Cells

Se Nyun Kim*[1,2], Gap Ryol Lee[1,2], Kwang Ho Cheong[1,2], Sang Dai Park[1], Seung Hwan Hong[1,2]
[1]Department of Molecular Biology and Research Center for Cell Differentiation, [2]Institute for Molecular Biology and Genetics, Seoul National University.

High level of type I protein kinase A has been correlated with cell growth and transformation. We have previously shown that overexpression of RIIβ subunit of protein kinase A, which markedly reduces RIα protein, induces growth inhibition in SK-N-SH human neuroblastoma cells. To determine whether the reduction of RIα or protein kinase A isozyme type I is essential in the growth inhibition of SK-N-SH cells, we overexpressed RIα in sense and antisense orientation. Type I protein kinase A activity was increased in the RIα-overexpressing cells and was decreased in the RIα antisense-expressing cells. However, the changes of type I protein kinase A activities in RIα-overexpressing and RIα antisense-expressing cells did not affect cell growth. Overexpression of RIIβ or Cα increased type II protein kinase A and inhibited cell growth in both cell lines regardless of type I protein kinase A level. These results indicate that type II protein kinase A activity is the main effector in the cAMP-mediated growth regulation of SK-N-SH human neuroblastoma cells.