F109 Expressions of CD44 variants in Korean colorectal cancer and metastatic lymph node

So Young Chun[1*], Ok Suk Bae[2] and Jong Bong Kim[1]
Department of Biology[1], Catholic University of Taegu-Hyosung,
Department of Surgery, Medical Science[2], Keimyung University

CD44 has been known as a lymphocyte homing receptor gene and is expressed on a wide variety of tissues. We designed primers CD44V6/7, CD44R1, CD44V and CD44V6-10, respectively. Expression of CD44 variants were investigated in normal colonic mucosa, colorectal cancers, adjacent mucosa around cancer tissue and lymph nodes in 44 human colorectal cancer patients by RT-PCR. CD44V6/7 was observed in 28 out of 39 cases(71.79%) of tumors and 15 out of 22 cases(68.18%) of lymph nodes, and CD44R1 containing variant exon 8, 9 and 10 was observed in 28 out of 39(71.79%) of tumors, 4 out of 11 cases(36.36%) of normal lymph nodes and 3 out of 4 cases(75.00%) of metastatic lymph nodes. CD44V6/7 and CD44R1 were significant at colorectal cancers and lymph nodes($p<0.05$). These results suggest that the expression of CD44 variants(containing variant exon 6, 7 and 8, 9 and 10) can be considered as marker for tumor and could for detect of metastasis of colorectal cancer.

F110 Induction of Apoptosis by EGCG in Human Colon Cancer Cell Line, HCT116 Cells

Kang YoonSung*, Lee YunKyung, Jung SungKook, Moon YoungJoon and Lee KwangHo
Department of Life Science, ChungAng Univisity

As previously reported, (-)-epigallocatechin gallate (EGCG), a catechin polyphenol compound, could induce an apoptosis in several human cancer cell lines. However, the molecular mechanism for the apoptosis caused by EGCG remains to be elucidated. In this study, we focused on whether p21 is involved in the apoptosis induced by EGCG in a human colon cancer cell line, HCT116. The EGCG treatment of HCT116 cells induced the apoptosis at various doses resulting in the formation of internucleosomal DNA fragment and the inhibition of cell growth. In order to access the molecular mechanism of growth inhibition of EGCG, we also transfected the plasmid vector containing antisense p21 cDNA into HCT116 cells. Transformants were confirmed by PCR and western blot analysis for the incorporation of vector and p21 suppression, respectively. It was found that the transformants, that is p21-suppressed cells, were less sensitive to the inhibition of growth than nontransfected HCT116 cells. These results may suggest that the apoptosis induced by EGCG in human colon cancer cells is regulated in the p21-dependant manner.