



Frequent inactivation of *p16* and *p15* expression in human esophageal squamous cell carcinoma detected by RT-PCR

Lian-Hua Jiao, Li-Dong Wang, Eric-Poe Xing, Guang-Yu Yang Yang, Chung S Yang

Lian-Hua Jiao, Li-Dong Wang, Laboratory for Cancer Research, Henan Medical University, Zhengzhou 450052, Henan Province, China

Eric-Poe Xing, Guang-Yu Yang Yang, Chung S Yang, Laboratory for Cancer Research, College of Pharmacy, Rutgers University, Piscataway, NJ 08854, United States

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Correspondence to: Dr. Lian-Hua Jiao, Laboratory for Cancer Research, Henan Medical University, Zhengzhou 450052, Henan Province, China

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Abstract

AIM: The cyclin-dependent kinase inhibitors *p16* and *p15* play important roles in the regulation of the cell cycle, and have been found to have tumor suppressor roles in a variety types of cancer. It has been shown that *p16* aberrant methylation and *p15* homozygous deletions were frequently involved in human esophageal squamous cell carcinoma (ESCC). The present study was to examine the impact of such molecular alterations on the expression of these genes.

METHODS: The mRNA level of both genes was measured in 21 frozen ESCC specimens using semi-quantitative RT-PCR.

RESULTS: Nineteen cases were observed at a low basal level

of *p16* expression (0.11 ± 0.07 , expression units normalized by housekeeping glyceraldehyde-3-phosphate dehydrogenase gene as internal standard) in the normal epithelia adjacent to the cancer tissue. Among the 19 cases, only 5 showed a significant elevation of *p16* expression (> 3.2 folds) in the tumor, whereas the remaining 14 showed either a slight increase (1-2 folds), or decreased *p16* expression compared to normal, whereas 11 had only a slight increase (1-2 folds) or a decrease in tumor. In the 5 cases where *p15* was already activated (> 0.5) in the adjacent normal epithelium, 4 of them had similar or a slightly lower expression level, but one had a great decrease in *p15* expression ($< 1\%$ of the normal level). For intact *p16* and *p15* genes, which encode cell cycle regulators, significant increase of their expression is expected in the cancer cells as a response to accelerated cellular proliferation. However, in our samples, significant activation was only seen in 7 cases for *p16* gene and 9 cases for *p15* gene. Fourteen cases for *p16* (70%) and 12 cases (57%) for *p15* either maintained low basal levels or had decreased expression levels in tumor, respectively, which indicate suppression or inactivation of the genes. No correlation between *p16* and *p15* expression was observed in our frozen samples.

CONCLUSION: The present findings indicate that distinct and independent mechanisms are involved in the inactivation of *p15* and *p16* genes in ESCC.

Key words: Esophageal neoplasms; Carcinoma, squamous cell; *p16* gene; *p15* gene; Gene expression

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