The Molecular Mechanism of FGFRs and Its Application for Cancer Therapeutics

Jun-Hyeog Jang, Ji-Hyun Kim, Jang Hyun Jun
Inha Univ. College of Medicine
TEL: +82-32-890-0933, FAX: +82-32-882-1877

In previous, We have previously demonstrated that the expression of *FGFR3* is frequently down-regulated in colorectal carcinoma cells[1]. In this study, we have found that *FGFR1* is overexpressed in colorectal carcinoma cells and the gene expressions between *FGFR1* and *FGFR3* are mutually exclusive. Moreover, we have also demonstrated that the disruption of *FGFR1* expression by introducing of FGFR1 siRNA was effective in elevating *FGFR3* expression and tumor suppressive activities[2]. Thus, *FGFR1* may confer a selectable advantage on clones of cells in colorectal tumorigenesis, favoring proliferation, whereas *FGFR3* may have the effect of an unfavorable negative regulation of progression of the carcinomas to malignancy, promoting differentiation. Our results indicate that the reciprocal relationship in gene expression between *FGFR1* and *FGFR3* in colorectal tissue plays an important role in the progression of the carcinomas to malignancy.

References

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