MUTATIONAL ANALYSIS OF PROAPOPTOTIC INTEGRIN BETA 3 CYTOPLASMIC DOMAIN IN COMMON HUMAN CANCERS

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Aims: Mounting evidence indicates that deregulation of apoptosis is involved in the mechanisms of cancer development. Integrins are cell adhesion receptors that mediate cell survival and migration. A recent study showed that unligated integrin beta 3 (ITGB3) induced apoptosis by recruitment of caspase-8. The aim of the present study was to explore the possibility that genetic alteration of the ITGB3 gene is involved in the development of human cancers possibly by inactivating the apoptosis function of ITGB3.

Methods: We analyzed the coding region of the cytoplasmic domain of the human ITGB3 gene for the detection of somatic mutations in 100 gastric, 90 colorectal, 100 non-small cell lung, 43 urinary bladder and 50 head-neck cancers by a polymerase chain reaction-based, single-strand conformation polymorphism.

Results: We found an identical ITGB3 mutation in two unrelated patient samples (one in colorectal and the other in bladder cancer). The ITGB3 mutation was a missense mutation which would substitute an amino acid (E757K).

Conclusions: The data suggested that the proapoptotic ITGB3 cytoplasmic domain is rarely mutated in common human cancers and may not play an important role in the development of the cancers.

Key words: apoptosis, cancer, integrin beta 3, mutation.

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