Tumori, 97: 211-213, 2011

Absence of paxillin gene mutation in lung cancer and other common solid cancers

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ABSTRACT

Aims and background. Mounting evidence indicates that deregulated cell adhesion is involved in the mechanisms of cancer pathogenesis. A recent study showed that the paxillin gene (*PXN*) encoding a focal adhesion protein was somatically mutated in lung cancers. The aim of this study was to confirm the presence of *PXN* mutations in lung cancers as well as in other common solid cancers.

Methods. We analyzed somatic *PXN* mutations in 45 lung, 45 gastric, 45 colorectal, 45 breast, 45 liver and 45 prostate cancers by polymerase chain reaction and single-strand conformation polymorphism assay.

Results. Neither lung nor other cancers were found to be associated with somatic mutations of *PXN*.

Conclusions. In contrast to the previous report, our study revealed that *PXN* mutation was absent in lung cancers and other common solid cancers, suggesting that *PXN* mutation may not play a principal role in solid cancer development.

Key words: paxillin, mutation, cancer, lung cancer.

Acknowledgments: This study was supported by a grant of the Korean Health Technology R&D Project, Ministry for Health & Welfare, Republic of Korea (A092258).

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Received June 17, 2010;
accepted October 21, 2010.