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[Medicine][Cancer] Academia Sinica Research Team Found Gene Reducing Invasion of Cancer Cells [Medicine][Cancer] Academia Sinica Research Team Found Gene Reducing Invasion of Cancer Cells (<u>Chinese</u> <u>Version</u>)

Academia Sinica's Newsletter (2009/05/27) announced, Taiwan researchers have now discovered that normal p53 restrains the metastasis of cancer cells from the primary cancer, thus reducing the possibility of secondary cancer growth. Their research was published online in the prestigious scientific journal, Nature Cell Biology on 17th-May.

The newsletter reported, the scientists discovered that the normal p53 gene induces the degradation of the tumor invasion factor called Slug, which results in less metastasis. Mutant p53 on the other hand, suppresses Slug degradation and thus increases cancer invasiveness. The discovery may be an important mechanism in lung cancer tumor formation and metastasis and may be useful for the development of new lung cancer therapies.

The research, supported by National Research Program for Genomic Medicine (Department of Health and National Science Council), was directed by Dr. Pan Chyr YANG, Dean of the College of Medicine at National Taiwan University, and Academician and Joint Appointment Research Fellow at Academia Sinica; and Tse-Ming HONG, Associate Professor of the Institute of Clinical Medicine at National Cheng Kung University. The project was implemented by Shu-Ping WANG a Ph. D. student at the Graduate Institute of Life Sciences at the National Defense Medical Center in Taipei.

The team discovered that normal p53 and its downstream molecule, MDM2, regulate the stability of the tumor invasion factor, Slug. The formation of the p53-MDM2-Slug complex facilitates Slug degradation, thus reducing metastasis. However, once p53 has mutated, this invasion factor becomes stable and increases the invasive and metastatic capability of tumor cells.

Dr. YANG also pointed out that the p53-MDM2-Slug regulating pathway was also clinically verified by lung cancer specimens, confirming that this new discovery may serve as an important mechanism for lung cancer tumorigenesis, invasion and metastasis and as a new target for lung cancer therapy.

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