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Understanding of How Cells Turn Cancerous Advances at UCR

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Researchers at the University of California, Riverside have uncovered a key step in how healthy cells protect themselves from mutating into cancerous tumor cells.

A research team, led by Professor of Biochemistry Xuan Liu, showed how proteins in the nucleus of cells, PTEN and p53, interact to prevent a cell that has suffered DNA damage from mutating into a cancerous tumor.

Liu, who has been studying the workings of tumor suppressor proteins for the past decade, says the latest discovery by researchers in her laboratory provides an exciting new link in understanding how these proteins work together to regulate how or whether the cell replicates or dies.

“Our previous paper showed that these two proteins (PTEN and p53) work together to regulate the cell,” she said. “This paper begins to show how the two work together.”

Liu published her research findings in a featured article titled Mechanistic insights into maintenance of high p53 acetylation by PTEN, in the Aug. 18 issue of *Molecular Cell*. Co-authors include UCR colleagues Andrew G. Li, Landon G. Piluso Xin Cai and Gang Wei; with William R. Sellers in the Department of Medical Oncology, Dana Ferber Cancer Institute of Harvard University in Boston.

They found that when a cell's DNA becomes damaged, PTEN forms a complex with another protein, p300, which in effect, switches on p53, a very important tumor suppressor.

"I would like to continue to expand our understanding of how p53 is activated in conjunction with PTEN and under what circumstances it functions to protect the cell," Liu said.

Because her work has been done mostly in the test tube, Liu would like to determine whether her findings can be replicated in the animal testing phase.

Source: University of California, Riverside