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Study identifies genetic cause of most common form of breast cancer

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The discovery of tumor-suppressor genes has been key to unlocking the molecular and cellular mechanisms leading to uncontrolled cell proliferation - the hallmark of cancer. Often, these genes will work in concert with others in a complex biochemical system that keeps our cells growing and dividing, disease free.

Now researchers at the University of North Carolina at Chapel Hill School of Medicine and UNC Lineberger Comprehensive Cancer Center have found that defects in one gene, called p18, may override the rest, eventually leading to cancer.

This discovery, combined with new laboratory techniques, will help scientists identify and test new treatments for luminal-type tumors, which account for between 70 and 80 percent of all breast cancers, but are generally slower growing than other types.

The results of the research appear in the May 2009 issue of *Cancer Cell*.

Defects in the p18 gene have been observed in different types of human cancer. Senior study author Yue Xiong, Ph.D., William R. Kenan Jr. Distinguished Professor of biochemistry and biophysics, observes, "When this gene is not expressed or is deleted, cells have no braking mechanism. They will continue to grow and divide until they turn into cancer."

Xiong and his colleagues specifically targeted the role that p18 plays in

the development of luminal breast cancers. Using genetically-engineered mice with deletion of p18 genes, they created a highly reliable model of human breast cancers. The researchers tested their model by analyzing the gene in samples from approximately 300 human breast cancer patients, proving that the decreased expression of the p18 gene is highly correlated with the development of luminal tumors.

"The mechanism behind these tumors is quite different from that of other forms of breast cancer. Understanding this mechanism and having a good mouse model allows us to specifically test how treatments work against these tumors, which may then benefit human patients," said Xiong.

Source: University of North Carolina School of Medicine (news : web)