

Protein helps cells duplicate correctly, avoid becoming cancer

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Xiaoqi Liu found that the loss of a particular protein can lead to uneven distribution of DNA during cell duplication, causing those cells to become cancerous. Credit: Purdue Agricultural Communication photo/Tom Campbell

A Purdue University researcher has discovered that the absence of certain proteins needed for proper cell duplication can lead to cancer.

Xiaoqi Liu, an assistant professor of <u>biochemistry</u>, found that cytoplasmic linker protein-170, or CLIP-170, plays a major role in proper cell duplication and DNA distribution. When the protein is removed, cell duplicates lack entire copies of DNA and can become cancerous. Liu's findings were published in the early online version of the <u>Journal of Biological Chemistry</u>.



"DNA has to be equally distributed from a mother cell to its daughter <u>cells</u>. If the cells are not identical to the mother cell, they become cancer," Liu said. "Normal cells have a very tightly regulated process to avoid aneuploidy, or the unequal distribution of <u>chromosomes</u>. Aneuploidy is a hallmark of cancer."

A cell will go through two important processes before it divides: It will create a second copy of its own DNA, and it will create two centrosomes, or poles, that will act as magnets to draw the DNA to themselves. When the centrosomes have attracted the DNA, the cell divides in a process called mitosis, creating two identical cells.

But when CLIP-170 was removed from a cell in Liu's lab, more than two centrosomes formed, pulling only two copies of the DNA in several directions. Each centrosome received less than a full copy of the <u>DNA</u>.

"If there are multiple poles, the cell becomes confused and becomes cancerous or transformed," Liu said.

Liu also found that a cyclin-dependent kinase called Cdc2, activates CLIP-170 in cells by attaching a phosphate group to the protein. Cdc2 is an enzyme that is considered the master regulator of cell growth, and without it, CLIP-170 does not do its job and multiple centrosomes form.

"Without either of these, a cell has an increased chance of becoming a cancer cell," Liu said.

The National Institutes of Health funded Liu's research. Liu will continue to look for regulators and mechanisms that turn normal cells into <u>cancer</u> cells

Source: Purdue University (<u>news</u> : <u>web</u>)



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