

Researchers discover switch that causes the body to produce cancerous cells

September 4 2009

A team of Syracuse University researchers discovered a second molecular switch within the Mixed Lineage Leukemia protein complex that they believe could be exploited to prevent the overproduction of abnormal cells that are found in several types of cancer, including leukemia.

The paper was designated as the "Paper of the Week" in the September 4 issue of the [Journal of Biological Chemistry](#) (*JBC*), published by the American Society for Biochemistry and Molecular Biology. Only the top 1 percent of the more than 6,600 articles published each year in *JBC* receives this prestigious designation.

The research team is led by biologist Michael Cosgrove, assistant professor in SU's College of Arts and Sciences. Anamika Patel, a post-doctoral researcher in Cosgrove's lab, who is being featured on *JBC*'s website, did much of the experimental work for the paper.

During the course of their research to better understand MLL, a protein switch that helps regulate the formation of white blood cells, Cosgrove's research group discovered a new [molecular switch](#) within the MLL complex, which they labeled W-RAD.

"We thought that MLL was the only switching mechanism present in this protein complex," Cosgrove said. "However, we discovered the complex is really two switches."

In normal cells, MLL combines with four proteins that comprise the W-RAD group to create a molecular switch that controls DNA packaging events required to form white blood cells. When the MLL switch is broken, white blood cells do not mature properly, resulting in a dangerous proliferation of abnormal cells.

Similarly, the proteins that form the W-RAD complex are overproduced in several types of cancer cells, but until now, scientists did not know the function of these proteins. Cosgrove's group discovered that the W-RAD proteins form a new kind of switch—one that has never been seen before.

"The W-RAD switching mechanism signals the cell to create multiple copies of [cancer](#) cells," Cosgrove says. "If we can find a way to turn off this switch, we might be able to slow or stop the production of [abnormal cells](#) and convert them to normal cells."

Source: Syracuse University

Citation: Researchers discover switch that causes the body to produce cancerous cells (2009, September 4) retrieved 2 May 2024 from <https://medicalxpress.com/news/2009-09-body-cancerous-cells.html>

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