Researchers reveal how Trop2 protein drives tumor growth in prostate, other epithelial cancers

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(Medical Xpress)—Researchers led by Tanya Stoyanova and Dr. Owen Witte of UCLA's Eli and Edythe Broad Center of Regenerative Medicine and Stem Cell Research have determined how a protein known as Trop2 drives the growth of tumor cells in prostate and other epithelial cancers.

This discovery is important because it may prove essential for creating new therapies that stop the growth of cancer, the researchers said. The study is featured on the cover of the Oct. 15 issue of the journal Genes and Development.

The Trop2 protein is expressed on the surface of many types of epithelial cancer cells—cells that form tumors that grow in the skin and the inner and outer linings of organs—but little was known about the protein's role in the growth and proliferation of cancer cells. The UCLA researchers discovered that Trop2 controls those processes through a mechanism that leads to the protein being cleaved into two parts, one inside the cell and one outside. This Trop2 division promotes self-renewal of the cancer cells, resulting in tumor growth.

"Determining the mechanism of this protein is important for planning treatments that stop the growth of prostate cancer, but it is also overexpressed in so many other types of cancer that it might be a treatment target for many more patients beyond that population," said

senior author Witte, director of the Broad Center and a professor in the department of microbiology, immunology, and molecular genetics at UCLA.

The finding may have a critical clinical impact, the researchers said, since preventing the cleavage of Trop2 by mutating those sites on the protein where it splits eliminates the protein's ability to promote tumor cell growth. Using this knowledge, they said, new therapy strategies can be developed that block Trop2 molecular signaling, thus stopping its ability to enhance tumor growth in a variety of epithelial malignancies, including prostate, colon, oral cavity, pancreatic and ovarian cancers, among others.

"The reason I became interested in Trop2 was that it is highly expressed in many epithelial cancers but no one knew precisely how the protein worked to promote the disease," said Stoyanova, the study's first author and a postdoctoral scholar in the department of microbiology, immunology and molecular genetics at UCLA.

Provided by University of California, Los Angeles

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