

Researchers discover new role for protein in cell division

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Pharmaceutical sciences researchers at Washington State University have discovered a protein's previously unknown role in cell division.

The well known protein ATF5, or Activating Transcription Factor 5, controls how often specific genes are expressed, or copied from DNA. ATF5 regulates genes that control cell survival.

But the research team has identified a part this protein is playing that is not related to its transcription factor role. Within the part of the cell called the centrosome, ATF5 is also acting as a structural protein.

Structural proteins perform much like their name suggests: they maintain cell shape and make up connective tissues like cartilage and bone.

Telling a cell biologist that a transcription factor is doubling as a structural protein is like telling your neighbor you are building a backyard shop on a foundation of pudding. The scientist—and your neighbor—wouldn't believe you. It's never been seen before.

"This is an eye opener for people working in the field," said David Liu, a member of the research team.

He is an associate professor at the WSU College of Pharmacy and corresponding author on the research, to be published in the July 30 issue of *Cell*.



Liu and five pharmaceutical sciences colleagues at the WSU Health Sciences campus in Spokane teamed with scientists from Penn State, the Chinese Academy of Sciences, Zhejiang Sci-Tech University and the University of Texas to complete the study that was funded in part by the American Cancer Society and the U.S. Department of Defense.

Cells studied include ovary and breast cancer and glioma (brain tumor).

The discovery provides the first evidence of structural interactions within the centrosome and the role of ATF5, which was "strategically located within the centrosome and playing a totally different role than we previously understood," said Liu.

The centrosome is the cell component vital to successful <u>cell division</u> and duplication, which affect a wide spectrum of larger processes from healing to cancer growth to fighting off disease.

"Failure of centrosome duplication can result in malformation of mitotic spindles, causing a variety of genomic instabilities," said Liu. Malformed <u>cells</u> contribute to tumor development and conditions such as dwarfism, ciliopathy, microcephaly and problems with cilia movement.

Cilia work like antenna and communicate with the rest of the cell to move it toward nutrients, but knowledge is limited on how cilia work, said Liu. This discovery helps connect the dots, and Liu is hoping this research will expand understanding of <u>cell survival</u>.

"Cell survival is fundamental and affects all of our cells, with broad implication across many types of disease. Knowing how to correct a defect, we have the hope to treat disease," he said.

In a previous study, Liu tagged the ATF5 protein with green dye, a fluorescent protein produced by jellyfish, and discovered ATF5's



presence in an area of the cell that didn't seem to make sense. He wanted to show that the presence of ATF5 outside its "normal place" was irrelevant so he could get back to studying what he was originally focused on: ATF5's role as a transcription factor.

"After finding this, I wanted to disprove it. But the more I worked on it, the more it was apparent it had real purpose," said Liu. "Ultimately, it became a big discovery."

Provided by Washington State University

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