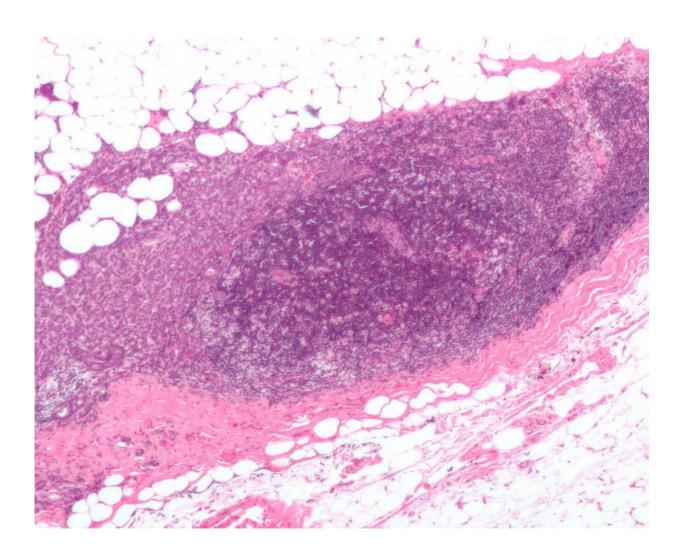


Can protein 14-3-3 sigma prevent or kill breast cancer tumors?

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Micrograph showing a lymph node invaded by ductal breast carcinoma, with extension of the tumour beyond the lymph node. Credit: Nephron/Wikipedia



Every parent knows the maxim "feed a cold, starve a fever." In cancer, however, exactly how to feed or starve a tumor has not been easy to determine.

A study led by scientists at The University of Texas MD Anderson Cancer Center has shown that a simple molecule called 14-3-3 sigma could be one answer for explaining <u>cancer metabolism</u>, the chemical process by which a tumor forms, grows or dies.

"We know that all cancers grow by learning how to reprogram their metabolism," said Mong-Hong Lee, Ph.D., professor of Molecular and Cellular Oncology. "But exactly how this occurs has not been fully understood. Our study showed that 14-3-3 sigma opposes and reverses tumor-promoting metabolic programs."

Lee's study results, which appear in the July 16, 2015 issue of *Nature Communications*, revealed new understanding about how 14-3-3 sigma - a cell cycle "controller" -regulates <u>cancer</u> metabolic programming, thus protecting healthy cells from turning into tumor-producing factories.

In vivo and in vitro experiments showed that, among many biochemical effects, 14-3-3 sigma suppresses cancer glycosis, which prevents cancer's ability to convert glucose into pyruvate, a substance essential for cell growth.

"14-3-3 sigma expression levels can help predict overall and recurrence-free survival rates, tumor glucose uptake, and metabolic gene expression in <u>breast cancer patients</u>," said Lee. "These results highlight that 14-3-3 sigma is an important regulator of <u>tumor metabolism</u>, and loss of 14-3-3 sigma expression is critical for cancer metabolic reprogramming."

Lee believes that the study findings provide additional insight about the "crosstalk" between cancer metabolism and <u>cell cycle</u>.



"We anticipate that pharmacologically elevating 14-3-3 sigma's function in tumors could be a promising direction for targeted anti-cancer metabolism therapy development in the future," he said.

Provided by University of Texas M. D. Anderson Cancer Center

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