

Researchers find new target for breast cancer treatments

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(PhysOrg.com) -- A study by Tulane University School of Medicine and Vanderbilt University School of Medicine suggests a new target in the fight against breast cancer - a cell receptor and associated protein molecule that create resistance to cancer drugs and help spread tumor cells throughout the body.

Published in the journal <u>Cancer Research</u>, the study demonstrates that CXCR4, a receptor on the membranes of cells and a protein molecule that binds to it are responsible for both creating resistance to estrogen hormone therapy (a treatment for breast cancer) and causing cancer cells to metastasize, or spread throughout the body.

Estrogen, essential to women's development and health, is also essential to the growth of breast cancer tumors. Current therapy for breast cancer uses "blockers" that deny estrogen to cancer cells. However, in approximately 20-30 percent of patients resistance to these blockers develops and tumors keep growing. To understand why, the researchers examined samples from breast cancer patients.

"We found that the presence of another receptor, CXCR4, is associated with poor prognoses and decreased long term survival," says researcher Matthew Burow, associate professor of medicine at Tulane University School of Medicine. "We looked at this receptor and were able to show that it bypasses the estrogen receptor."

At this point, the cancer is no longer dependent on estrogen but is fed by



a new set of proteins associated with the CXCR4 receptor. Because these are not hormones, anti-estrogen drugs cannot block them.

"Expression of CXCR4 in an active form increases the growth rate and metastatic capacity of the <u>breast cancer</u> cells. Therefore this receptor represents a target for testing potential <u>cancer drugs</u>," said Ann Richmond, Vanderbilt University School of Medicine and VA Medical Center.

"There are agents that target the CXCR4 receptor that have been used in other types of disease," says Burow. "Our study shows that certain agents can reverse some of the effects of CXCR4, that is, we can reverse resistance to endocrine therapy--that is exciting."

Provided by Tulane University

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