

Regulator of gene expression responsible for the progression of breast cancer

28 February 2014, by Bill Hathaway



Credit: Shutterstock

The authors say their evidence suggests that RBP2 regulates a critical epigenetic switch that sets the stage for [tumor metastasis](#). They say the enzyme offers a novel target for development of therapies designed to inhibit [tumor progression](#) and metastasis.

"Metastasis is the major cause of breast cancer-related death," said senior author Qin Yan, assistant professor of pathology at Yale School of Medicine. "Our study provides the first evidence, in genetically engineered mice, that a new class of enzymes could be targeted to suppress tumor metastasis."

Provided by Yale University

Yale Cancer Center researchers have identified a regulator of gene expression that is responsible for the progression of breast cancer and its metastasis to the lung. The study appears online in *Cell Reports*.

In women, breast cancer is the most common cancer, and the second leading cause of cancer-related death. When it metastasizes, it does so primarily to the lung, brain, and bone. Only limited treatment options are available, and scientists are working to identify and test new drug targets for the development of effective therapies.

Recent studies suggest that abnormal gene expression contributes significantly to tumor formation and progression. But the regulators of such changes in metastasis are poorly understood.

The Yale researchers analyzed [gene expression](#) datasets of human breast tumors, as well as those of cancer cells, and found that overexpression of the enzyme RBP2 is critical for [breast cancer metastasis](#) to the lung. Loss of RBP2, they also found, suppressed [tumor formation](#) in mouse models.

APA citation: Regulator of gene expression responsible for the progression of breast cancer (2014, February 28) retrieved 19 April 2021 from <https://medicalxpress.com/news/2014-02-gene-responsible-breast-cancer.html>

This document is subject to copyright. Apart from any fair dealing for the purpose of private study or research, no part may be reproduced without the written permission. The content is provided for information purposes only.