

New cell mechanism discovery key to stopping breast cancer metastasis

January 2 2014

Researchers from Huntsman Cancer Institute (HCI) at the University of Utah discovered a cellular mechanism that drives the spread of breast cancer to other parts of the body (metastasis), as well as a therapy which blocks that mechanism. The research results were published online in the journal *Cell Reports* on January 2.

"Genetic mutations do not drive this mechanism," said Alana Welm, PhD, senior author of the study, associate professor in the Department of Oncological Sciences, and an investigator at Huntsman Cancer Institute. "Instead, it's improper regulation of when genes turn on and off."

The new discovery focuses on a protein called RON kinase (RON), which signals some areas of tumor cell DNA to become active. Normally, RON operates mostly during embryonic development and is not highly expressed in healthy adults. But in about 50 percent of [breast cancer](#) cases, RON becomes re-expressed and reprograms genes responsible for metastasis, making them active.

"If there's an entire program in the tumor cell that's important for metastasis, blocking one small part of that program, for example, the action of a single gene, will probably not be an effective strategy," said Welm. "But if you could find a way to turn off the entire program, you're more likely to have the desired effect. We found that inhibiting RON turns off the entire metastasis program in these tumor cells."

"No one has ever described a specific pathway driving this kind of reprogramming in [metastasis](#), much less a way to therapeutically block it,' Welm added. "Also, RON has not previously been known to be involved in reprogramming gene expression."

Future work will include investigating the potential of detecting the RON-dependent program in [tumor cells](#) as a way to identify patients that are more likely to develop metastases and as a predictor of therapeutic response to drugs that inhibit RON.

Provided by University of Utah Health Sciences

Citation: New cell mechanism discovery key to stopping breast cancer metastasis (2014, January 2) retrieved 16 July 2023 from <https://medicalxpress.com/news/2014-01-cell-mechanism-discovery-key-breast.html>

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