

Researchers unlock how progesterone increases breast cancer risk

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Researchers have identified how the hormones progesterone and estrogen interact to increase cell growth in normal mammary cells and mammary cancers, a novel finding that may explain why postmenopausal women receiving hormone replacement therapy with estrogen plus progestin are at increased risk of breast cancer.

The discovery that both [estrogen](#) and progesterone must be present for the increased production of the protein amphiregulin, which binds to mammary cells and promotes cell growth, could lead to new treatment methods for the disease, said Sandra Haslam, director of Michigan State University's Breast Cancer and the Environment Research Center and lead researcher on the project.

The study, funded by the Department of Defense's Breast Cancer Research Program and published in *Hormones and Cancer*, looked at why progesterone combined with estrogen may contribute to increased breast cancer risk. In the study, researchers used both the native [hormone](#), progesterone, and a [synthetic compound](#), progestin – obtaining the same results.

The finding might help explain earlier results from the groundbreaking Women's Health Initiative showing the risk of breast cancer is significantly greater for [postmenopausal women](#) who received [hormone replacement therapy](#) with combined estrogen plus progestin compared to women receiving estrogen alone.

"Also, breast cancers that develop in women receiving estrogen plus progestin are more invasive and deadlier," Haslam said. "What is the progestin doing to increase the risk of tumor growth?"

Along with co-investigator Anastasia Kariagina, a colleague in the College of Human Medicine and Department of Physiology, Haslam identified the [protein](#) amphiregulin and its receptor as one potential culprit.

"Amphiregulin – acting through its receptor, epidermal growth factor receptor – along with progesterone leads to the activation of intracellular pathways that regulate cell growth," Haslam said. "When activated, this promotes normal cell growth and the growth of tumors."

The study was performed in rats because breast cancers in rats contain receptors for estrogen and progesterone – similar to the human breast – and tumor growth is hormone-dependent, as are the majority of human breast cancers. The research team also confirmed the same phenomenon in human breast cancer cell cultures.

In addition, the research team found that Iressa, a cancer drug that blocks the epidermal growth factor receptor, effectively stopped the proliferation caused by amphiregulin. While those studies were done only in cell cultures and not on tumors growing in animals, the results are promising, Haslam said.

"The results indicate that the interactions between estrogen, progesterone and epidermal growth factor receptor pathways may be considered relevant targets for the treatment of hormone-dependent breast cancers," she said. "This may be especially important in premenopausal breast cancer because women produce their own estrogen and [progesterone](#)."

"A combined approach of inhibiting both the hormones and the

epidermal growth factor receptor may be beneficial for some women in treating hormone-dependent [breast cancer](#)."

More information: The article can be viewed at [www.springerlink.com/content/2 ... 5833472/fulltext.pdf](http://www.springerlink.com/content/2...5833472/fulltext.pdf)

Provided by Michigan State University

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