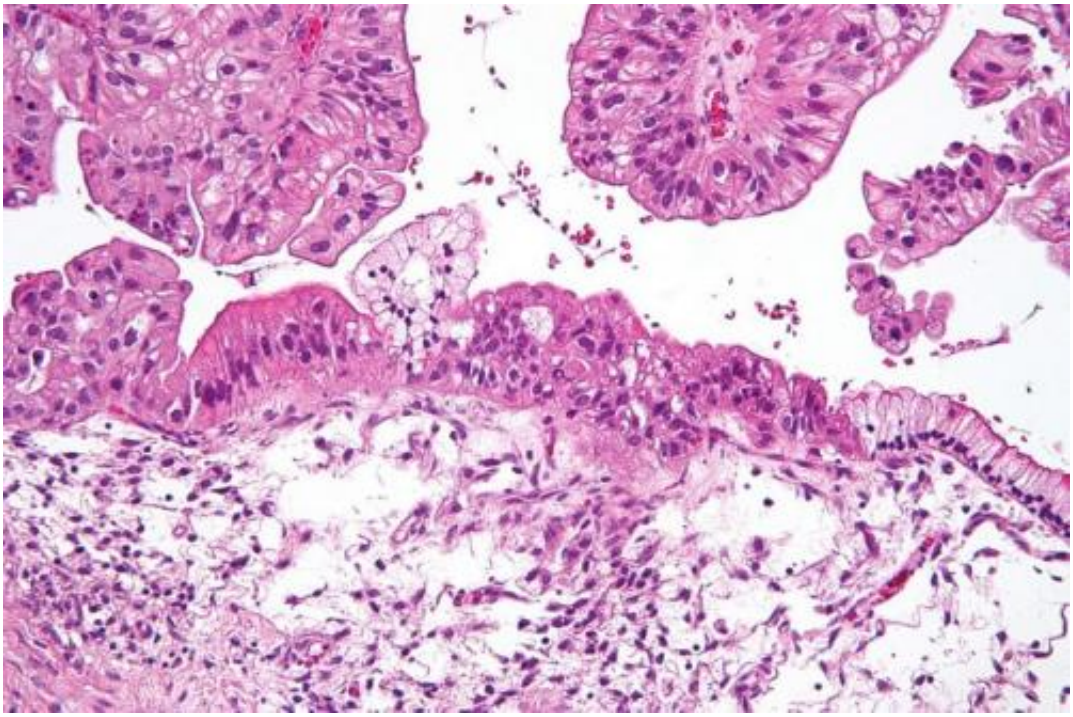
Changes in gene contribute independently to breast and ovarian cancer

January 31 2017



Intermediate magnification micrograph of a low malignant potential (LMP) mucinous ovarian tumour. H&E stain. The micrograph shows: Simple mucinous epithelium (right) and mucinous epithelium that pseudo-stratifies (left - diagnostic of a LMP tumour). Epithelium in a frond-like architecture is seen at the top of image. Credit: Nephron /Wikipedia. CC BY-SA 3.0

Defects in a key gene - long thought to drive cancer by turning off the protection afforded by the well-known BRCA genes - spur cancer growth on their own, according to a study led by researchers from NYU

Langone Medical Center.

The study gene, known as EMSY, has some of the same functions as BRCA1 and BRCA2, which are known to protect against breast and ovarian cancer when normal. When defective, BRCA genes block the body's self-defense against cancer-causing genetic mistakes.

The new study, published online Jan. 13 in *Oncotarget*, helps to explain why some women with healthy BRCA1 and BRCA2 genes develop cancer. The findings may also expand treatment options for the roughly 11 percent of women with breast and ovarian cancer and normal BRCA genes, say the study authors.

"Now that we know exactly how changes in EMSY spur cancer cell growth, we can start to design therapies to specifically target that activity and hopefully stop it," says senior author Douglas Levine, MD, director of the Division of Gynecologic Oncology at NYU Langone and its Perlmutter Cancer Center.

"This work also suggests that treatments that work for patients with BRCA1 or BRCA2 mutations might also be effective against EMSY-driven cancers because the disease mechanism is similar," says first study author Petar Jelinic, PhD, a research assistant professor at NYU Langone. "The best way to go rapidly from bench to bedside is to find new ways to use existing treatments."

When normal, EMSY, BRCA1 and BRCA2 give the body's cells instructions to create proteins that help to repair DNA damage that can cause cancer. When those genes are altered, the repair process fails and cancer grows. Overly active EMSY, like mutated BRCA1 or BRCA2, changes those instructions, so that the DNA damage repair process is blocked.

This new study dispels prior theories that EMSY's activation merely turned off the cancer suppression function of BRCA2, says Jelinic.

Earlier work by Levine and others pointed toward EMSY activation as a culprit in breast and ovarian cancer, but had only examined certain parts of the EMSY protein. The new study was the first to evaluate the full-length EMSY protein and to show that it acts independently of BRCA1 or BRCA2.

Furthermore, the research revealed the part of the EMSY protein is changed by an enzyme called protein kinase A. When there is more active EMSY than normal, this enzyme reacts with the EMSY protein to more thoroughly suppress the DNA repair process.

Breast cancer is the second most common cancer among women in the United States, after skin cancer. Ovarian cancer is the fifth leading cause of cancer death among women, according to the National Cancer Institute.

Provided by New York University School of Medicine

Citation: Changes in gene contribute independently to breast and ovarian cancer (2017, January 31) retrieved 25 April 2024 from https://medicalxpress.com/news/2017-01-gene-contribute-independently-breast-ovarian.html