

Researchers discover that same gene has opposite effects in prostate, breast cancers

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Researchers at Cleveland Clinic have discovered that a gene – known as an androgen receptor (AR) – is found in both prostate and breast cancers yet has opposite effects on these diseases.

In prostate cancer, the AR gene promotes cancer growth when the gene is "turned on." In breast cancer, the AR gene promotes cancer growth when the gene is "turned off," as is often the case after menopause, when AR production ceases in women.

What this means is that treating prostate and breast cancers require completely opposite approaches to AR. In treating prostate cancer, the strategy should be to block AR; in breast cancer, the strategy should be to support AR production.

Researchers from Cleveland Clinic's Lerner Research Institute, including Charis Eng, M.D., Ph.D., Chair, Genomic Medicine Institute; Robert Silverman, Ph.D., and Warren Heston, Ph.D., both of the Department of Cancer Biology; focused on whether the androgen receptor (AR) molecule offers evidence of the tumor suppressor protein PTEN. The research discovered that AR inhibits PTEN expression in prostate cancer cells, but stimulates it in breast cancer cells.

The conclusions, published in the Oct. 21, 2011 issue of *Oncogene*, explain why prostate cancer progression is associated with increased AR expression (and a common prostate cancer treatment strategy involves blocking AR), while most breast cancers occur post-menopause, after

AR production has ceased (making AR supplementation a strategy for treating breast cancer).

Dr. Eng and her colleagues have mapped the interaction between AR and PTEN in both prostate and [breast cancer](#) cells, which suggests that this interaction activates or represses subsequent gene expression depending on organ-specific cofactors. Although PTEN is a known tumor suppressor, and loss of PTEN expression has been associated with numerous cancers (including breast and prostate cancers), its regulation has not been well understood. The current data provide new information regarding PTEN regulation, and suggest that identifying regulatory cofactors will be a valuable next step in determining cancer risk, as well as potential new therapies.

"We now see how androgen affects PTEN expression – and ultimately cancer," said Dr. Eng. "Our observations help explain why this prostate cancer risk can be halved by drinking red wine, which increases PTEN expression. Our data also suggest that treatment of the exact same cancer must be personalized for males and for females."

Provided by Cleveland Clinic

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