

Closing in on Breast Cancer

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Columbia oncologists have demonstrated for the first time how a mutated gene associated with breast cancer actually contributes to runaway cell growth, a finding that could lead to new treatments. Scientists first linked the gene BRCA1 to breast cancer in the 1990s, but until now they haven't understood how it works.

The oncologists, led by Columbia professor Ramon Parsons, showed recently that BRCA1, whose job is to fix routine DNA damage in other genes, often fails to repair damage in a gene called PTEN when BRCA1 itself mutates. The gene PTEN, which Parsons discovered in 1997, is known to increase dangerously the activity of proteins that trigger cell growth.

"Ever since the link was established between BRCA1 and breast cancer, we've been frustrated by our lack of understanding about how its mutations work," says Parsons, who directs the Avon Foundation Breast Cancer Research Laboratory and the Breast Cancer Program of the Herbert Irving Comprehensive Cancer Center at Columbia University Medical Center. "Now that we know it acts on PTEN, we have a target for therapy."

Breast cancers associated with the BRCA1 gene account for about 10 percent of all cases and tend to be aggressive and tricky to diagnose. Parsons estimates that PTEN will be found to be involved in about half of BRCA1 breast cancers once a complete chromosomal analysis is done.

Columbia oncology student Lao Saal '07GSAS, '09PS was lead author of the paper, which appeared in the January issue of Nature Genetics. Other collaborators included the oncologist Åke Borg at Sweden's Lund University.



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