

# News Release

**EMBARGOED:** Hold for release until Thursday, August 22, 2013 at 7:01 p.m. EDT

**MULTIMEDIA ALERT:** For audio and video of Sahra Borges, Ph.D., talking about the study, visit the <u>Mayo</u> <u>Clinic News Network</u>. 4500 San Pablo Road Jacksonville, Florida 32224

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#### Drug Used for Blood Cancers May Stop Spread of Breast Cancer Cells, Mayo Clinic Finds

JACKSONVILLE, Fla. — A drug used to treat blood cancers may also stop the spread of invasive <u>breast</u> <u>cancer</u>, <u>researchers</u> at <u>Mayo Clinic in Florida</u> have discovered. Their study, published online in <u>Breast</u> <u>Cancer Research</u>, found that in the lab and in animals, the drug decitabine turns on a gene coding for protein kinase D1 (PRKD1) that halts the ability of cancer cells to separate from a tumor and spread to distant organs.

"Treatment with low doses of decitabine in an animal model of breast cancer restored PRKD1 expression, reduced tumor size, and blocked metastasis to the lung," says the study's senior investigator, <u>Peter Storz, Ph.D.</u>, a <u>biochemist and molecular biologist</u> at Mayo Clinic in Florida.

"The outcome of patients with invasive breast cancer is less than optimal despite many attempts to improve treatment, including advanced chemotherapy and hormonal therapy," says Dr. Storz. "We hope this study offers a new avenue to prevent breast cancer from becoming aggressive and untreatable."

The <u>research</u> team, which includes first author Sahra Borges, Ph.D., a postdoctoral researcher in Dr. Storz's lab, found that the gene coding for PRKD1 was silenced in all but one subtype of invasive breast cancer, including aggressive triple negative breast cancer. That subtype is invasive lobular carcinoma.

Dr. Borges also developed an assay that can be used to measure the amount of PRKD1 that is silenced in patients' breast tumors.

"Because we found that PRKD1 is increasingly silenced as breast cancer becomes aggressive and spreads, the hope is that this test can be further developed and used to predict which patients are at risk for cancer metastasis, and thus may benefit from decitabine," Dr. Borges says.

Decitabine, approved by the <u>U.S. Food and Drug Administration</u> for use in some <u>blood cancers</u>, is a demethylating agent, meaning that it can switch on beneficial genes such as PRKD1 that cancer has silenced in order to grow.

## Drug Used for Blood Cancers May Stop Spread of Breast Cancer Cells, Mayo Clinic Finds - page 2

Treating genes that are silenced is much easier than trying to restore function of a mutated gene, Dr. Storz says. The normal function of PRKD1, which is expressed in cells of the mammary gland, is to maintain normal function by preventing cells from morphing into a state where they can dislodge and spread, he says.

The researchers hope this study will help them to design a clinical trial in collaboration with Mayo Clinic physicians using decitabine to promote re-expression of PRKD1 and agents that activate PRKD1.

The study's other key researchers at Mayo include Heike Doeppler, <u>Edith Perez, M.D.</u>, Cathy Andorfer, Ph.D., <u>Zhifu Sun, M.D.</u>, <u>Panos Anastasiadis, Ph.D.</u>, <u>E. Aubrey Thompson, Ph.D.</u>, and <u>Xochiquetzal J. Geiger, M.D.</u> The study was supported by the <u>National Institutes of Health</u> (GM086435), the Bankhead-Coley Program of the Florida Department of Health, <u>Mayo Clinic Breast Cancer SPORE</u>, Breast Cancer Foundation, and from the <u>26.2 with Donna Foundation</u>.

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#### About Mayo Clinic Cancer Center

As a leading institution funded by the National Cancer Institute, <u>Mayo Clinic Cancer Center</u> conducts basic, clinical and population science research, translating discoveries into improved methods for prevention, diagnosis, prognosis and therapy. For information on cancer clinical trials, call 507-538-7623.

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