

# Researcher examines link between cancer, Down syndrome

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There's new hope for breast cancer research, and it's coming from a very unlikely place. Researchers at the Texas A&M University College of Veterinary Medicine & Biomedical Sciences recently published articles in the journals *Molecular and Cellular Biology* and *Carcinogenesis* indicating that a protein long suspected to play a role in Down Syndrome may also contribute to treating this devastating disease.

It has long been known that Down Syndrome is caused when an individual has an extra copy of the 21st chromosome, giving them a total of three instead of the normal chromosome pair. With improved medical care, people with Down Syndrome are now living longer, healthier lives. With this advance came the observation that individuals with Down Syndrome have a significant decrease in risk for several types of tumors. Most striking is the observation that women with Down Syndrome are 10-25 times less likely to develop breast cancer.

This effect is thought to be due to the presence of one or more "tumor suppressor" genes on chromosome 21. However, the identity of such genes has not been known, until now.

"Years of research into the genetics of Down Syndrome have helped us to discover a very important gene on chromosome 21," said Dr. Weston Porter, associate professor in the Veterinary Integrative Biosciences Department. "This gene, called Single-minded 2 or SIM2 is thought to play an important role in Down Syndrome by regulating neuron growth in the developing brain. Based on its developmental role, we

hypothesized that SIM2 may also be involved in breast cancer, which is essentially a disease of uncontrolled growth.”

For the last five years, Porter and his colleagues, Richard Metz, Brian Laffin and Elizabeth Wellberg, have been using human breast cells and mouse models as part of a research grant from the National Institutes of Health to validate this hypothesis, and what they have found they consider very promising. SIM2 is lost or suppressed in a majority of human breast tumors, and deletion of the SIM2 gene triggers rapid tumor growth in mice.

However, the process by which SIM2 suppresses breast cancer is complex and not fully understood. This same protein which may hold so much promise for breast cancer treatment is also thought to contribute to the negative effects of Down Syndrome.

“As we move forward,” said Porter, “it will be important for us to understand the circuit of SIM2 and how it is turned on and off. In light of the available data on breast cancer incidence in the Down Syndrome population and our experimental data, knowing how to turn SIM2 expression on and off and identification of down-stream targets should have great therapeutic value.”

While still in the early stages, this research represents a promising weapon in the fight against breast cancer as it sheds light on a previously unknown target for which to shoot.

“What we are seeing now is a paradigm shift in breast cancer research” said Porter. “For years we have gone after the wrong kinds of cells. It was all about getting rid of the tumor itself. This has led to a dandelion effect where, we didn’t get to the root and the cancers kept coming back and spreading. Now we’re looking at ways to get to the root of breast cancer, and not simply shrinking the tumor to come back another day.”

While it may be years before their research results in a definitive treatment or cure, Weston says it is impacting our approach towards understanding breast cancer today.

Source: Texas A&M University

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