Science Buzz!
Targeting Obesity: A window of opportunity in breast cancer prevention

Grantees from left to right: Elizabeth Wellberg, Ph.D; Pepper Schedin, Ph.D; Paul MacLean, Ph.D; Steven Anderson, Ph.D; Ann Thor, Ph.D, Erin Giles, Ph.D

Numerous studies have shown a strong link between obesity and breast cancer risk, but the complex mechanisms behind this link are not fully understood. However, recent studies by Komen-funded researchers have begun to connect the dots between obesity and breast cancer risk and have identified a potential window of opportunity to reduce this risk.

A team of researchers at the University of Colorado Cancer Center and the Anschutz Health and Wellness Center, which includes Komen grantees Steve Anderson, Paul MacLean, Anne Thor, and Pepper Schedin, has been studying how obesity during perimenopause – the time around menopause – may affect breast cancer risk. Studies show that weight gain after menopause increases the risk of breast cancer, but little is known about how body weight just prior to menopause affects that risk. Results from ongoing studies, supported in part by a Komen grant to Dr. Anderson, suggest that in some high-risk women, being overweight just prior to menopause may increase the risk of breast cancer. These studies also suggest that early intervention with weight control or medication during this time may prevent or slow breast cancer growth.

To study this question, the University of Colorado research team designed a unique animal model to mimic menopause, breast cancer, and obesity. In work published in 2010 in the journal Obesity, they showed that breast tumors grew much faster in overweight rats during menopause, than in normal weight rats. In a follow-up article in Cancer Research in 2012, the research team found that overeating immediately after menopause caused tumors to grow even faster in the obese rats, but not in the lean rats. The researchers believe this occurs because obese rats stored excess calories from overeating in the breast tumor, providing it with food and energy to grow. In contrast, the lean rats responded normally to overeating by storing the excess calories in the liver, muscle and healthy fat tissue.

Animal models designed to mimic human diseases allow scientists to study complicated questions in human biology that cannot be done in humans.

The researchers also found that the tumors in obese rats had higher levels of a molecule called progesterone receptor (PR) compared to normal weight rats. Interestingly, the increase in PR was linked to changes in genes that control energy metabolism and cell growth. When the obese rats were given metformin - an anti-diabetic drug that blocks obesity-related disorders – tumor growth significantly decreased and PR levels also went down. "We are excited about the potential use of metformin, an FDA-approved, well tolerated drug for short-duration chemoprevention", says Anderson.

These studies indicate that PR may play an important, and previously unrecognized, role in obesity-induced breast cancer after menopause. Knowing this can inform doctors about how best to manage a woman's future risk. They also suggest that a woman's body weight just prior to menopause may be an important predictor of her future risk of breast cancer when other risk factors are present. Knowing this could identify women who might benefit from weight loss or medication, such as metformin, for prevention of future breast cancer.

Learn more about progesterone and estrogen receptor and breast cancer here.

March, 2013