Do Beta-Blockers Also Block Ovarian Cancer?

By Josh Bloom — August 24, 2015

In today's "you never know what is around the corner" department, a surprising story out of M.D. Anderson Cancer Center in Dallas reports that beta-blockers heart drugs that are used to lower blood pressure and slow the heart may have another use. That would be reducing the toll of ovarian cancer, which is one of the hardest types to treat.

While there appears to be a significant effect, this is not a magic bullet. But, given the poor treatment options available for treating the disease, this study may shed light on one of the mechanisms of cancer growth.

There are three main classes of beta-receptors: beta-1 (primarily in the heart), beta-2, (smooth muscle cells, especially in lungs and uterus) and beta-3 (fat cells). Each main receptor has a number of subtypes. Beta-3 receptor agonists (activators) were the focus of an enormous effort as potential antiobesity drugs in the pharmaceutical industry in the 1990s. While these drugs were effective in causing dramatic weight loss and lowering of blood glucose in genetically obese mice and rats, all of them bombed in the clinic, including what would later become the gold standard CL-316243 [1], which came out of my lab at Wyeth.

Not only are there different classes of beta-blockers, but there are different drugs within each class. Some work better than others in prolonging life. My 2012 op-ed [2] in New Scientist discussed this. Some beta-blockers are non-selective (they interact with more than one type of beta-receptor), and some are selective, interacting with only one. The selective class is usually preferred in treatment of heart disease because of fewer side effects.

The most interesting aspect of the Anderson study was that the type of beta-agonist that the women took for cardiovascular disease strongly determined the apparent impact on their cancer.

The Anderson study, which appears [3] in the current issue of the American Cancer Society's journal Cancer, used a retrospective analysis of 1,425 women with ovarian cancer, who were treated between 2000 and 2010. As a group, women who took selective beta-blockers had a median survival time following diagnosis of 48 months vs. 42 for women who took no beta-blockers of any kind.

But, for that group of women who took non-selective beta-blockers, that median survival time jumped to 90 months. This apparent increased survival is not only clinically significant, if confirmed by other studies (since being a retrospective study, no "cause-and-effect" conclusions can be drawn), but also provides some very interesting clues about one of the mechanisms of cancer cell growth. The author thinks that stress hormones may fuel ovarian cancer growth, and beta-blockers tend to suppress those same hormones.

Dr. Anil Sood, M.D., professor in Gynecologic Medical Oncology and Cancer Biology at Anderson,
said, "the ability to show improved survival using nonselective agents which inhibit a specific stress pathway is the culmination of years of research into ovarian cancer biology and pathogenesis."

It stands to reason that the next step should be a randomized double-blind prospective trial of women with ovarian cancer, some of whom will receive non-selective beta-blockers, while some of whom will receive selective beta-blockers (as compared to no such therapy) over the course of several years.

The migration away from cytotoxic drugs to treat cancer and toward those that target specific growth pathways continues to go strong after two decades. Findings like this will continue to help redefine what cancer actually is, and bring us closer to the holy grail of cancer therapy more effective and less toxic drugs.

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