

Could a Cancer Vaccine Developed Long Ago Hold the Key to Long-Term Survival?

New research shows a decades-old breast cancer vaccine may have unlocked a powerful immune memory response, now supercharged by a new antibody.

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DURHAM, N.C. – A small group of women with advanced breast cancer received a vaccine via a clinical trial more than 20 years ago. Today, they're all still alive. Scientists say that kind of long-term survival is almost unheard of for patients with metastatic breast cancer, and it's what caught the attention of researchers now.

Scientists at Duke Health studied the immune systems of the women in that trial, which was led by [Herbert Kim Lyerly, MD](#), George Barth Geller Distinguished Professor of Immunology at [Duke University School of Medicine](#). They found something remarkable: The women still had strong, long-lasting immune cells that recognized their cancer.

These cells carried a special marker, called CD27, which helps the immune system remember and fight off threats like cancer. The findings, published in *Science Immunology* October 28, suggest that targeting CD27 could dramatically improve the effectiveness of cancer vaccines.

"We were stunned to see such durable immune responses so many years later," said [Zachary Hartman, PhD](#), senior author of the study and associate professor in the Departments of [Surgery](#), [Integrative Immunology](#) and [Pathology](#) at Duke University School of Medicine. "It made us ask: What if we could boost this response even more?"

To find out, the team tested a CD27-targeting stimulatory antibody in combination with a vaccine targeting HER2 (a protein on the surface of some cells, including breast cancer) across different mouse models. They found nearly 40% of mice treated with the combination therapy experienced complete tumor regression, compared to just 6% with the vaccine alone.

The researchers found that the antibody worked by supercharging a type of immune cell called CD4+ T cells.

Hartman said these "helper" CD4+ cells are often overlooked in cancer research, which tends to

focus on CD8+ “killer” T cells. Hartman said the study indicates the CD4+ cells are playing a starring role in this application, driving long-term immune memory and helping other immune cells do their job better.

Moreover, the subsequent addition of a different antibody that helps CD8+ T cells further improved tumor rejection rates in mice to nearly 90%.

“This study really shifts our thinking,” Hartman said. “It shows that CD4+ T cells aren’t just supporting actors; they can be powerful cancer fighters in their own right and are possibly essential for truly effective anti-tumor responses.”

The team also found the CD27 antibody only needed to be given once, at the same time as the vaccine, to have a lasting effect. This could make it easier to combine with existing cancer treatments, including immune checkpoint inhibitors and antibody-drug conjugates already used in patients.

Hartman believes this approach could help unlock the full potential of cancer vaccines.

“We’ve known for a long time that vaccines can work against cancer, but they haven’t lived up to the hype,” he said. “This could be a missing piece of the puzzle.”

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