Breast Cancer Hijacks the Lung Repair System to Fuel Tumor Growth, Study Finds

Colorado University research shows how breast cancer cells may lead to lung metastases. A repurposed drug may slow that process.

April 13, 2026 By University of Colorado Cancer Center and Laura Kelley

---

Researchers at the CU Anschutz Cancer Center have discovered how breast cancer cells that spread to the lungs may take advantage of the body's natural healing response and how a commonly used drug might slow that process. They found that breast cancer cells that spread to the lungs trigger the lung's normal repair system, creating conditions that unintentionally help tumors grow.

The study was published this month in Cancer Research Communications.

Key Findings

- Breast cancer cells can trigger the lung's natural healing response.
- Tumors have developed ways to utilize that response to facilitate growth.
- An existing anti-inflammatory drug slowed outgrowth of breast cancer lung metastases in mice.
- Data from breast cancer patient samples indicate that targeting these processes may be a promising therapeutic strategy.

How Cancer Uses the Lung’s Healing System

When breast cancer spreads to the lungs, it damages tiny air sacs needed for people to breathe. Normally the lung would quickly work to repair this damage.

Researchers found that cancer cells prolong the damage repair mode leading to chronic inflammation.

Specialized lung cells called alveolar type II cells normally help rebuild lung tissue after injury. When cancer cells are present, these lung cells begin releasing signals that encourage tumors to grow.

“The lung is doing what it is designed to do and that is to clear debris and repair damage,” said Jessica L. Christenson, PhD, first author of the study and an instructor in the Department of Pathology at CU Anschutz. “But in this case, cancer cells are taking advantage of that repair response.”

A Cycle That Helps Tumors Grow

The researchers found that cancer cells and lung cells begin signaling to each other.

Cancer cells activate lung cells which then release substances that fuel cancer growth. This eventually allows tumors to expand.

A Possible New Way to Slow Metastatic Cancer

The team tested roflumilast, a medication approved to treat Chronic Obstructive Pulmonary Disease (COPD).

In mouse models of metastatic breast cancer, the drug slowed tumor cell growth and reduced the size of tumors in the lungs. Rather than killing cancer cells, the drug prevented the lung environment from helping the tumors grow.

“This suggests a new strategy for treating metastatic cancer,” said Jennifer K. Richer, PhD, senior author of the study and professor of pathology at the CU Anschutz Cancer Center. “In addition to targeting cancer cells themselves, we may also be able to target the environment that allows them to thrive.”

Why This Matters

Breast cancer is more difficult to treat once it spreads to other parts of the body. It often spreads

to the lungs.

About one third of people with metastatic breast cancer develop lung metastases, and treatment options are limited.

Because roflumilast is already approved for another condition, it may be possible to study its potential use in metastatic cancer more quickly.

What Comes Next

Researchers plan to study how this drug might work together with chemotherapy or immunotherapy and whether it can effectively alleviate common side effects of cancer treatment. They are also exploring inhaled versions of similar drugs that could deliver treatment directly to the lungs.

“We are very excited to translate these findings to the clinic and evaluate roflumilast as a treatment for patients with triple-negative breast cancer to prevent recurrence in the lungs,” said Jennifer R. Diamond, MD, a professor of medical oncology and medical director of the Cancer Clinical Trials Office (CCTO) at the CU Anschutz Cancer Center and close collaborator on this project.

The study was supported by the National Institutes of Health, University of Colorado Cancer Center, American Cancer Society and METAvivor.

This article was originally published March 25, 2026, by the University of Colorado Cancer Center. It is republished with permission.
