

Experimental Treatment Uses Engineered Fat Cells to “Starve” Tumors

When implanted into mice, engineered fat cells consumed nutrients that tumors need, slowing their growth.

April 9, 2025 By [National Cancer Institute](#)

Scientists have long been exploring ways to kill cancer cells by starving them of the nutrients they need to survive. A new study suggests that [genetically modified](#) fat cells could help researchers realize this goal.

In the study, researchers genetically engineered white fat cells—the most common type of fat in the body—to aggressively consume nutrients such as [glucose](#) and [fatty acids](#). When the engineered fat cells were implanted near tumors in mice, the [tumors grew more slowly than tumors in mice without the engineered cells](#).

The approach slowed the growth of cancer in mice even when the engineered fat cells were implanted far from a tumor, the researchers reported in *Nature Biotechnology* on February 4.

“We believe the engineered cells are outcompeting tumors for essential nutrients, suppressing the proliferation of cancer cells,” said study leader Nadav Ahituv, Ph.D., director of the University of California, San Francisco (UCSF) Institute for Human Genetics. “The findings suggest that engineered fat cells could be a new form of cellular therapy.”

Existing cellular therapies for cancer, such as [CAR T-cell therapy](#) and [TIL therapy](#), are made using a patient’s own [immune cells](#). These cells are collected from a patient, engineered to attack cancer cells, and returned to the patient.

The new treatment, which the researchers call adipose manipulation transplantation (AMT), may be the first experimental cellular therapy for cancer to collect and modify a patient’s own fat cells.

“The use of genetically modified fat cells is an innovative and promising therapeutic strategy that deserves further study,” said Jung Byun, Ph.D., of NCI’s Division of Cancer Treatment and Diagnosis, who is an expert in genetic engineering but was not involved in the research.

In the NCI-supported study, Dr. Ahituv and his colleagues used a [gene](#)-editing tool called CRISPRa to turn ordinary white fat cells, whose main function is to store energy, into energy-consuming beige fat cells. When these cells were implanted into mouse models of breast, pancreatic, colon, and prostate cancer, tumor growth in the mice slowed, the researchers reported.

They also were able to customize how they engineered the fat cells so the cells would consume specific energy sources. For example, the researchers engineered fat cells to outcompete pancreatic tumors for a molecule these cancer cells need called uridine.

“Conventional cancer therapies such as chemotherapy and radiation primarily focus on killing cancer cells directly,” Dr. Byun said. “In contrast, AMT offers a nontoxic treatment approach that disrupts tumor growth by strategically outcompeting cancer cells for essential nutrients.”

Potential use of fat cells in developing therapies

The human body has two main types of fat. White fat, which is more common, stores energy and can contribute to conditions such as obesity, while brown fat—sometimes called “good fat”—stores energy and burns that energy to help the body maintain a stable temperature.

When the body is cold, brown fat cells are activated to use glucose, fat, and [amino acids](#) to generate heat. The fat cells have an abundance of energy-producing structures known as [mitochondria](#), and the cells’ brown color comes from the iron found in mitochondria.

Dr. Ahituv and his postdoctoral fellow at the time, Hai Nguyen, Ph.D., were inspired to develop AMT by a study showing that [cold temperatures slowed tumor growth in mice](#). In that study, the cold caused brown fat cells to be activated and consume large amounts of glucose, a critical fuel for cancer cells, the researchers concluded.

Intrigued by those results, Drs. Ahituv and Nguyen set out to develop a cancer therapy based on fat cells that did not require cold temperatures. They chose to work with white fat in part because brown fat, which is primarily found around the shoulders, becomes less active as people age.

“When it comes to developing therapies for diseases such as cancer, fat cells have generally been overlooked,” Dr. Ahituv said. But these cells, he continued, have attributes that make them strong candidates for use in therapies.

For example, fat cells can secrete [hormones](#) and other substances that could help treat a disease. And fat cells that are collected from a person, modified, and returned back to that individual are unlikely to trigger an immune response.

Turning white fat cells into beige fat cells

To turn human white fat cells into voracious consumers of nutrients, the researchers modified a

gene involved in energy production in the cells. The gene, UCP1, is active in brown fat cells but typically dormant in white fat cells. The genetic change turns the white fat cells into beige fat cells.

In their initial experiment, the researchers grew human engineered fat cells and human cancer cells in a special type of petri dish. The fat cells and the cancer cells were in separate compartments, but they shared nutrients such as glucose.

By the end of the experiment, the tumor cells were so depleted that the researchers thought they had made an error. “We repeated the experiment a number of times and kept getting the same result,” Dr. Ahituv said. “We were extremely excited.”

When his team moved on to do experiments using three-dimensional [tumor models](#) known as organoids and then in mice, the engineered fat cells continued to perform as the researchers had hoped, regardless of the type of cancer.

The engineered cells behaved like “energy vacuums,” taking away the fuel that cancer cells in the tumors need to survive, explained another investigator on the study team, Jennifer Rosenbluth, M.D., Ph.D., of the UCSF Helen Diller Family Comprehensive Cancer Center.

“AMT seemed to reduce tumor growth in essentially all of the tumor models that we tested,” Dr. Rosenbluth added.

The exception was mice that were fed high-fat or high-glucose diets. The treatment did not work as well on mice fed these diets as on mice fed balanced diets. This finding, Dr. Ahituv said, supports the idea that it is competition for nutrients that caused the tumors in the mice to shrink.

“When nutrients are plentiful in the tumor environment, it might not be possible for engineered fat cells to outcompete tumor cells for these resources,” he said.

Delivering the engineered cells into the body

A team led by Tejal Desai, Ph.D., a biomedical engineer and researcher at Brown University, created scaffolds to “house” the engineered fat cells so that they could be implanted into the mice in a controlled manner and then retrieved as needed.

“The key to developing these constructs was using a material that supports the functions of the cells and is biocompatible with the body,” said Dr. Desai, who also leads a research team at UCSF.

For the study authors, demonstrating that it was possible to pause AMT by removing the implanted fat cells from the body was an important part of the study. Doctors might need to turn off AMT if a person were to develop unforeseen clinical complications from the therapy, they said.

Unanswered questions about AMT

Before the treatment can be tested in people, Dr. Ahituv cautioned, more work is needed to better understand and improve the engineered cells. His team plans to study engineered cells in which the activity of multiple genes is increased rather than just a single gene.

The researchers would also like to learn more about the mechanisms underlying the results in their study. Although competition for nutrients appeared to be the main way that AMT slowed the progression of cancer, there might be other factors as well.

“It’s possible that the engineered fat cells could be just improving the whole [metabolic](#) program of the animal,” Dr. Ahituv said.

Further research is needed to explore other questions about AMT.

It is not known, for instance, how long the engineered fat cells might remain active in the human body and, thus, how long their tumor-suppressive effects could last. Another question is whether cancer cells could become resistant to AMT by developing alternative ways to make up for the loss of nutrients.

“Although AMT aims to starve tumor cells by redirecting nutrient consumption to engineered beige fat cells, it will be important to determine whether this metabolic competition could also impact normal, healthy cells,” Dr. Byun said.

AMT’s effectiveness, she continued, may vary among patients due to individual metabolic differences, the characteristics of tumors, and genetic or environmental factors.

“Understanding these variables will be critical for optimizing treatment outcomes and for identifying patients who are most likely to benefit from this approach,” Dr. Byun said.

As scientists explore such questions about engineered fat cells, they might also investigate the use of AMT in other health conditions, such as diabetes, Dr. Ahituv noted.

“We hope scientists will adopt this technology and use engineered fat cells in many different ways,” he said. “It will be exciting to see the results of this work over the next few years.”

This blog was published by the [National Cancer Institute](#) on March 27, 2025. It is republished with permission.