

# New cancer treatment disrupts tumor growth

**A team including ASU scientists has developed a novel therapy that stops cancer cells from spreading**

By Richard Harth, ASU News  
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A new discovery may bring science closer to stopping cancer in its tracks.

Researchers with the [Biodesign Center for Applied Structural Discovery](#) at Arizona State University and the team of Professor Tim Marlowe with the University of Arizona's College of Medicine in Phoenix have developed a powerful new approach: a custom-designed peptide that prevents cancer cells from anchoring, multiplying and ultimately spreading.

The findings, published in the journal [Nature Communications](#), describe how this candidate molecule disrupts key survival mechanisms that cancer cells rely on. Unlike many traditional treatments that attack cancer by blocking the activity of kinases — enzymes that help control cell growth and signaling — this new therapy blocks cancer cells from securing themselves in place, an essential step for their survival and metastasis.

The strategy could lead to better treatments for aggressive and hard-to-treat cancers, including melanoma, breast, pancreatic and lung cancers.

## Looking at cancer anew

Conventional cancer treatments, including chemotherapy and kinase inhibitors, often harm healthy cells as well as cancer cells, leading to serious side effects. Even when effective, these treatments may lose potency over time as cancer cells develop resistance.

Peptide 2012, the experimental molecule at the center of this study, offers a more precise approach by targeting cancer cells while minimizing harm to healthy tissue. Because it selectively pinpoints cancer cells, it may offer a safer and more effective option for patients with difficult-to-treat tumors, reducing the risk of unintended toxicity.

To understand how peptide 2012 interacts with cancer cells, researchers used high-resolution X-ray crystallography to map its structure. This analysis provided key insights into its mechanism of action.

“This first high-resolution X-ray structure will guide the design of even more potent peptide drugs in a rational approach to fight many cancers,” says study co-author Raimund Fromme. These

structural insights pave the way for the development of highly specific peptide-based therapies.

"I am so excited about our collaborative work as it opens a new avenue for structure-based drug development," adds co-author Petra Fromme. "While most current drugs are small synthetic molecules that can cause serious side effects, the new drug design is based on structure-based custom-designed peptides that block the protein interactions in the pathway that is critical for cancer metastasis."

Petra is the director of the Center for Applied Structural Discovery and Regents Professor at the [School of Molecular Sciences](#) at ASU. Raimund is an associate research professor with the school and does research at the center.

## **Severing cancer's lifeline**

Cancer cells must attach to their surroundings to grow and spread. A key protein, focal adhesion kinase (FAK), helps them form these strong connections. FAK is overproduced in up to 80% of solid tumors, making it a major factor in cancer progression.

Scientists have long attempted to stop FAK by targeting its kinase function — the part of the protein that helps cancer cells send signals to grow. Drugs called kinase inhibitors were developed to block this function, but these treatments have serious limitations; blocking the kinase function alone isn't enough.

That's because FAK does more than send signals — it also acts as a "scaffold," giving cancer cells the structure they need to cling together and survive. Even with its kinase function blocked, cancer can still hold on, using FAK's scaffolding ability to spread. The researchers realized that to stop FAK completely, they had to target both functions at once.

## **Disrupting cancer's support system**

To block FAK's ability to support cancer cells, the research team strategically engineered peptide 2012. This lab-designed molecule is reinforced to be more stable and effective in disrupting cancer's survival mechanisms.

The peptide prevents FAK from connecting with another protein called paxillin, which cancer cells need to survive. Without this connection, cancer cells lose their ability to anchor themselves, making it much harder for them to survive, grow and spread.

Unlike previous FAK inhibitors, which could only slow cancer's progress, peptide 2012 directly triggers cancer cell death. In laboratory tests with mouse models, cancer cells treated with peptide 2012 could no longer hold on, leading them to self-destruct. In the experiments, tumors treated with peptide 2012 shrank by 80%, a dramatic reduction that previous FAK inhibitors had failed to achieve.

Even more promising, the therapy showed no harmful side effects and selectively targeted cancer cells, reducing the risks seen with traditional treatments like chemotherapy, which damage healthy tissue. This innovative approach could lead to better treatments for some aggressive and resistant cancers, overcoming the limitations of current therapies that often lose effectiveness over time.

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## Text image(s)



Raimund Fromme







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