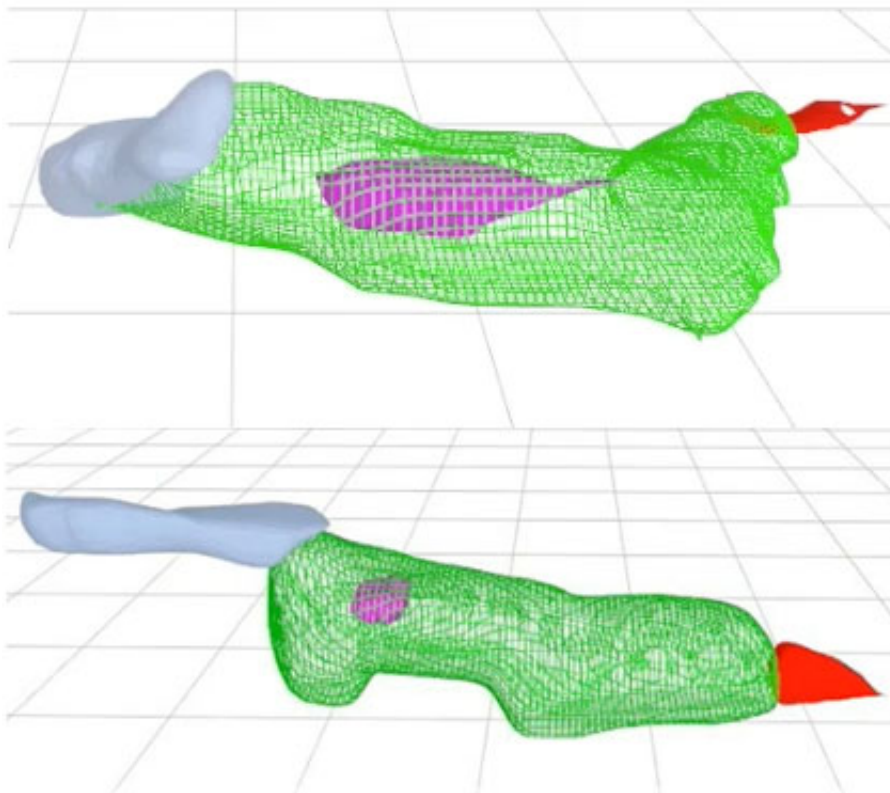


# Treating cancer: Biologists find gene that could stop tumors in their tracks

October 14 2014

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Here is a normal PTEN gene (top) as compared to a mutated PTEN gene (bottom), where the mutated gene will lose directional ability and its "false foot" (light blue) will force it to walk to different areas of the body and metastasize. Credit: Illustrations courtesy of the Developmental Studies Hybridoma Bank, University of Iowa.

The dirt in your backyard may hold the key to isolating cancerous tumors and to potential new treatments for a host of cancers.

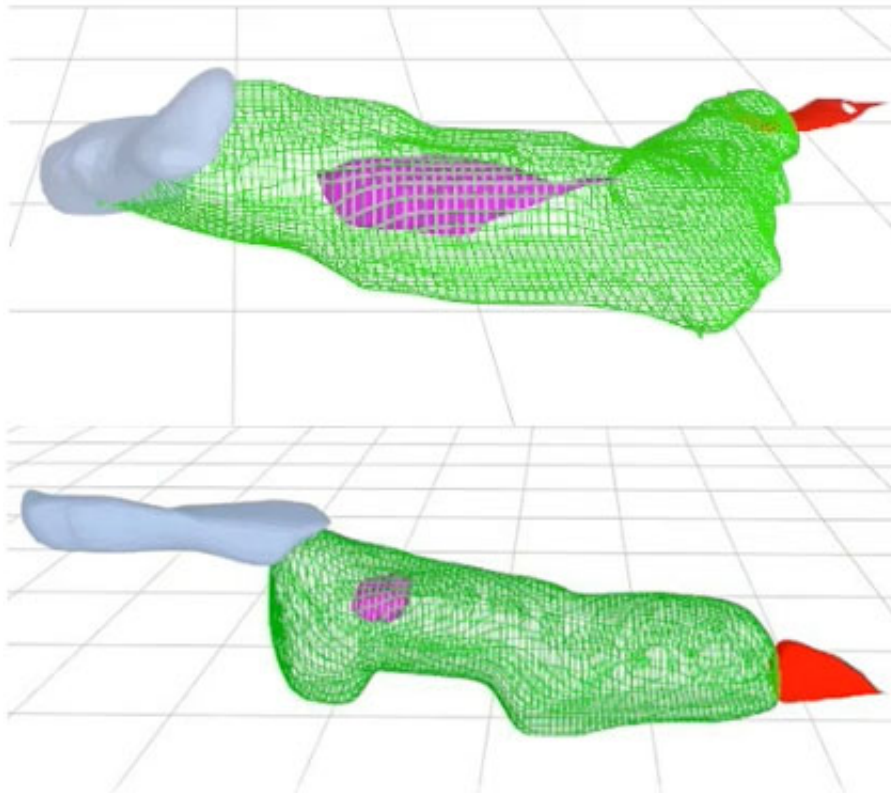
University of Iowa researchers have found a gene in a soil-dwelling amoeba that functions similarly to the main tumor-fighting gene found in humans, called PTEN.

When healthy, PTEN suppresses tumor growth in humans. But the gene is prone to mutate, allowing cancerous cells to multiply and form tumors. PTEN mutations are believed to be involved in 40 percent of [breast cancer](#) cases, up to 70 percent of [prostate cancer](#) cases, and nearly half of all leukemia cases, according to a review of the literature by the UI researchers. Combined, more than 465,000 new cases of breast and prostate [cancer](#) have been documented in 2014, according to data from the American Cancer Society.

"If you look at tumors across the board—and that doesn't mean just breast cancer or prostate cancer—you find that PTEN is the most generally mutated gene. And when you mutate PTEN in mice, you cause tumors," says David Soll, biology professor and corresponding author on the study, published in the journal *PLOS ONE*.

While it's unknown how to prevent PTEN mutations, the UI researchers became interested in finding out whether other human genes may substitute for PTEN, like a player coming off the bench when the star has been injured.

After some searching, the team found that an amoeba, *Dictyostelium discoideum*, has the gene *ptnA*, which mutates similarly to the human PTEN gene and causes behavioral defects in the cell. They also found a close relative of *ptnA* in the amoeba, which they called *lptn* that performs the same functions of *ptnA*, but to a lesser degree—a possible bench player in the amoeba's genome.



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The researchers hypothesized that ramping up the presence of lpten, making it the star on the court, could overcompensate for the mutated ptenA.

Soll and his team tested their hypothesis by placing lpten in a plasmid behind a powerful promoter designed to over-express the gene – essentially cranking up its power. They then introduced the super-

charged lpten into a cell with the mutated ptenA gene. The researchers found that the over-expressed lpten gene fully overcompensated for all of the defects in the ptenA mutant.

If the hypothesis holds true for human cells, it could lead to a new way to treat cancer. The researchers want to look for a drug that would activate the promoter for one of PTEN's close relative genes. Once a patient is diagnosed with cancer caused by a PTEN mutation, the patient could take the drug, over-express the PTEN bench player gene, and potentially stop cancer in its tracks, Soll says.

That could save many cancer patients from undergoing chemotherapy and radiation treatment for breast and other common cancers.

The finding has led the UI team to study other human [genes](#) that may be able to step in for the mutated PTEN gene and perform the same tumor-suppressing role. There are at least two close relatives of PTEN the researchers are currently studying.

"And nature might have put them there just for that, that's the curious thing," Soll explains. "Somewhere, there may be a backup system, what we call 'redundancy,' that might be the basis for better identifying tumors and possibly creating cancer-fighting drugs. You have another gene which might be able to step in for the broken gene to keep things normal, and that's what we're playing with here. It's very sophisticated."

Provided by University of Iowa

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