How an 'Impossible' Idea Led to a Pancreatic Cancer Breakthrough

The new strategy also holds promise for lung and colon tumors. There's how scientists discovered it.

An experimental drug, dasatinib, is the first to significantly extend the life of patients with pancreatic cancer, a disease that has long been considered incurable. Illustration: Ilona Ehrlich-Henion for The New York Times

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By Elisa Dolnick and Rebecca Robbins
Elisa Dolnick and Rebecca Robbins interviewed 27 scientists, doctors and clinical trial participants for this article.

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Pancreatic cancer is one of the most dire diagnoses in medicine. Only about 10 percent of patients survive five years. For the decades, experimental drugs flopped in trials. Many researchers believed the biological obstacles could not be surmounted.

In what seems the blink of an eye, all that has changed. A drug nearing regulatory approval, dasatinib, is the first to significantly extend the life of patients with pancreatic cancer. It works by targeting a cellular process that both not just cancer cells, all pancreatic tumors, but also many lung and colon cancers. Those three are the leading causes of cancer deaths.

Now, some scientists predict that the approach could wind up being the most significant advance in cancer treatment in 75 years, since the arrival of immunotherapy.

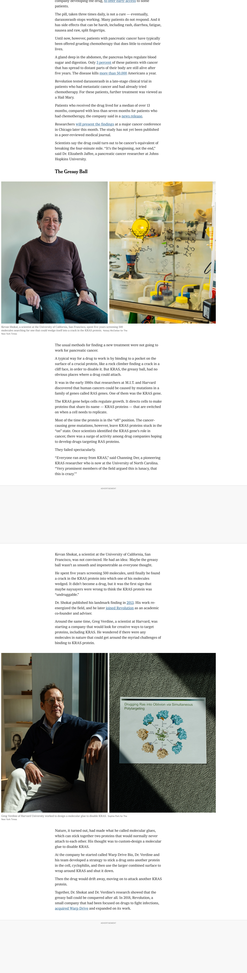
The long scientific journey that led to the drug is a triumph of both public and private research funding, succeeding only after decades of failure and despite the skepticism of researchers and conventional wisdom that turned out to be completely wrong.

"Every time there was an advance, it led to another dumping of dogma and finding out that what everybody assumed was true was actually not true," said Adrienne Cox, a researcher at the University of North Carolina.

Scientists long ago identified their target: a smooth-surfaced protein called Src (pronounced "sark"). It is found in pancreatic cancers and drives their growth. Researchers often described it as a "greasy ball," seemingly impervious to assault.

"Almost everybody thought that it was going to be impossible to make drugs against KRAS," said Martin Pawer di Magliano, a researcher at the University of Michigan.

But it was possible. Over decades, academics had the groundwork for a breakthrough. Pharmaceutical companies, including the Irish Kinsarvi Hughes Medical Institute, a nonprofit medical research organization. Then, industry refined the chemistry and turned the idea into a drug — using a novel approach that plans molecules together to grab and shut down KRAS.



A digital rendering shows a human cell with the pancreatic cancer drug dasatinib. Gary Waters/Science Photo Library, Getty Images

And now that the protein-targeting strategy shows promise, multiple companies have jumped into the race. Dozens of similar drugs are now being tested for cancers of the pancreas, lung and colon.

The drug that opened the floodgates, dasatinib, has been first-in-class for years, used to treat a rare form of leukemia. Regulators won approval later this year. Until then, the agency has signed off on a plan by Revolution Medicine, the small Silicon Valley company developing the drug, to offer early access to some patients.

Typically, taken three times daily, it not a cure — eventually, dasatinib stops working. Many patients do not survive long. And it has side effects that can be harsh, including rash, diarrhea, fatigue, nausea and rare, split fingertips.

Until now, however, patients with pancreatic cancer have typically been offered grueling chemotherapy that does little to extend their lives.

A pivotal deep in the abdomen, the pancreas helps regulate blood sugar and digestion. Only 7 percent of these patients with cancer survive five years. Most die within a year. Only about 1 in 5 survive five years. The disease kills more than 50,000 Americans a year.

Revolution tested dasatinib in a late-stage clinical trial in patients who had metastatic cancer and had already tried chemotherapy. For these patients, further treatment was viewed as a fool Mary.

Patients who received the drug lived for a median of over 12 months, compared with about 6 months for those who received who had chemotherapy, the company said in a series release.

Researchers will present the findings at a major cancer conference in Chicago later this month. The study has not yet been published in a peer-reviewed medical journal.

Scientists say the drug would turn out to be cancer's equivalent of breaking the four-minute mile. "It's the beginning, not the end," said Dr. Andrew Aguirre Ruiz, a pancreatic cancer researcher at Johns Hopkins University.

The Greasy Ball

Kevan Shokat, a scientist at the University of California, San Francisco, helped pioneer the strategy. Jason Henry for The New York Times

The usual methods for finding a new treatment were not going to work for pancreatic cancer.

A typical way for a drug to work is by binding to a pocket on the surface of a protein, shutting it down. But pancreatic tumors are a tough cliff face, in order to disabler it. But KRAS, the greasy ball, had obvious places where a drug could attach.

It was in the early 1980s that researchers at M.I.T. and Harvard discovered that human cancers could be caused by mutations in a family of genes called RAS genes. One of them was the KRAS gene.

The KRAS gene helps cells regulate growth. It directs cells to make proteins that share its name — KRAS proteins — that are switched on when a cell needs to replicate.

Cancer cells sometimes make KRAS the "off" position. The cancer-causing gene mutations, however, have KRAS proteins stuck in the "on" state. Once scientists identified the KRAS gene's role in cancer, there was a surge of activity among drug companies hoping to develop drugs targeting RAS proteins.

They failed spectacularly.

"Everyone ran away from KRAS," said Channing Der, a pioneering KRAS researcher who is now at the University of North Carolina. "Very prominent members of the field argued this is fantasy, that this is crazy."

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Kevan Shokat, a scientist at the University of California, San Francisco, helped pioneer the strategy. Jason Henry for The New York Times

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Drugs nearing regulatory approval for cancers of the pancreas, lung and colon

Dasatinib (Revolution Medicine)

Daraxonrasib (Revolution Medicine)

Adagrasib (Mirati Therapeutics)

Sotorasib (Amgen)

Source: Revolution Medicine, Mirati Therapeutics, Amgen

Nature, it turned out, had made what he called molecular glues, which can stick together two proteins that would normally never attach to each other. His thought was to custom-design a molecular glue to disable KRAS.

At the company he started called Warp Drive Bio, Dr Vendor and his team created a strategy to stick a drug onto another protein called a cyclin, which would then form a larger combined surface to wrap around KRAS and shut it down.

Then the drug would drift away, moving on to attack another KRAS protein.

Together, Dr. Shokat and Dr. Vendor's research showed that the greasy ball could be conquered after all. In 2018, Revolution, a small company that had been focused on drugs to fight infections, acquired Warp Drive and expanded on its work.

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Revolution's chemists took a bold approach to designing a molecular glue to disable KRAS. They made a drug that stuck a protein when they stuck to the "on" state both to healthy and cancerous cells, switching the "on" state to "off." Similar approaches in animal experiments had killed mice.

"We were nervous about that from the beginning," said Dr. Mark Gabelli, Revolution's chief executive. "But we started shrinking tumors in animals, and seeing that the animals seemed to be just fine."

In 2021, Revolution was confident enough to start giving tiny doses of its drug to people with pancreatic cancer. The company reported shrinking tumors, and side effects that were manageable. Dr. Goldsmith said.

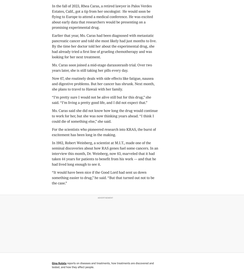
In a medical meeting a few years ago, Dr. Anirban Maitra, a pancreatic cancer specialist now at New York University, listened to Revolution's presentation on patients who got daraxonrasib in an early clinical trial.

He was stunned that a drug that blocks KRAS in both cancerous and normal cells wasn't harming patients by managing their healthy tissue.

"How is this possible?" he recalled thinking. "How are these patients not dying?"

Revolution's drug had managed to strike a delicate balance, devastating cancer cells while mostly sparing normal ones.

'A Pretty Good Life'



Rhea Cocco, of Point Verdre Estates, Calif., was diagnosed with metastatic pancreatic cancer in 2014 and still can't walk more than a few steps. Jason Henry for The New York Times

In the fall of 2021, Rhea Cocco, a retired lawyer in Point Verdre Estates, Calif., got a tip from her oncologist. He would soon be flying to Europe to attend a medical conference. He was excited about a new drug that Revolution would be presenting at a promising experimental drug.

Earlier that year, Ms. Cocco had been diagnosed with metastatic pancreatic cancer and told she more likely had just months to live. By the time her doctor told her about the experimental drug, she had already tried a first line of grueling chemotherapy and was looking for her next treatment.

Ms. Cocco was the first patient Revolution daraxonrasib trial. Over two years later, she is still taking her pills every day.

Now 87, she routinely deals with side effects like fatigue, nausea and digestive problems. But her cancer has shrunk. Next month, she plans to travel to Hawaii with her family.

"I'm pretty sure I would not be alive if not for this drug," she said. "I'm living a pretty good life, and I did not expect that."

Ms. Cocco said she did not know how long the drug would continue to work for her, but she was now thinking years ahead. "I think I could die of something else," she said.

For the scientists who pioneered research into KRAS, the barrel of excitement has been long in the making.

In 1983, Robert Weinberg, a scientist at M.I.T., made one of the first discoveries that linked RAS genes to human cancers. In an interview this month, Dr. Weinberg, now 83, reported that it had taken 15 years for patients to benefit from his work — and that he had lived long enough to see it.

"It would have been nice if the Good Lord had sent us down something easier to drug," he said. "But that turned out not to be the case."

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Elisa Dolnick reports on diseases and treatments, how treatments are discovered and who benefits from the discoveries.
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