

Fred Hutch Study Dramatically Expands Therapeutic Range of Approved Kinase Drugs

Large-scale analysis reveals new uses for existing therapies across cancer types and non-cancer diseases.

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Rare cancer expert [Taran Gujral, PhD](#), cares more about what a cancer drug can do than what it is designed to do.

His lab at Fred Hutch Cancer Center investigates a promising target for cancer drugs — enzymes called kinases, which send signals regulating almost every activity in a healthy cell's life cycle including growth, division and the controlled death of damaged cells.

“In cancer, many of these kinases become overactivated in different ways,” Gujral said. “They can be switched on by mutation, by fusing with other genes, or simply by being produced in higher amount, which is why they’re such important target for cancer treatment.”

A class of drugs that blocks the molecular activity of malfunctioning kinases — called kinase inhibitors — grew from a single drug in early 2000s to 100 kinase inhibitors by the end of 2025, each designed for one specific kinase and most aimed at treating cancer.

In one of the most comprehensive studies to date, [published recently in the journal Nature Biotechnology](#), Gujral and his team show that many FDA-approved drugs can block more than the single kinase they were designed to target, including many cancer-causing mutations.

For example, Gujral and his colleagues found that kinase inhibitors designed for mutated forms of lung cancer also work on specific types of brain and pancreatic cancer. A drug designed to treat leukemia can also help a drug designed for lung cancer work better.

The study expands the number of kinases that can be inhibited by an FDA-approved drug from 89 to 235, with relevance across many kinds of cancer.

Gujral's lab in the [Human Biology Division](#) at Fred Hutch also developed a free, public interactive web-based tool called Kinase Inhibitor Repurposing Hub (KIRhub) to visualize that data, which

maps new avenues for research, precision medicine, and potential therapies for challenging cancers that lack effective treatment options.

Drugs that block the on-switches for cancer-fueling processes

Kinases make things happen in the cell by precisely regulating the activation of specific proteins — the cell's molecular workers — which enables them to initiate signaling pathways for a wide variety of cellular processes essential to life.

To do this, a kinase must first grab an energy-storing molecule called ATP, which contains three chemical groups called phosphates that function like a kind of battery.

The kinase tucks ATP into a binding pocket between its two halves. It then removes one of ATP's phosphates and attaches it to the adjacent protein that the kinase is made to activate.

The transferred phosphate “battery” carries the extra energy needed to change the protein's function and initiate cellular signaling.

Kinase inhibitors are small-molecule drugs that prevent this battery transfer by nestling into the pocket where ATP normally fits, blocking ATP from binding.

“If they occupy the ATP-binding pocket so the ATP cannot bind anymore, then there's no phosphate to be transferred and the whole signal stops,” Gujral said.

But most kinases have ATP-binding pockets with similar structures, which makes it difficult to block just one kinase without also affecting others.

Drug developers must be careful that a kinase inhibitor targeting something cancerous doesn't accidentally also inhibit something else that healthy cells need to survive, making the drug unsafe.

The rapid expansion of FDA-approved kinase inhibitors has generated a mountain of molecular data about the drugs that Gujral and others are mining to find out what else these kinases — which are already proven to be safe — can do against other cancers that share a similar underlying biology.

Casting a wide net

To find out what a drug can do beyond what it is designed to do, Gujral's lab teamed with an outside partner, [Reaction Biology](#), to screen FDA-approved drugs against a wide range of kinases — including many cancer-causing mutations.

Other studies have profiled kinase inhibitors, but they lacked the size, scope, and especially the focus on mutant variants seen in Gujral's study.

Gujral's team analyzed 92 clinical kinase drugs across 758 kinases, including 349 mutant or kinase gene-fusions spanning a wide range of tumor types.

Lung, lymphoid, skin, brain and central nervous system cancers contributed the most kinase mutations to the dataset, along with breast, uterus, liver, sarcoma and large intestine tumors.

"These mutated versions are often what drives cancer, so we tested drugs against 349 of them," Gujral said. "That hadn't been done before."

By casting such a wide net, Gujral's team expanded the range of kinase targets for FDA-approved drugs from 89 to 235. Their comprehensive analysis also captured kinase variants from cancers with limited treatment options.

Overall, they found that at least one drug could strongly block the vast majority of cancer-driving changes, including 94% of kinase mutations and 97% of gene fusions.

Testing the findings with experiments

The systematic analysis tested drugs against kinases in purified "test-tube" conditions, but Gujral's team also used the data to conduct several pilot experiments using mice and tumor cell models, focusing on commonly mutated kinases in lung cancer.

They discovered, for example, that the drug tepotinib, an inhibitor designed to block a growth-promoting kinase called MET in lung cancer, can also block the kinase IRAK1/4 in a brain cancer called glioblastoma.

And because IRAK1/4 also plays a crucial role in innate immunity and inflammation by regulating cholesterol balances, tepotinib might also provide a treatment strategy in cardiovascular disease.

They also discovered new workarounds when drugs lose their effectiveness because cancer adapts and becomes resistant. For example, gilteritinib, a drug designed to treat leukemia by inhibiting FLT3, a kinase involved in the production of new blood cells, can also work on drug-resistant MET mutants in lung cancer.

And some kinase inhibitors, they found, can be combined in a one-two punch that not only blocks the primary kinase but keeps another kinase from initiating a signaling pathway that would help the cancer become drug-resistant.

Their experiments showed that drugs targeting a particular kinase may not be effective for all mutated variants of that kinase, rendering a standard treatment useless for some patients, especially those with rare cancers and variants. But another drug designed for something else might do the trick.

"The goal of precision oncology is simple: match the mutation to the right drug," Gujral said.

“Developing new drugs for rare cancers is incredibly challenging, but if existing drugs already target these mutant variants, then that gives us a powerful head start.”

An online tool for anyone to search for answers and insights

Rather than plowing through the vast datasets that Gujral’s team produced, researchers and clinicians can use a public, web-based tool his lab developed called [KIRhub](#) to quickly and easily find connections.

The tool helps users identify drugs that target specific kinases, visualize how those drugs interact with many kinases and explore which kinases are important in different cancer types.

Users can enter the name of an FDA-approved kinase inhibitor and see which kinases it can block beyond what it was designed to block. Or they can enter a particular mutant kinase and see what drugs pop up and to what degree they block or reduce its activity.

“Just enter the kinase name, and the tool instantly shows which existing drugs specifically target it,” Gujral said.

Scientists who also treat patients in the clinic can use it to inform treatment strategies based on mutations in kinases within a patient’s tumor and to identify alternative drugs that may boost the effectiveness of standard therapies.

KIRhub is designed to be interactive and frequently updated as new drugs come on the market and research reveals new connections and potential uses.

“Since our manuscript was accepted, we’ve already added six more drugs to KIRhub,” Gujral said. “We expect that 20 to 50 additional drugs will be added over the next five years.”

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