

## Tortoise and the hare: New drug stops rushing cancer cells, slow and steady healthy cells unharmed

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The American Cancer Society estimates that 44,000 new cases of pancreatic cancer will be diagnosed this year and that 37,000 people will die from the disease. These are not strong odds. A new drug, rigosertib, allows pancreatic cancer cells to rush through replication – and then stops them cold, killing them in the middle of a step called M phase. Healthy cells that don't rush are unharmed.

Data from a phase I clinical trial of patients with advanced pancreatic cancer and additional solid tumors recently published in the journal *Clinical Cancer Research* shows the strategy has promise. While the goal of any phase I trial is to establish the dosage that best balances effectiveness against side effects, 11 of the 19 patients treated achieved stable disease, which lasted for a median of 113 days.

"Really, the drug takes one of cancer's greatest strengths and turns it into a weakness," says Wells Messersmith, MD, co-leader of the Developmental Therapeutics Program at the University of Colorado Cancer Center and the clinical trial's national principal investigator.

Instead of going with the flow of the natural [cell cycle](#), cancer cells amplify two signals – PLK1 and PI3K – which allows them to blast through the cell cycle and divide much more quickly. In the process, they break this step of the natural cell cycle, known as the G1 regulatory mechanism, and thus depend on the kick of PLK1 and P13K to push at a

frenzied pace through [replication](#).

It's specifically these two signals, PLK1 and PI3K, that rigosertib targets. With these signals turned off, [cancer cells](#) get stuck and die in the stage of the cell cycle called M phase – while [healthy cells](#) that stuck to the slower, natural method of division chug past unharmed.

Wells Messersmith, MD, co-leader of the Developmental Therapeutics Program at the University of Colorado Cancer Center

"This one-two punch, targeting these two distinct signaling pathways, allows us to interfere twice with cancer cells' ability to replicate," Messersmith says. And it also allows doctors to target cancers that may have evolved resistance to one or the other target.

Provided by University of Colorado Denver

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