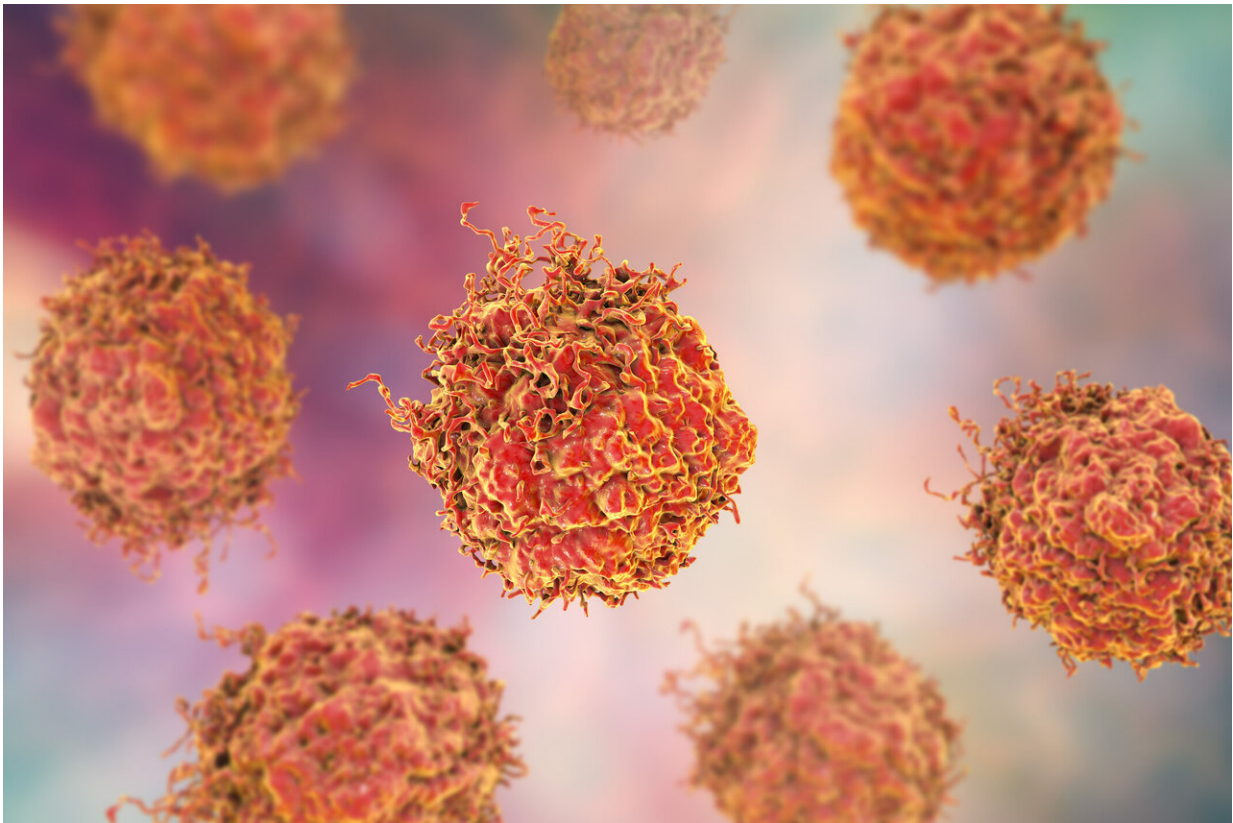


# Blocking specific protein could provide new treatment for deadly form of prostate cancer

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Blocking CDK7 sets off a chain reaction that results in the death of prostate cancer cells that have spread and are resistant to standard therapies. Credit: Penn Medicine

Blocking a kinase known as CDK7 sets off a chain reaction that results

in the death of prostate cancer cells that have spread and are resistant to standard therapies, according to a new study from researchers in the Abramson Cancer Center at the University of Pennsylvania. The team identified the role of CDK7 as the on/off switch that controls Med-1, a process that works in partnership with the androgen receptor to drive prostate cancer growth. Researchers show turning the switch off eventually leads to the death of cancer cells in mice. *Cancer Discovery* published the findings today.

Androgen deprivation therapy is a standard approach to treating [prostate cancer](#), but over the course of treatment, a majority of patients will eventually become resistant to the therapy, allowing the [cancer](#) to grow and spread. This is referred to as metastatic castration-resistant prostate cancer (CRPC). There are two drugs approved by the U.S. Food and Drug Administration for these cases, but patients see little or no long-term survival benefit from these therapies.

Since the [androgen receptor](#) (AR) continues to be the main the driver of cancer growth in CRPC, taking away its function is still critical. Given the disease's resistance to therapies that try to take on AR directly, a new approach is needed. While these cancers do not have additional mutations or other genetic overexpression, the Penn team was still able to identify a new target thanks to what researchers called "AR's co-pilot."

"We know that AR does not work alone; that it needs Med-1 as its partner," said the study's senior author Irfan A. Asangani, Ph.D., an assistant professor of Cancer Biology in the Perelman School of Medicine at the University of Pennsylvania. "Our study found a way to turn off Med-1, leaving AR without its co-pilot which means the cancer cannot grow and the cells eventually die."

Using an inhibitor to turn off CDK7 led to the death of CRPC cells in

both the lab setting and in animal models. Researchers also saw very limited off-target effects of this approach, since [healthy cells](#) have redundancies in place to deal with the loss of Med-1, meaning only the cancer cells end up dying off.

"Our theory is that these cancer cells are addicted to Med-1 and AR but other cells are not, so we're essentially cutting them off from their addiction," Asangani said.

CDK7 inhibitors are already being tested in phase I [clinical trials](#) for other cancers—including leukemia, lung cancer, glioblastoma, and breast cancer—but Asangani said this study shows the rationale for testing them in CRPC.

Reyaz ur Rasool, Ramakrishnan Natesan, and Qu Deng are co-first authors on the study. Other Penn authors include Shweta Aras, Priti Lal, Samuel Sander Efron, Erick Mitchell-Velasquez, Jessica M. Posimo, Lauren E. Schwartz, Daniel J. Lee, and Donita C. Brady.

Provided by Perelman School of Medicine at the University of Pennsylvania

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