Using CRISPR to improve accuracy of cancer-fighting drugs

Cancer drug developers may be missing their molecular targets—and never knowing it. Many recent drugs take aim at specific cell proteins that drive the growth of tumors. The strategy has had marked successes, such as the leukemia drug Gleevec. But a study now finds that numerous candidate anticancer drugs still kill tumor cells after the genome editor CRISPR was used to eliminate their presumed targets. That suggests the drugs thwart cancer by interacting with different molecules than intended.

The study, published [September 11] in Science Translational Medicine, points to problems with an older lab tool for silencing genes that has been used to identify leads for such drugs. The results also hint that the drugs in question, most of which are in clinical trials, and perhaps others could be optimized to work even better by pinning down their true mechanism.

“The work is very well done and it’s a great public service. I hope people talk about it. I don’t find any of it surprising, unfortunately,” says William Kaelin of the Dana-Farber Cancer Institute in Boston, who has written about why promising preclinical findings are often not reproducible, or fail to lead to drugs.

Read full, original post: Some cancer drugs miss their target. CRISPR could improve their aim