

Scientists identify possible KRAS downstream target for pancreatic cancer therapy

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While the mutated KRAS oncogene is associated with many cancers, it has not yet been successfully targeted by a therapeutic agent. Scientists are trying to find another way to target the gene by blocking signals from another protein downstream.

further pharmacologic studies," said Baldwin.

Provided by University of North Carolina Health Care

A University of North Carolina School of Medicine team offers first evidence of the role of a protein called GSK-3 alpha in promoting oncogenic KRAS function. When the scientists inhibited GSK-3 in a model of pancreatic tumors, the team got a strong anti-[tumor response](#), thus offering a potential [therapeutic option](#). Their findings are published in the June issue of the journal *Cancer Discovery*.

Albert Baldwin, William Rand Kenan Professor of Biology, study senior author, and associate director of UNC Lineberger Comprehensive Cancer Center, says, "GSK-3 promotes activity of a protein called NF-kappa B. Our lab has been studying NF-kappa B for a number of years and has published that this protein is important in KRAS signaling. But how KRAS activates NF-kappa B has not been well understood. We have found a link."

GSK-3 has alpha and beta forms. The beta form has been studied and is considered to be a [tumor suppressor](#). GSK-3 alpha was considered to be redundant to GSK-3 beta. The research team studied GSK-3 alpha in mice with human [pancreatic tumors](#) and found through their experiments that its function is different from GSK-3 beta.

"Our data suggest that GSK-alpha is really an onco-protein and that KRAS utilizes GSK-alpha to activate both NF-kappa B pathways, called canonical and noncanonical. This finding is important because GSK-3 alpha sits on top of the two pathways and inhibits them both, thus making it a viable [therapeutic target](#). We are conducting

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