Team pinpoints role of key protein in pancreatic ductal adenocarcinoma

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A team based at North Carolina Central University (NCCU) and UNC Lineberger Comprehensive Cancer Center has established a connection between a known cancer gene called KRAS and a protein called Pim-1 kinase.

Pancreatic ductal adenocarcinoma is responsible for more than 95 percent of pancreatic cancer. This very aggressive form of cancer is responsible for more than 35,000 deaths each year in the United States, with only 20 percent of patients surviving one year after diagnosis.

A team based at North Carolina Central University (NCCU) and UNC Lineberger Comprehensive Cancer Center has established a connection between a known cancer gene called KRAS and a protein called Pim-1 kinase. The team found that mutations in the KRAS gene resulted in higher levels of Pim-1 expression in several laboratory models of human pancreatic ductal adenocarcinoma, and that suppression of Pim-1 in KRAS-dependent models resulted in decreased growth – key indications that Pim-1 is required for the growth of these cancerous cells and is a promising therapeutic target. Their results were published in the journal *Carcinogenesis*.

“There are two key implications for the treatment of pancreatic cancer,” said Antonio T. Baines, PhD, assistant professor of biology and a member of the cancer research program at NCCU’s J.L. Chambers Biomedical/Biotechnology Research Institute.

“Not only does Pim-1 look like a promising target for new therapies, but it may also serve as an effective biomarker for the activity of mutated KRAS in pancreatic cancers.”

Testing for and molecular targeting of KRAS-mutated cancers has been pursued for several years in the treatment of pancreas, lung, colorectal and other cancers. New, targeted treatments are currently in clinical trials.

The team also tested the role of Pim-1 in cell invasion, part of the overall mechanism through which cancer cells metastasize (spread in the human body). The team also found that Pim-1 plays a role in radioresistance, a phenomenon where cancer cells are less susceptible to common radiation therapies for cancer. When Pim-1 expression is inhibited in the laboratory, the cells’ ability to spread and to resist ionizing radiation is also reduced.

“We hope that this research can contribute to similar breakthroughs in targeted therapies for pancreatic ductal adenocarcinoma,” said Baines.

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