Comparing liver and pancreatic cancers

Surprising similarities in cellular mechanisms drive both liver and pancreatic cancers

PUTNAM VALLEY, NEW YORK, USA, July 2, 2018 /EINPresswire.com/ -- Putnam Valley, NY (July 2, 2018) – Liver cancer and pancreatic cancer are known to share one statistical outcome – both have poor survival rates. Now, researchers in Belgium have also found that the two deadly cancers share similar tumor developmental mechanisms in terms of tissue types, genetics, and cell mutations, among other similarities. Will this new knowledge lead to better prevention and treatment for both cancers?

The striking similarities between various forms of liver cancer and pancreatic cancer may stem from the liver and the pancreas having developmental similarities in their embryonic stages, said researchers from the Catholic University of Louvain in Brussels, Belgium.

“Because the liver and pancreas are closely associated organs, sharing an embryonic origin, unraveling the process in tumor development in one organ may provide a better understanding of the process in the other organ,” said corresponding author Dr. Patrick Jacquemin.

According to the researchers, the liver and pancreas share molecular properties because they originate in embryonic tissues in adjacent regions, but their advancing development differentiates maturing cells into liver cells (hepatocytes and cholangiocytes) and for the pancreas, endocrine cells (the small “island” cells (the islets of Langerhans) and “exocrine” (acinar) cells, responsible for producing and transporting enzymes.

“Considering their embryonic developmental similarities, we reasoned that some common mechanisms driving tumor development may also be similar,” said co-author Dr. Ivan Borbath of the university’s Department of Hepato-Gastro-Enterology.

In their study, the researchers compared two types of liver cancer (HCC and CCA) to two types of pancreatic cancer (PDAC and ACC). They compared cell type similarities, genetic factors that may cause cell mutations in either organ, and common intracellular “signaling” mechanisms. They also investigated several genetic mutations and pathways and several stood out as significant.

For example, pancreatic ductal adenocarcinoma (PDAC) is one of the most common pancreatic tumors, found in 85 to 95 percent of pancreatic tumors. The authors note that “a striking characteristic of PDAC” is the prevalence of the Kirsten RAt Sarcoma virus (KRAS) mutation found in 88 to 100 percent of PDAC tumors.

KRAS, an “oncogene,” works as an on/off switch and, as a signaling gene, is associated with many cancers when it mutates.

“This very high mutation rate suggests that KRAS plays a key role in pancreatic tumor development,” explained co-author Dr. Elsa Ghurburrun. “The KRAS genetic mutation is also found in CCA liver cancers, but its mutation rate is much less important than in PDAC.”

In both pancreatic cancer and liver cancer, KRAS mutations are also accompanied by two other

mutations, GNAS and RNF43.

“The mere presence of KRAS in the acinar genome is not sufficient to initiate tumor development and malignancy,” added co-author Dr. Frederic P. Lemaigre. “Tumor development requires association of a KRAS mutation and inflammation.”

Although the liver and pancreas are closely associated organs sharing an embryonic origin, similar tissue organization, and similar molecular mechanisms that can lead to cancer development, the authors conclude that more knowledge is required to know exactly to what extent pancreatic and liver cancers can be considered similar.

“Current data suggests that mechanisms of tumorigenesis in the liver and pancreas might significantly overlap,” concluded the authors. “However, a more complete understanding of the role of KRAS, GNAS and RNF43 requires the development of new laboratory animal models for testing.”

Their paper appears in the current issue of Gene Expression: The Journal of Liver Research. It is freely available on-line as an unedited, early epub at: http://ingentaconnect.com/content/cog/ge

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Patrick Jacquemin
Universite catholique de Louvain
+32 2 764 75 31
email us here
