MedicalXpress

In 'spontaneous' liver cancer, researcher sees a cure

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Adding more good news to last week's announcement that Nexavar® (sorafenib) may be the first effective treatment for advanced liver cancer, researchers at the Lombardi Comprehensive Cancer Center at Georgetown University have uncovered a new molecular mechanism that may "spontaneously" cause liver cancer.

Part of the well-known TGF-ß tumor suppressor pathway, the molecule disappears in the cells of nearly 90 percent of human hepatocellular cancers, the most common type of liver cancer. Lopa Mishra, MD, professor and vice chair in the department of surgery at Georgetown University Medical Center, showed that loss of only one copy of the embryonic liver fodrin, or ELF gene, can result in spontaneous development of liver cancer in human cell cultures and in vivo models.

In a paper published online by *Oncogene* on June 4, Mishra and her team also reported that by reintroducing ELF to the cancer cells, the proteins driving cell division and growth were kept in check. To the research team, this implies that ELF or another inhibitor of downstream cell division and growth proteins could be developed into an effective new therapy.

"We're looking for ways of treating untreatable cancers," explained Mishra. "Pancreatic and liver cancers are the third- and fourth-leading causes of cancer death in the world."

Hepatocellular cancer has a very low 5-year survival rate – less than 5

percent – and the incidence of the disease has risen in the United States over the past several years.

One difficulty in treating liver cancer is the variety of different mutations seen in among patients. The findings about ELF may indicate that it is a critical component that could be targeted to treat 90 percent of patients with this disease. Currently, only 12 percent of patients are eligible for surgery, and very few other treatment options are available.

Mishra's findings on the role of ELF in the development of liver cancer also suggest a method for preventing the disease. Because the cancer forms as a multistep process, beginning with cirrhosis and following a known progression, it's possible that the same ELF molecule can be targeted to pre-cancerous lesions in the liver.

Source: Georgetown University Medical Center