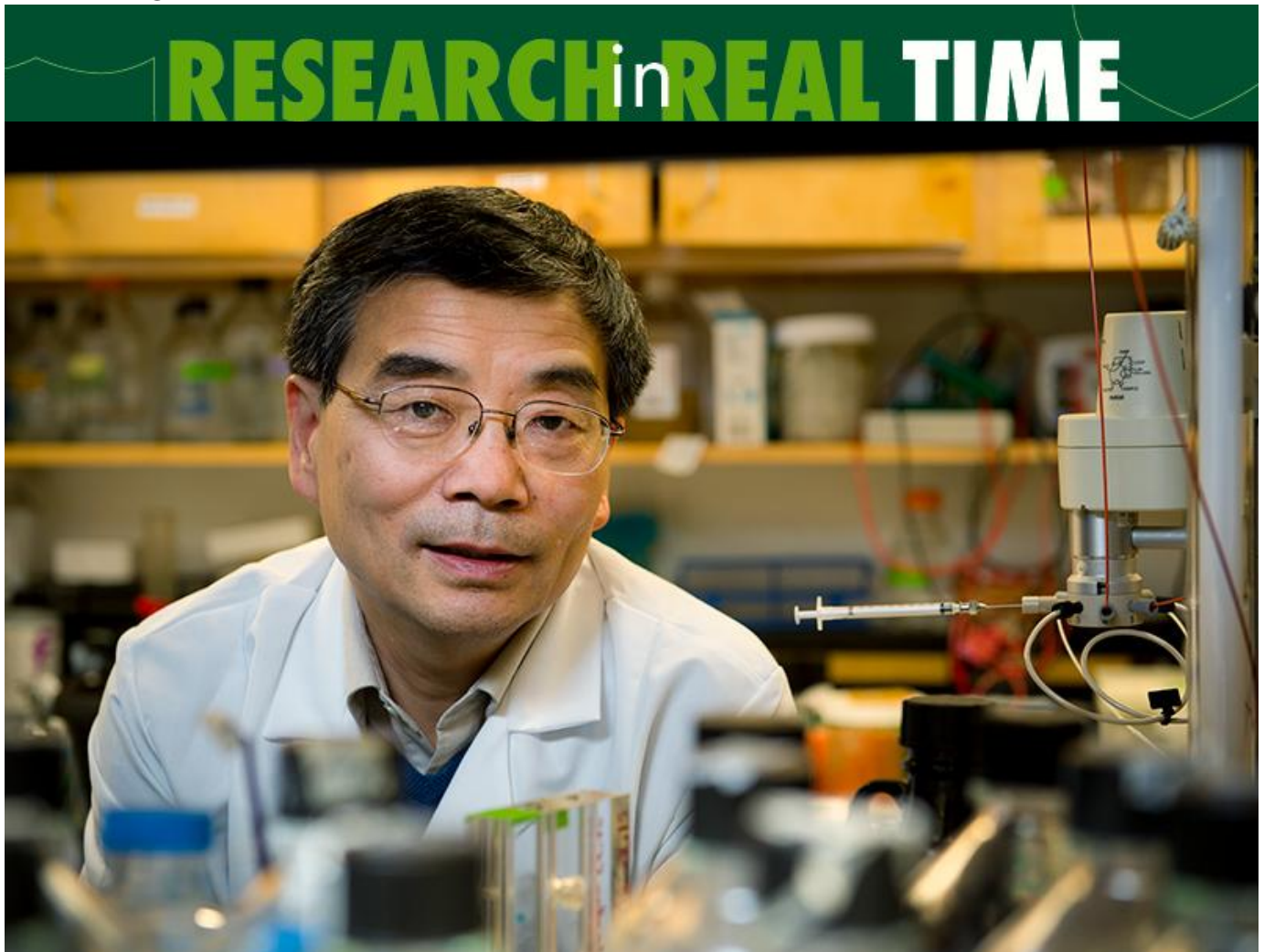


Researcher probes genetic mutation that leads to liver cancer

March 27, 2018 10:15 AM Melanie Cross
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Dr. Hua Lu, chair of the Tulane University Department of Biochemistry, led a research team that identified a genetic mutation that is associated with liver cancer in certain countries. The findings were published in 'Molecular Cell.' (Photo by Paula Burch-Celentano)

Dr. Hua Lu, chair of the Tulane University Department of Biochemistry, and his research team **recently published a study in *Molecular Cell*** that sheds light on a unique genetic mutation believed to be associated with a higher

incidence of liver cancer in China and some African countries.

"P53, a protein that normally functions as a tumor suppressor, preventing the growth and spread of cancerous cells, is highly mutated in most human cancers, causing a loss of normal function," said Lu, who also holds the Reynolds and Ryan Families Chair in Translational Cancer at the School of Medicine.

In some parts of China and Africa, the mutation of p53 in liver cancers occurs mainly at one particular amino acid — a "hot spot" in the genome.

"We believe this hot spot mutation is highly associated with exposure to the carcinogenic chemical aflatoxin B1, which is produced by fungi that infect food stores, like peanuts and some dried foods, in China and Africa," said Lu.

These areas are particularly vulnerable because of warm, moist climates and limited access to air conditioning or refrigeration. "Many of those who eat these foods don't realize they are infected with the fungi," said Lu. "Fortunately, this is not a problem in the United States or other countries with more modern food storage methods."

Unlike the United States, where hepatitis C virus (HCV) is often associated with liver cancer incidence, it's hepatitis B virus (HBV) that is more prevalent in China and Africa. Scientists believe the combination of aflatoxin B1-induced p53 mutation and hepatitis B infection creates an environment conducive to the development of liver cancer by reprogramming p53 to help promote tumor growth.

"The mechanism through which this transformation takes place is not entirely clear and is the focus of this paper," said Lu. "Our study unveils a unique molecular pathway that renders mutated p53 more oncogenic in promoting liver cancer cell growth."

This pathway could ultimately become a target for the therapeutic treatment of liver cancer.

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