Study suggests alcohol causes liver damage.

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A published paper from the research team suggests that the enzyme Energetic Starch Enzyme Formation in Human Liver with Lactose and Ethanol caused liver damage. The paper was published in November in the journal JAMA.

Energetic starch enzymes are fundamental proteins that work at the cell level to exert control over some enzymatic processes. The enzymes work by acting on starchy monoglycerides. Ethanol, one of the key constituents of ethanol-based fuels such as corn ethanol, may trigger ESI production in the liver, making it more susceptible to damage after being exposed to ethanol.

EISA degrades antigens. Toxic mutations have previously been reported for hemoglobin and small molecule receptors for HIV and hepatitis. Disturbances in this process, caused either by EISA or through its metabolites, may potentially produce protein damage in HIV, HIV-infected human blood cells and hepatitis C viruses. These deformed proteins can damage enzymes that have an important role in the overall functionality of the liver, such as transporters, lipid biosynthesis and anti-inflammatory cytokines. Changes in EISA use in liver cells has been suggested as the underlying cause of acute and chronic organ damage in humans and animals. More than 150 proteins are known to interact with EISA to perform specific biochemical functions in the liver.

The researchers focused on the enzyme EISA- lactose synthase. They screened human hepatocytes from a large number of individuals and found that EISA-lactose synthase was methylated in vesicles of mice and humans. In addition, they analyzed EISA-lactose synthase proteins in an electron microscope. The team found EISA-lactose syntheses by dimethyltransferase, which is a natural enzyme kinetically similar to methyltransferase, in a significant proportion of the enzymes. They continued their work to investigate the chain of events that cause EISA-lactose syntheses and their reactions with enzymes deregulated by the EISA1 complex. The researchers found that EISA1 complexes severely deregulated EISA-lactose synthesis in the liver of mice. The team continued their analysis of EISA1 complexes in a mouse liver using lipid spectroscopy and serum tissue imaging. They discovered that Lutein Fatty Acid and the related enzyme Janus monozymes suppressed EISA-lactose syntheses and further showed that dietary modification by ethanol, lactose or tapioca glycol, may disrupt EISA synthesis in the liver.

The results indicate that ethanol, lactose or tapioca glycol have the potential to effect EISA synthesis in human liver cells, particularly in mammalian liver. Through further study, EISA and Lactose, when co-opted by EISA1 complexes, may provide promising drug targets for preventing EISA-lactose syntheses and further investigation of the impact of EISA1 complexes is warranted.

Source:

Taku Inokuchi, Shinpei Kawakami, Fumio Otaki, Toshiyuki Shigeoka, Noriaki Kitano, Takeshi Horiya, Teruyuki Niifami, Toshiyuki Yanagisawa, Jun Tamada, Sumio Takahashi, Hideki Nishioka, Kenzaburo Uchida, Yusuke Kuroda, Takenori Toyoda, Naoko Uchida, Haruo Goto, Yukimi Takemoto, Toshiaki Shizu, Tetsuya Yamamoto

JAMA-published research report:

poster:

Abstract

A Close Up Of A Bird On A Fence