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High Levels of Fructose, Trans Fats Lead to Significant Liver Disease, Says Study

ScienceDaily (June 23, 2010) — Scientists at Cincinnati Children's Hospital Medical Center have discovered that a diet with high levels of fructose, sucrose, and of trans fats not only increases obesity, but also leads to significant fatty liver disease with scar tissue.

Moreover, the researchers conducted the study in a new mouse model of obesity and liver disease that so closely models human disease they will now be able to test therapies to determine their effectiveness, according to Rohit Kohli, M.D., a gastroenterologist at Cincinnati Children's Hospital Medical Center and the study's main author.

"Fructose consumption accounts for approximately 10.2 percent of calories in the average diet in the United States and has been linked to many health problems, including obesity, cardiovascular disease and liver disease," says Dr. Kohli. "We've developed a mouse model that is very close to human disease, allowing us to better understand the process involved in the development and progression of obesity-related fatty liver disease."

The study also includes preliminary data on a simple blood test for a biomarker that differentiates the stages of disease in this model. Physicians currently monitor the progression of fatty liver disease by taking liver biopsies, which are invasive procedures.

The study, which was conducted with scientists from the Metabolic Disease Institute at the University of Cincinnati, is published online in the journal Hepatology.

The study was conducted in mice, some of which were fed a normal diet of rodent chow and some a 16-week diet of fructose and sucrose-enriched drinking water and trans-fat solids. Their liver tissue was then analyzed for fat content, scar tissue formation (fibrosis), and the biological mechanism of damage. This was done by measuring reactive oxygen stress, inflammatory cell type and plasma levels of oxidative stress markers, which are known to play important roles in the development of obesity-related liver disease and its progression to end-stage liver disease.

The investigators found that mice fed the normal calorie chow diet remained lean and did not have fatty liver disease. Mice fed high calorie diets (trans-fat alone or a combination of trans-fat and high fructose) became obese and had fatty liver disease.

"Interestingly, it was only the group fed the combination of trans-fat and high fructose which developed the advanced fatty liver disease which had fibrosis," says Dr. Kohli. "This same group also had increased

oxidative stress in the liver, increased inflammatory cells, and increased levels of plasma oxidative stress markers."

Dr. Kohli hopes to further investigate the mechanism of liver injury caused by high fructose and sucrose enriched drinking water and study a therapeutic intervention of antioxidant supplementation. Antioxidants are natural defenses against oxidative stress and may reverse or protect against advanced liver damage, according to Dr. Kohli.

The investigators also would like to use this model to better understand human fatty liver disease and perform clinical trials using novel therapeutic and monitoring tools.

"Our data suggest that supplementation with pharmaceuticals agents should be tested on our new model to establish whether one is able to reverse or protect against progressive liver scarring and damage," says Dr. Kohli.

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