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Fructose Consumption Linked to the Increase of Liver Disease Among Adolescents and Children

New study indicates that sugar intake and uric acid concentration independently contribute to the incidence and progression of liver disease, reports the Journal of Hepatology

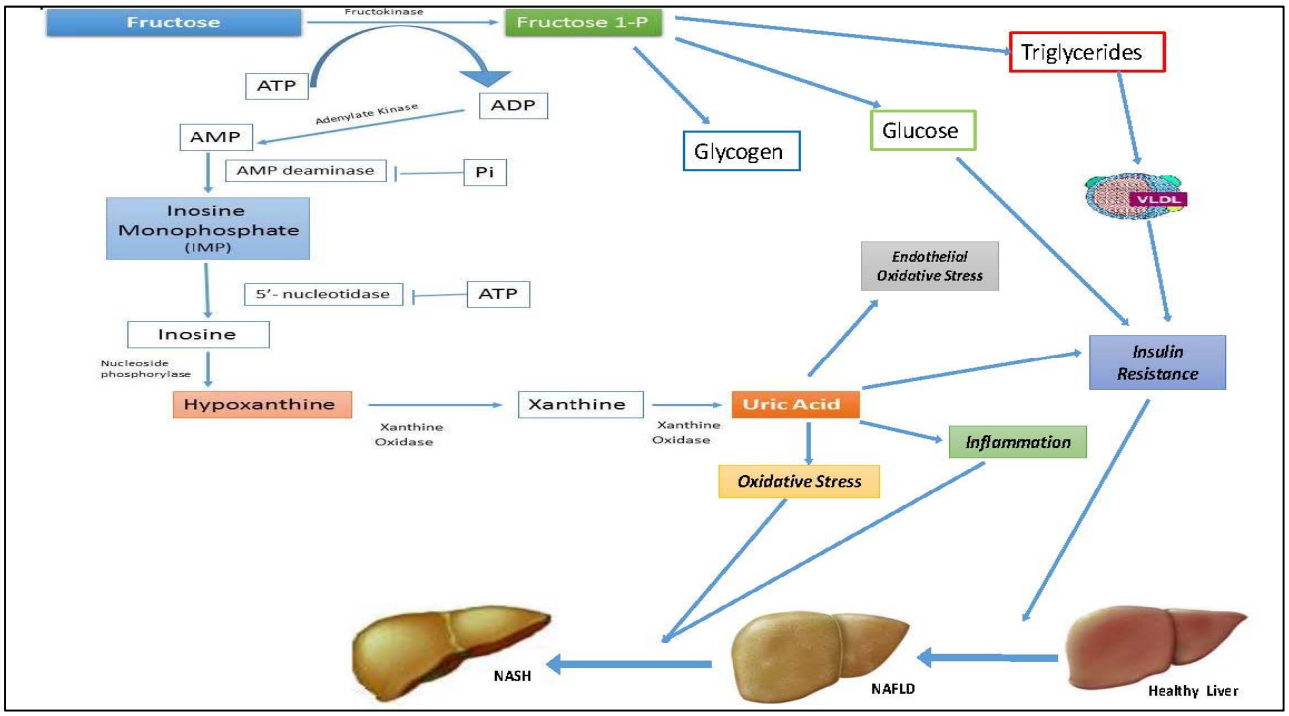
Amsterdam, The Netherlands, February 14, 2017 – Recent research suggests that dietary fructose intake may increase serum uric acid concentrations and that both uric acid concentration and fructose consumption may be increased in individuals with non-alcoholic fatty liver disease (NAFLD). Investigators have now established that both dietary fructose consumption and serum uric acid concentrations are independently associated with non-alcoholic steatohepatitis (NASH). Their conclusions are published in the Journal of Hepatology.

NAFLD, the accumulation of extra fat in liver cells in people who drink little or no alcohol, is recognized as the fastest growing cause of liver disease in both Western and developing countries. It is estimated to affect up to 30% of the general population in Western countries and up to 9.6% of all children and 38% of obese children across a spectrum of liver disease, including NASH (defined as steatosis, hepatocyte ballooning and inflammation). Although NASH is a less aggressive form of NAFLD, it can progress to severe fibrosis and cirrhosis, with development of hepatocellular carcinoma in adults.

“It is plausible that dietary fructose intake and uric acid concentrations are potential risk factors for liver disease progression in NAFLD. Numerous studies have shown that high uric acid levels are associated with metabolic syndrome and NAFLD, but to date, to the best of our knowledge, no studies have tested the independence of associations among uric acid concentrations, fructose consumption, and NASH confirmed by biopsy,” explained senior investigator Valerio Nobili, MD, Chief of the Hepatometabolic Unit Liver Diseases Laboratory, Bambino Gesù Hospital, IRCCS, Rome, Italy.

A team of researchers in Italy and the UK studied 271 obese children and adolescents with NAFLD (155 males, mean age 12.5 years) who underwent liver biopsy. All patients completed a food frequency questionnaire, indicating when specific foods were consumed (breakfast, morning snack, lunch, afternoon snack, dinner, etc.), how often (every day of the week, sometimes, or never), and portion size. Major sources of dietary fructose among children and adolescents are soda and other sweetened beverages. Nearly 90% reported drinking sodas and soft drinks one or more times a week. Almost 95% of patients regularly consumed morning and afternoon snacks consisting of crackers, pizza and salty food, biscuits, yogurt, or other snacks.

In the group of patients studied, 37.6% of patients had NASH and 47% of patients with NASH had high uric acid compared with 29.7% of patients who did not have NASH. Fructose consumption was independently associated with high uric acid, which occurred more frequently in patients with NASH than in not-NASH patients.



Caption: Depiction of how fructose consumption is related to an increase of serum uric acid concentrations (via xanthine pathway) and the progression liver damage (via oxidative stress).

“In this study, we show for the first time that uric acid concentrations and dietary fructose consumption are independently and positively associated with NASH. The development of NASH may markedly affect life expectancy and quality of life in affected individuals and therefore it is crucial to understand the risk factors for NASH in children and adolescents in order to design effective interventions which can be used safely to treat this young group of patients,” Dr. Nobili concluded.

Efforts geared towards behavior modification, nutrition education, and limiting access to soda and other sweetened beverages could potentially reduce fructose consumption in this particular population. Several countries have already launched campaigns to ban soda vending machines in schools.

Notes for Editors

The article is “Serum uric acid concentrations and fructose consumption are independently associated with NASH in children and adolescents,” by Antonella Mosca, Valerio Nobili, Rita De Vito, Annalisa Crudele, Eleonora Scorletti, Alberto Villani, Anna Alisi, and Christopher D. Byrne (http://dx.doi.org/10.1016/j.jhep.2016.12.025). It will be published in the *Journal of Hepatology*, volume 66, issue 5 (May 2017) by Elsevier.

Full text of this article is available to credentialed journalists upon request; contact Sybrand Boer Iwema at +31 20 485 2781 or hmsmedia@elsevier.com. Journalists wishing to interview the authors should contact Valerio Nobili at nobili66@yahoo.it. For questions concerning the *Journal of Hepatology*, please contact Editor-in-Chief Rajiv Jalan at hmsmedia@elsevier.com.

For additional information on the topic of the toxic effects of fructose on the liver the Editors of the *Journal of Hepatology* recommend visiting www.sugarscience.org/the-toxic-truth/#.WJIRdxAqXvA.

About the *Journal of Hepatology*

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