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Fructose consumption as a risk factor for non-alcoholic fatty liver disease.

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Abstract

BACKGROUND/AIMS: While the rise in non-alcoholic fatty liver disease (NAFLD) parallels the increase in obesity and diabetes, a significant increase in dietary fructose consumption in industrialized countries has also occurred. The increased consumption of high fructose corn syrup, primarily in the form of soft drinks, is linked with complications of the insulin resistance syndrome. Furthermore, the hepatic metabolism of fructose favors de novo lipogenesis and ATP depletion. We hypothesize that increased fructose consumption contributes to the development of NAFLD.

METHODS: A dietary history and paired serum and liver tissue were obtained from patients with evidence of biopsy-proven NAFLD (n=49) without cirrhosis and controls (n=24) matched for gender, age (+/-5 years), and body mass index (+/-3 points).

RESULTS: Consumption of fructose in patients with NAFLD was nearly 2- to 3-fold higher than controls [365 kcal vs 170 kcal ($p<0.05$)]. In patients with NAFLD (n=6), hepatic mRNA expression of fructokinase (KHK), an important enzyme for fructose metabolism, and fatty acid synthase, an important enzyme for lipogenesis were increased ($p=0.04$ and $p=0.02$, respectively). In an AML hepatocyte cell line, fructose resulted in dose-dependent increase in KHK protein and activity.

CONCLUSIONS: The pathogenic mechanism underlying the development of NAFLD may be associated with excessive dietary fructose consumption.

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