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Nutrition in nonalcoholic fatty liver disease (NASH)

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Excessive accumulation of triglycerides (TG) in liver, in the absence of significant alcohol consumption is nonalcoholic fatty liver disease (NAFLD). NAFLD is a significant risk factor for developing cirrhosis and an independent predictor of cardiovascular disease. High fructose corn syrup (HFCS)-containing beverages were associated with metabolic abnormalities, and contributed to the development of NAFLD in human trials. Ingested carbohydrates are a major stimulus for hepatic de novo lipogenesis (DNL) and are more likely to directly contribute to NAFLD than dietary fat. Substrates used for the synthesis of newly made fatty acids by DNL are primarily glucose, fructose, and amino acids. Epidemiological studies linked HFCS consumption to the severity of fibrosis in patients with NAFLD. New animal studies provided additional evidence on the role of carbohydrate-induced DNL and the gut microbiome in NAFLD. The excessive consumption of HFCS-55 increased endoplasmic reticulum stress, activated the stress-related kinase, caused mitochondrial dysfunction, and increased apoptotic activity in the liver. A link between dietary fructose intake, increased hepatic glucose transporter type-5 (Glut5) (fructose transporter) gene expression and hepatic lipid peroxidation, MyD88, TNF-α levels, gut-derived endotoxemia, toll-like receptor-4, and NAFLD was reported. The lipogenic and proinflammatory effects of fructose appear to be due to transient ATP depletion by its rapid phosphorylation within the cell and from its ability to raise intracellular and serum uric acid levels. However, large prospective studies that evaluated the relationship between fructose and NAFLD were not performed yet.

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