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Fructose Metabolism and Health Risks

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Mini Review

Fructose is a monosaccharide mainly found in fruits, vegetables and honey [1]. It has been commonly consumed in human diet, however the daily intake has increased in the past 40 years due to its industrial production [2]. High fructose corn syrup (HFCS), also called glucose or fructose syrup, is used as a sweetener in many products such as sweets, cakes and drinks. Consumption of fructose as a single nutrient is rare and its absorption decreases due to high fiber content of its natural sources. Chemical structures of glucose and fructose are presented in Figure 1.

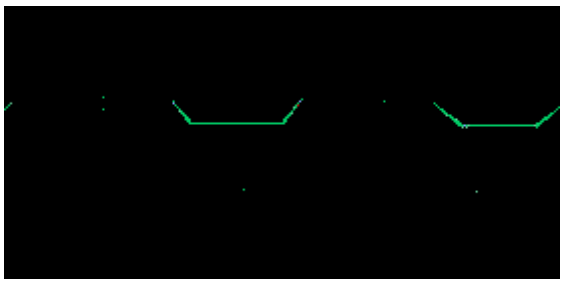

Figure 1: Chemical structures of glucose and fructose.

The main source of fructose in human diet is sucrose or HFCS that is composed of fructose and glucose in different ratios such as HFCS 55 indicating 55% fructose [1,2]. These sweeteners are preferred in food industry because they are much cheaper and have better functional characteristics.

In recent years, there has been a parallel increase in dietary exposure to fructose as well as in the incidences of metabolic diseases. This leads to the idea that consumption of high fructose may have a negative impact on human health. In a number of scientific reports, dietary fructose has been implicated in several diseases. In a study carried out with young men and women, high fructose corn syrup was reported to increase postprandial triglycerides, LDL cholesterol, and Apolipoprotein-B which are indicators of cardiovascular disease risk factors [3]. In a cross-sectional analysis of the data collected from the National Health and Nutrition Examination Survey NHANES from 2003 to 2006, high fructose intake was determined to be significantly associated with elevated blood pressure levels [4]. Recent literature survey based on article search in scientific databases including PubMed after the year 2000, high fructose intake was assessed to be associated to nonalcoholic fatty liver disease and fructose malabsorption [5]. However, there are also contradictory results extracted from human studies reporting no association between high fructose intake and liver diseases [6] and insufficient association was reported due the data obtained from meta analysis of studies on children and adults [7].

Fructose absorption and metabolism

Fructose either from the natural sources or HFCS is transported across the intestinal epithelium by the facilitative glucose transporter GLUT5 of the enterocytes [8]. Fructose in the cell is transported through blood by GLUT2 located on the basolateral surface of the enterocytes. GLUT2 is Na^+ dependent whereas GLUT5 is Na^+ independent and Km of GLUT5 for fructose is approximately 10 times lower than GLUT2 [9,10]. GLUT2 is not constitutively located on that membrane but it is reported to be transiently upregulated by glucose [8]. GLUT2 recruitment to the brush border membrane of human is thought to be an important adaptation when exposed to high doses of fructose [11]. GLUT5 is detected in the small intestine, kidney, heart, skeletal muscles and the brain, and in plasma membranes of adipocytes [12].

Fructose in the blood is transported to the liver where it is mainly metabolized. In the muscles and kidneys, fructose can be phosphorylated by hexokinase as a major pathway, however in the liver, fructose is metabolized by fructokinase into fructose-1-phosphate which is converted to glyceraldehyde and **dihydroxyacetone phosphate** (DHAP) by **fructose-1-phosphate aldolase (aldolase B)**. DHAP is converted into glyceraldehyde-3-phosphate (GA3P) by triose phosphate isomerase. Glyceraldehyde is phosphorylated by ATP and triose kinase to GA3P. Therefore, both products of fructose hydrolysis enter the glycolytic pathway as GA3P [10] (Figure 2).

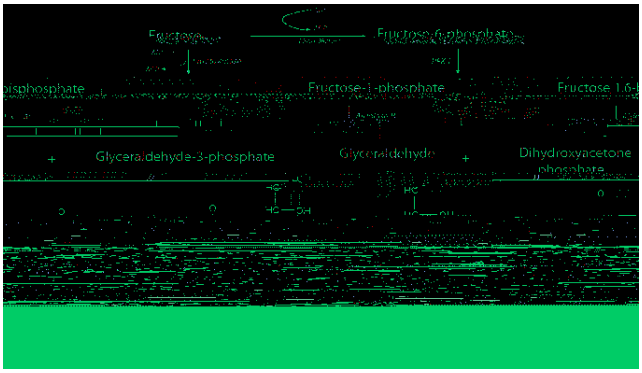

Figure 2: Fructose metabolism in liver.

Glucose and fructose carbons are utilized through the glycolysis, gluconeogenesis, glycogenolysis, tricarboxylic acid cycle, Cori cycle,

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