

Inborn Metabolism Errors and Cancer: Fructose and Mannose

Amelia Williams *

Editorial Office, Organic Chemistry: An Indian Journal, UK

*Corresponding author: Amelia Williams, Editorial Office, Organic Chemistry: An Indian Journal, UK

E-Mail: organicchem@journalres.com

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EDITORIAL

A significant phase in the cultural transition from nomadism to sedentism was the cultivation of grains to provide a steady source of carbohydrates. Humans have evolved improved glucose consumption as the primary carbon source for catabolic and anabolic pathways, as well as long-term storage of glucose as glycogen. Glucose, the principal dietary monosaccharide with six carbon atoms (hexose), is an essential component of a healthy diet, and as a result, the study of glucose metabolism has received a lot of attention. Dysregulations of glucose metabolism-specifically, perturbations in central carbon metabolism (Glycolysis, Pentose Phosphate Pathway (PPP), and Tricarboxylic Acid (TCA) cycle)-have been identified as key steps not only in metabolic disorders (e.g., obesity, insulin intolerance, and nonalcoholic fatty liver disease) but also in cancer progression through a variety of mechanisms and interactions. However, glucose is not the only hexose processed by the cell; fructose and mannose are two more significant hexoses used by cells for energy production and complex cellular processes like glycosylation. While fructose and mannose are not required in the human diet, the importance of metabolism in disease has pushed the discipline to emphasise the study of these sugars. Since its discovery by Augustin-Pierre Dubrunfaut in 1847 and the explication of its configuration by Emil Fischer's stereochemistry studies in the late 1800s, fructose, often known as fruit sugar, has been extensively investigated. Due to the increased availability of sucrose and high-fructose corn syrup, fructose has become more prevalent in the Western pattern diet. Fructose has been demonstrated to play a historically and statistically important influence in the growing prevalence of obesity and metabolic syndrome since it has become a staple of the average diet in the form of processed breads and colas. Given that central obesity, hyperglycemia, dyslipidemia, and hypertension are all diagnostic criteria for metabolic syndrome, fructose's role in the development of high morbidity conditions underscores the importance of studying and understanding fructose metabolism as it relates to disease development and progression. Enzymatic deficits in the fructose metabolism pathway, as well as inadequate enzymatic performance, can lead to poor metabolic outcomes. Fructokinase deficiency, aldolase B deficiency, and fructose-1,6-bisphosphatase deficit are the three recognized inborn defects in the fructose metabolic pathway. These limitations highlight the relevance of fructose metabolism via anabolic and catabolic mechanisms, such as fructose entrapment in the cell, fructose contribution to metabolic intermediates, and fructose functions in glycogenesis. The process of fructose metabolism and the consequent energy realisation is known as fructolysis. The phosphorylation of fructose to fructose-1-phosphate (F1P) by fructokinase is the first step in fructolysis once fructose enters the cells. Fructokinase, sometimes called ketohexokinase (KHK), is an ATP-dependent enzyme that converts fructose to Fructose-1-Phosphate (F1P). Fructokinase A (KHK-A) and Fructokinase C (KHK-C) are two isoforms of fructokinase produced by alternative splicing of the KHK gene on chromosome

(KHK-C). KHK-C is mostly expressed in the liver, kidney, and gut, whereas KHK-A is found throughout the body. While KHK-C plays the most essential function in liver fructose metabolism, whole-body deletion of both isoforms was found to be much more effective in preventing metabolic syndrome, showing that KHK-A plays an important role in fructose metabolism in extrahepatic tissues with low KHK-C expression. Small amounts of fructose are normally digested by the gut, but when fructose levels surpass the capability of intestinal fructose absorption, fructose can be processed by the liver and colon bacteria.