Special Issue

Fructose Metabolism and Diabetes – Where Do We Stand Now?

Message from the Guest Editor

Fructose is a monosaccharide with a higher protein glycolytic capacity than glucose that metabolizes faster than acetyl CoA due to the absence of rate-limiting enzymes such as glucokinase. It is also considered more toxic than glucose. Excessive sucrose intake is associated with obesity, hyperuricemia, type 2 diabetes, dyslipidemia, NAFLD, frailty, and cancer. Moreover, fructose has been thought to be metabolized in the liver, not in the small intestine. However, recent studies have shown that small amounts of fructose are completely converted to glucose, and its metabolites have been found in the small intestine. Furthermore, it has been shown that unabsorbed fructose is converted to acetic acid by intestinal bacteria, absorbed through the portal vein, and used for lipid synthesis. Therefore, when considering the mechanisms underlying fructose-induced fatty liver development, it is important to consider the role of the small intestine and even the intestinal microbiota in addition to the conventional liver. Furthermore, exogenous as well as endogenous fructose has been reported to cause renal damage.

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Nutrients is an on-line open access journal that was first published in 2009. *Nutrients* adheres to rigorous peer-review and editorial processes and publishes only high quality manuscripts that address important issues related to the impacts of nutrients on human health. The Impact Factor of *Nutrients* has risen rapidly since its establishment and it is now ranked in the first quartile of journals publishing in the field of nutrition and dietetics research.

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