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Kidney disease induced by high fructose intake

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Abstract

Fructose intake is advised to regulate serum glucose concentration and diabetes. However, high intake of fructose has been shown to be associated with the outbreak of obesity, diabetes mellitus, hypertriglyceridemia and also non-alcoholic fatty liver disease. Furthermore, it seems to elevate the risk of gout and hence kidney stones. In this regard, the mechanism of fructose on the induction of chronic kidney disease is ascribed to its role in hypertension and diabetes. With respect to the contradictory reports of fructose intake on diabetes, the aim of this review is to explain this inconsistence and its effect on induction of renal disease.

Keywords: Gout, Chronic kidney disease, Fructose, Renal disease, Hyperinsulinemia, Hypertriglyceridemia

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Introduction

It has been recorded that approximately 24 million people in the United States manifest diabetes and 180 000 people suffer from chronic kidney disease amongst diabetic people (1). The main source of fructose in diet is free fructose and half of sucrose intake (2). Intake of high fructose corn syrup is usually replacing sucrose in processed feeds, because of being inexpensive and sweeter, resulting in induction of overeating in humans (3). Fruit and vegetables are rich in fiber;as the consequence, it delays the absorption of carbohydrates after a meal and lowers insulinemic response to its carbohydrates (4). Feeding fructose in diet led to the improvement in glycemic control and also the greater insulin sensitivity (5). Although patients suffering from diabetes were advised to consume fruit sugar (fructose), individuals with diabetes mellitus type 2 have problem with protein and lipid metabolisms not with sugar (6); because fructose intake has no negative effect on blood glucose level in the short time, but it adversely affect the other aspects of metabolism (6). After the absorption of fructose in small intestine, it transports to liver through blood circulation (7). Lipogenesis and triglyceride accumulation in the liver are started when it is exposed to large quantities of fructose;as the consequence, it leads to the decreased insulin sensitivity and hence the induction of hepatic resistance (8). Excessive intake of fructose in animals results in the increased insulin, lipids and uric acid levels in blood (9-11). Administration of 60% fructose in diet of rats for 10 weeks induces hypertriglyceridemia, hyperinsulinemia and hyperuricemia (12). Decreasing uric acid in animals fed on high fructose is considered to inhibit or reverse metabolic syndrome including hyperinsulinemia and hypertriglyceridemia (12).

Objectives

Since fructose had desirable effects on health in particular regulating diabetes, many scientists proposed it to preserve health. However, its influences on health are dependent on dosage, duration of intake, strain, and physiological status of body. Thus, the aim of this study is to find whether fructose intake has positive impact on health or it induces diseases.

Materials and Method

For this mini-review, we used a variety of sources by searching through Web of Science, PubMed, directory of open access journals (DOAJ), EMBASE and Scopus. The search was performed using combinations of the following key words and or their equivalents such as gout, chronic kidney disease, fructose, renal disease, hyperinsulinemia and hypertriglyceridemia.

Fructose and diabetes

Diabetes has been remarkably increased around the world. In this case, it has been estimated that the incidence of diabetes mellitus was increased up to 8.3% in the United States (13). Fructose is considered to exist in nature of fruits as sucrose. Drinking fruit juice absorbs easily via the intestinal walls and in turn, increasing blood sugar level, while, drinking fruit in drink decreases protein, calcium and vitamin D levels (14,15). Interestingly, it has been recommended that high fruit intake prevented various diseases especially type 2 diabetes (16). Although eating fruit prevents diabetes, drinking fruit juice elevates

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Implication for health policy/practice/research/ medical education

High intake of fructose has been shown to be associated with the outbreak of obesity, diabetes mellitus, hypertriglyceridemia and also non-alcoholic fatty liver disease. Furthermore, it seems to elevate the risk of gout and hence kidney stones. In this regard, the mechanism of fructose on the induction of chronic kidney disease is ascribed to its role in hypertension and diabetes.

the risk of diabetes (17). Notably, diabetic patients had higher serum and urine fructose concentrations in comparison with healthy people (18). Though Muraki et al (19) reported that high intake of specific whole fruits including blueberries, grapes and apples reduced the risk of type 2 diabetes, higher intake of fruit juice increased the risk of type 2 diabetes. Moreover, the long-term intake of moderate content of fructose depressed glucose tolerance (20), feeding high fructose in diet induced diabetes mellitus (21). The adverse effect of fructose on health is resulted from its metabolism after food intake (7). Fructose caused less insulin secretion rather than glucose did (22).

Taken fructose is absorbed through small intestine as much as 80% to 90% by facilitate diffusion (23). Fructose as a safe form of sugar is advised to be administrated, because fructose does not need the insulin for uptake into the cells (22). When the liver is exposed to large quantities of fructose, it results in rapid induction of lipogenesis and consequently triglyceride accumulation (8,24). This, in turn, causes the decrease in insulin sensitivity and the stimulation in hepatic insulin resistance (8). Shapiro et al (25) detected that administration of high fructose in diet causes leptin resistance in rats and as the result, it induces obesity. In another study, Faeh et al (26) showed that consumption of 3 g fructose in kg of body weight per day led to an increase in total caloric intake up to 25% and induction of insulin resistance after 6 days as compared to control diet.

Fructose and renal disease

Kidney, referred as one of the vital organs, is adversely affected by diabetes (27). Diabetic kidney disease is considered to be a cause of end stage kidney disease. Notably, it is responsible for 44% of kidney disease in the United States (13,28). Remarkably, chronic kidney disease is occurred even if diabetes is controlled (29). Diabetic kidney disease takes several years. At first, small amounts of albumin start to penetrate into the urine, which is called microalbuminuria. Kidney functions normally in this stage (30,31). More albumin penetrates into the urine, when the disease advances, which is referred as macro-albuminuria. The filtration of kidney depresses as the disease progresses (32).

In spite of suffering patients with diabetes type 2 from kidney disease (approximately 30%), but they do not have diabetic nephropathy (33). Nephropathy incidence is occurred 10 to 20 years after diabetes (34). The outbreak of diabetes type 2 is 9-fold higher than diabetes type 1 (34). The constituents of urine are impacted by dietary habits (35). Feeding high fructose in diet results in the increased serum uric acid concentrations (36). Moreover, intake of large amounts of fructose in men for 12 days markedly raised both serum and urinary uric acid levels (37). The interesting point is that fructose ingestion causes an increase in uric acid excretion (38). Fructose is positively related to uric acid caused by diabetes (39). Cunha et al (40) observed that high fructose intake impaired renal function in mice as evidenced by the increases in urinary protein excretion and hence urinary volume. Similarly, Gersch et al (41) reported that consumption of high fructose increased the progression of kidney disease in rats with problems in renal function. Furthermore, Asselman and Verkoelen (42) reported that high fructose intake is independently related to kidney stone disease. High fructose intake leads to outbreak of renal hypertrophy, afferent thickness, and glomerular hypertension (43). Hence, it led to glomerulosclerosis and tubulointerstitial fibrosis. It has been established that fructose causes kidney hypertrophy in rats through two pathways. First, fructose increased uric acid resulting in an afferent arteriolopathy and therefore glomerular hypertension (43). Second, fructose can filter into the urine;in turn, it induced intracellular generation of uric acid and hence local inflammation (44).

Fructose and hypertriglyceridemia

Numerous investigations reported that the high intake of fructose negatively affect health via increasing the adiposity. It has been demonstrated that excessive fructose intake induces hepatic de novo lipogenesis;as the result, it stimulates hypertriglyceridemia (26). In this regard, hypertriglyceridemia, hyperinsulinemia and hyperurecemia were stimulated after, rats administrated 60% fructose in diet (12). Teff et al (45) found an increase in serum triglyceride levels in women administrated high fructose in meals. However, some studies showed that consumption of excessive fructose had no undesirable influence on serum lipids in healthy subjects (46,47). Additionally, some studies have reported that induction of nonalcoholic fatty liver disease might be attributable to excessive dietary fructose intake (48,49).

Conclusion

High intake of fructose has been shown to be associated with the outbreak of obesity, diabetes mellitus, hypertriglyceridemia and also non-alcoholic fatty liver disease. Furthermore, it seems to elevate the risk of gout and hence kidney stones. In this regard, the mechanism of fructose on the induction of chronic kidney disease is ascribed to its role in hypertension and diabetes.

Author's contribution

HN is the single author of the manuscript.

Conflicts of interest

The author declare no conflict of interests.

Ethical considerations

Ethical issues (including plagiarism, data fabrication, double publication) have been completely observed by the author.

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