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REVIEW PAPER

FRUCTOSE – AN EFFECT ON METABOLIC DISORDERS*

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Abstract

Fructose is a monosaccharide, a component of some disaccharides. It is the sweetest of all simple sugars; it delivers, like other carbohydrates, 4 calories per 1 g. Fructose absorption occurs in the duodenum according to the concentration gradient. It is the highest when the same amount of glucose is absorbed at the same time. Absorption also improves adaptively with higher dietary fructose intake. Fructose is a substrate in the lipogenesis process (a precursor of acetyl-CoA and 3-phosphoglycerol aldehyde), leading to the formation of triglycerides, which, in the absence of fructose metabolism regulation, may contribute to dyslipidemia and nonalcoholic fatty liver disease. Excessive consumption of fructose may also increase the formation of uric acid in the liver and thus increase the risk of cardiovascular disease and contribute to the accumulation of adipocytes in the adipose tissue. The effect of fructose on the reduction of insulin sensitivity of tissues, and thus on the development of type 2 diabetes and metabolic syndrome, is also described. Until now, no recommendations for population have been issued regarding the consumption of fructose alone. The WHO recommends – to both adults and children – limiting the intake of simple sugars to 10% of the energy value of the diet. Reduction in fructose consumption is recommended to people with diabetes and cardiovascular diseases. Most fructose is found in sweet drinks and products sweetened with sucrose, fructose syrup and fructose. There have been reports of various health effects of fructose syrups and fructose naturally occurring in products.

Keywords: fructose, obesity, metabolic disorders, diet.

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INTRODUCTION

Fructose is one of carbohydrates referred to as monosaccharides. It does not usually occur in nature in the form of free molecules, but as a component of disaccharides (fructose in sucrose) and polysaccharides (e.g. fructose in inulin). As a monosaccharide, fructose is naturally found in honey and in smaller amounts in fruit and vegetables. It is also added during the production of processed food (Mahan et al. 2011). Over the past two centuries, a 100-fold increase in per capita consumption of dietary fructose has been noted, currently accounting for about 10% of daily caloric intake in the United States (Jang et al. 2018).

Fructose absorption occurs in the duodenum and the beginning portion of the small intestine (Mahan et al. 2011, Sadowska, Rygielska 2014). Simultaneous absorption of the same amount of glucose makes the process the most intense, while the absence of glucose as well as the presence of sucrose or sucrose enzyme inhibitors suppress fructose absorption. The absorption also adaptively increases with more marked dietary intake (Sadowska, Rygielska 2014).

The liver is mostly responsible for metabolizing fructose, where it enters the metabolic pathway of glucose, at the same time bypassing the main controlling enzyme in the process of glycolysis – phosphofructokinase. Therefore, its metabolism is not regulated (Mahan et al. 2011, Miller et al. 2011, Sadowska, Rygielska 2014, Sievenpiper et al. 2014, Grupińska et al. 2015). The absence of control in fructose metabolism results in the lack of regulating triglyceride formation in the liver, which leads to the development of dyslipidemia and nonalcoholic fatty liver disease (NAFLD) (Sievenpiper et al. 2014). High fructose consumption is also considered a culprit in metabolic disease. Epidemiological studies indicate a strong correlation between high fructose intake and obesity, type 2 diabetes, kidney dysfunction and cardiovascular disease (Caliceti et al. 2017, Jegatheesan, De Bandt 2017), but the biological mechanisms underlying this link are still controversial (Macdonald 2016, Jang et al. 2018).

STRUCTURE AND PROPERTIES

The most important monosaccharides are glucose, galactose and fructose. They are hexoses made of six carbon atoms. Their chemical formulae are the same, but they differ in the chemical structure which facilitates the formation of isomers such as glucose and galactose. In the solid state glucose, fructose and galactose may only have the ring structure, while in aqueous solutions balance is reached between the chain and ring structure. Fructose and glucose form sucrose, a disaccharide which naturally occurs in a variety of food. It is also added during the production of processed food. Fructose and glucose are components of inverted sugar which is found in honey, numerous

fruits, table sugar, high-fructose corn syrup, in which the glucose-fructose ratio is 1 to 1, and others. Fructose is the sweetest monosaccharide. Inverted sugar is frequently used in food industry, because it is sweeter than the same amount of sucrose, which makes food processing more efficient. It is used in candy and icing production due to the fact that its crystals are finer than those of sucrose. Similarly to other absorbable carbohydrates, ingested fructose delivers 4 kcal per g of energy available for the body.

Fructans are fructose polymers (sometimes binding with glucose molecules) which include fructooligosaccharides (FOS), inulin, inulin-type fructans and oligofructose. Inulin belongs to a heterogeneous group of fructose polymers and is a carbohydrate storage in plants. Oligofructose is a subgroup of inulin, containing fewer than 10 fructose molecules. Fructans are poorly digested in the gastrointestinal tract with 1 g delivering only 1 kcal of energy. They are considered prebiotics due to their ability to stimulate the growth of beneficial intestinal microflora and a minor degree of digestibility in the gastrointestinal tract. Fructans have a sweet clean flavour and are half as sweet as sucrose (Mahan et al. 2011).

METABOLISM IN HUMAN BODY

Monosaccharides, including fructose, are absorbed in the duodenum and the beginning portion of the small intestine. Sucrase, a brush border enzyme, disintegrates sucrose to glucose and fructose. Fructose absorption occurs according to the concentration gradient. Its most abundant absorption occurs with simultaneous absorption of the same amount of glucose, while the absence of glucose, the presence of sucrose or sucrose enzyme inhibitors suppress fructose absorption. Its absorption also adaptively increases with more marked dietary intake. Fructose passes through intestinal mucous membrane cells and reaches the bloodstream via villus capillaries, from where it is carried to the liver by the portal vein. By means of a facilitative transporter fructose is absorbed from the intestinal lumen to enterocyte interior with glucose transporter 5 (GLUT 5) located on its cell membrane. Its transport from the cell to the bloodstream occurs mainly with the use of GLUT 2 transporter (Mahan et al. 2011, Sadowska, Rygielska 2014, Grupińska et al. 2015).

Most fructose is metabolized in the liver, where it enters the metabolic pathway of glucose, but it bypasses the main controlling enzyme in the process of glycolysis – phosphofructokinase. Therefore, its metabolism is not regulated (Mahan et al. 2011, Miller et al. 2011, Sadowska, Rygielska 2014, Sievenpiper et al. 2014, Grupińska et al. 2015). Fructose is a substrate in the lipogenesis process in the liver. It is a precursor of such substances as 3-phosphoglycerol aldehyde, pyruvate, acetyl-CoA (Czyżewska et al. 2010, Mahan et al. 2011, Sadowska, Rygielska 2014). Acetyl-CoA undergoes transformation to malonyl-CoA (an initial product of lipogenesis), which inhibits fatty acid ß-oxidation and enhances hepatic lipogenesis (Sadowska, Rygielska

2014). According to classic biochemistry, the absence of control in fructose metabolism is responsible for the lack of regulation of triglyceride formation in the liver, which leads to the development of dyslipidemia and nonalcoholic fatty liver disease – NAFLD (Sievenpiper et al. 2014). Lipid synthesis is also intensified in adipocytes. Glucose absorbed with fructose stimulates insulin secretion, which results in the intensification of the described processes (Sadowska, Rygielska 2014). Moreover, fructose generates the production of ADP (adenosine diphosphate) in the liver, which is a substrate for uric acid synthesis. Enhanced uric acid production in the liver may increase the risk of cardiovascular diseases (Bray 2010).

EFFECT ON HUMAN HEALTH

Fructose does not cause dental caries and it shortens the duration of alcohol metabolism. Fructose consumption in the form of isolated monosaccharide may result in its incomplete absorption and cause intensified gas production and diarrhoea. High-fructose diet, including fructose-sweetened beverages, may be responsible for ectopic fat deposition (fat storage within e.g. muscles, the liver, i.e. away from the sites of its natural deposition) and for the accumulation of adipocytes in visceral adipose tissue. It may also cause fat storage resulting from enhanced hepatic lipogenesis (Bray 2010, Miller et al. 2011, Kłosiewicz-Latoszek, Cybulska 2011, Welsh et al. 2011, Artym 2012, Evert et al. 2014) leading to obesity (Bray 2010, Mahan et al. 2011, Kłosiewicz-Latoszek, Cybulska 2011).

Additionally, fructose leads to unfavourable changes in blood lipid concentrations as it enhances the synthesis of low-density lipoprotein cholesterol (LDL-C), triglycerides (TG) and it elevates triglyceride concentration and the chylomicron remnants to a larger extent than glucose (ADA 2008, Bray 2010, Czyżewska et al. 2010, Miller et al. 2011, Kłosiewicz-Latoszek, Cybulska 2011, Welsh et al. 2011, Evert et al. 2014). It also contributes to the reduction in high-density lipoprotein cholesterol (HDL-C) concentration in the blood, the development of atherosclerosis and cardiovascular diseases. Numerous authors stated that it may also promote the development of gout and increased blood pressure. However, the data are ambiguous (Bray 2010, Kłosiewicz-Latoszek, Cybulska 2011, Welsh et al. 2011, Evert et al. 2014, Stanhope 2017, Wu et al. 2017, Rodrigues et al. 2018). The results of a double-blind randomized trial revealed that pure fructose or glucose-fructose syrup consumed at the 50th percentile level for the population did not cause hyperuricaemia or increased blood pressure (Angelopoulos et al. 2015).

A negative influence of fructose on insulin sensitivity has also been reported, although it stimulates insulin secretion to a lesser extent than glucose. It is thought that the said influence leads to the development of type 2 diabetes mellitus and metabolic syndrome although it increases thermogenesis (Bray 2010, Mahan et al. 2011, Kłosiewicz-Latoszek, Cybulska 2011, Evert et al. 2014, Stanhope 2017). A meta-analysis of 15 studies which con-

centrated on fructose consumption with processed food, including beverages, showed a significant influence of fructose consumption on fasting glucose increase, TG, systolic blood pressure and a decrease in HDL-C and total cholesterol – TC (Kelishadi et al. 2015).

According to the American Heart Association (AHA), the consumption of fructose in amounts of ≤ 100 g per day does not lead to a marked increase in fasting TG concentration, regardless of whether it substitutes other carbohydrates in the diet such as sucrose, starch or glucose. However, its consumption over 100 g per day is responsible for elevated blood triglyceride concentration, depending on a dose. The AHA emphasizes that there is evidence to corroborate an increase in the blood TG concentration with fructose consumed at 50 g per day, so it is recommended to limit its consumption to this amount in persons with an elevated blood triglyceride concentration (Miller et al. 2011). The Canadian Diabetes Association (CDA) emphasizes in the recommendations that fructose consumed at > 10% of daily energy supply or > 60 g per day may slightly increase the blood TG concentration in type 2 diabetes mellitus patients (Dworatzek et al. 2013).

There is also a hypothesis which assumes that fructose contributes to lowering the amount of adenosine triphosphate (ATP) in the cell, which results in an increase in the uric acid concentration in the body, which in turn contributes to the development of metabolic syndrome. Fructose may also disrupt signals coming from hormones regulating the perception of hunger and satiety, such as leptin, ghrelin or insulin, stimulating their secretion to a lesser extent than glucose, which results in higher food intake, enhanced body weight increase and cardiometabolic complications (Sievenpiper et al. 2014).

Increased blood glucose concentration leads to the most intense secretion of insulin in the pancreas. Insulin activates a protein which regulates the synthesis of fatty acids and triglycerides. Regardless of insulin activity, proteins which stimulate the liver to synthesize triglycerides are also activated in response to the activity of carbohydrates such as fructose (Miller et al. 2011). A meta-analysis including 20 studies on the influence of the consumption of fructose and glucose (in isocaloric amounts) with an optimal energy supply with the diet on various biochemical parameters demonstrated:

- a positive influence of fructose compared to glucose on: body weight reduction, fasting glucose and insulin concentration reduction, a reduction in the concentration of glycated blood proteins (glycated haemoglobin – HbA1c, fructosamine and glycated albumins), a reduction in average and systolic blood pressure;
- no influence of fructose compared to glucose on the concentrations of: the majority of fasting blood lipids, NAFLD markers – lipid concentration inside hepatocytes and alanine aminotransferase (ALT);
- a negative influence of fructose compared to glucose on: increased fasting TC and uric acid concentration, postprandial triglycerides.

Excessive fructose or glucose supply (in isocaloric amounts) causing overconsumption of energy with diet compared to dietary requirements translated into:

- a positive influence of fructose compared to glucose on: a reduction in body weight increase;
- no influence of fructose compared to glucose on: fasting glucose and insulin concentrations, fasting blood lipid concentrations, glycated blood proteins, blood pressure, NAFLD markers;
- a negative influence of fructose compared to glucose on: increased fasting TC and uric acid concentration, postprandial TG.

The results of this meta-analysis suggested lower unfavourable activity of fructose compared to glucose, both with optimal and excessive dietary energy consumption (E). The authors also stated that it was still unknown whether fructose naturally occurring in fruit had a different effect on the body than that used as a sweetener, although no differences were reported. The effect of fructose also remains unknown if it replaces other healthier carbohydrates with a low glycaemic index, unsaturated fatty acids or its effect of long-lasting overconsumption (Sievenpiper et al. 2014).

There are few studies, especially those conducted in children, concerning the effects of fructose consumed with a diet on anthropometric and biochemical indexes. Couch et al. (2013) reported a significant relationship between fructose consumption and blood triglyceride concentration in children with type 1 diabetes mellitus. An increase in fructose consumption by 22 g (contained in approx. 340 ml of alcohol-free beverages) was associated with a triglyceride concentration increase of 4%. The effect of fructose consumption on concentrations of TC, LDL-C or HDL-C was not reported.

Pollock et al. (2012) studied a group of 14- to 18-year-old participants and reported higher concentrations of fasting insulin, homeostatic model assessment – insulin resistance (HOMA-IR), LDL-C, TG, more marked visceral adipose tissue mass and a lower concentration of HDL-C with an increase in fructose consumption. Higher fructose consumption in adolescents was associated with numerous markers of cardiometabolic risk, but the authors emphasized that these correlations were most probably indirectly related to visceral obesity. Kretowicz et al. (2011) reported no correlation between daily fructose consumption and the BMI (body mass index), LDL-C and TG in patients with chronic renal disease.

An interventional study was conducted on a group of American participants aged from 15 to 20 years. They were divided into two groups and served 710 ml of high-fructose beverages (HF – 50 g fructose and 15 g glucose) or high-glucose beverages (HG – 50 g glucose and 15 g fructose) over two weeks. Then, the influence on lipid and carbohydrate metabolism was assessed. It was demonstrated that the consumption of moderate quantities of HF and HG beverages over 2 weeks had no significant influence on cholesterol, TG and glucose concentrations, both fasting and postprandial, in physically active adolescents with ideal body weight (Heden et al. 2014).

Jin et al. (2014) studied two groups: one group was served a beverage containing 33 g of fructose 3 times a day and the other was given 3 portions of 33 g of glucose over 4 weeks. The participants consumed no other beverages during that period. The following parameters were assessed: changes in body weight, fasting TG, glucose and insulin concentrations, and HOMA-IR. No significant changes were reported as regards the values of these parameters ($p \geq 0.05$) both in the group with higher (high-fructose beverages) and lower (beverages with glucose) fructose consumption.

Sun et al. (2011) also reported no influence of fructose on triglyceride, HDL-C, glycated haemoglobin, uric acid concentrations, blood pressure, waist circumference and the BMI. In a study by Rodrigues et al. (2018), reduced postprandial glycaemia was achieved with the substitution of sucrose with fructose.

In another American study, various groups of patients were served different doses of glucose or fructose. The authors reported no influence of these carbohydrates on lipid metabolism indexes, fasting glucose, body weight, BMI, waist circumference or body fat percentage (Lowndes et al. 2014). However, a Swiss study conducted on young adult men (average age 26.3 ± 6.6 years), who were served 40 g of fructose daily for 3 weeks, and then, after a break, 80 g of fructose daily, showed a significant increase as regards: average fasting glucose concentration, high-sensitivity C-reactive protein (hsCRP) and waist to hip ratio (WHR) compared to baseline values. After 3 weeks of consuming 40 g of fructose per day, the average fasting glucose concentrations increased from 4.44 mmol L^{-1} to 4.72 mmol L^{-1}, and after 3 weeks of consuming 80 g of fructose daily, it increased from 4.44 mmol L^{-1} to 4.83 mmol L^{-1}. In both interventions the WHR values increased to 0.93 compared to the baseline value of 0.92 ($p < 0.01$), hsCRP levels increased from baseline 1.96 nmol L^{-1} to 3.57 nmol L^{-1} and 4.1 nmol L^{-1} ($p < 0.01$) with the respective consumption of beverages containing 40 and 80 g of fructose daily. After an intervention of 80 g of fructose, a significant ($p < 0.01$) increase was also reported as regards body fat percentage in study participants (from 15.4% to 15.7%). In both cases, the changes in body weight, BMI, waist circumference, glucose concentration measured 120 minutes postprandially and HOMA-IR values were not significant (Aeberli et al. 2011).

Maier et al. (2011) conducted a study among obese German children aged from 5 to 8 years. They reported that the average fructose consumption in this group was at the level of 46.3 g per day. The average percentage of energy coming from fructose was 11.8%. It was demonstrated in this study that diminished fructose consumption in children inhibited a BMI increase instead of reducing it, because the reduction in its consumption occurred simultaneously with a sucrose amount reduction and lowering the dietary energy in the study group.

RECOMMENDATIONS CONCERNING FRUCTOSE CONSUMPTION

It is estimated that the dietary fructose intake per capita has increased 100-fold over the last two centuries. It currently represents about 10% of the energy value of the diet (E) in the United States (Jang et al. 2018). Its consumption is the highest among adolescents aged 12-18 and delivers an average of 12.1%E, and one-fourth of teens consumes more than 15%E from fructose (Bray 2010). Despite worldwide scientific attention, the actual fructose consumption in many non-US populations is not clear. In the Netherlands, fructose consumption is 9% of the average daily energy in the general population aged 7-69 years. The main food sources of fructose are soft drinks, juices and fruit (Sluik et al 2015).

Recommendations concerning fructose consumption in the population are ambiguous. The World Health Organization recommends limiting the consumption of monosaccharides (also including fructose) to less than 10% of dietary energy value (E) both by adults and children. It is also recommended to reduce it further below 5%E (WHO 2015). Polish guidelines are similar, although it is considered that the recommendations for limiting less than 5%E from free sugars (mono- and disaccharides added to food at the stage of production and food processing and used by the consumer for sweetening, as well as sugars naturally found in fruit juices, syrups and honey) are currently difficult to implement in practice, because there are no legal regulations on the labeling of free sugars on products and the lack of tools to calculate this value in the diet (Jarosz et al. 2017). Similar dietary guidelines, updated in 2015, were published for Americans (Dietary Guidelines for Americans 2015-2020), which recommended the consumption of less than 10% of dietary energy coming from added sugars (USDA 2015), while the Institute of Medicine recommended not to exceed 25%E of daily added sugar consumption (IOM 2005).

Fructose consumption is discussed in the recommendations issued by the American Heart Association, recommendations for diabetic patients issued by the American Diabetes Association ADA, Canadian Diabetes Association CDA and Polish Diabetes Association PTD (Miller et al. 2011, Dworatzek et al. 2013, Evert et al. 2014, PTD 2017). According to the ADA recommendations for diabetic patients, the consumption of sweetened beverages (including those with added sucrose or fructose syrup) should be limited taking into consideration the risk of body weight increase and cardiometabolic risk. It is also emphasized that if fructose coming from fruit does not exceed 12%E, it is no more harmful than other sugars. It is also claimed that numerous products recommended for diabetics are rich in fructose, although it is not listed as an ingredient, but is added as, for example, agave syrup. Such products should not be consumed in excess by diabetic patients in order to avoid excessive fructose consumption and increased dietary energy value (Evert et al. 2014).

Moreover, fructose consumption as a sweetener is not recommended, also

by PTD. Additionally, diabetics are suggested to limit fructose consumption to 50 g per day (PTD 2017). However, there are no reasons for reducing naturally occurring fructose intake (e.g. fruit, vegetables), because these sources usually deliver only 3-4%E from fructose (ADA 2008).

According to the AHA recommendations (Miller et al. 2011), individuals with elevated blood triglyceride concentrations should consume limited amounts of fructose (Table 1).

Table 1

Recommended daily intake of fructose for people with elevated triglycerides (AHA 2011)

Concentration of triglycerides in the blood	Recommended daily intake of fructose
8.33 – 11.06 (mmol L^{-1})	< 100 g
11.11 – 27.72 (mmol L^{-1})	50 – 100 g
≥ 27.78 (mmol L^{-1})	< 50 g

FRUCTOSE CONTENT OF FOOD

Products which are a very good source of fructose (34 g^{-1} litre on average) include beverages (such as coke-type beverages) and products sweetened with sucrose, fructose syrup or fructose (Evert et al. 2014, NIHW 2017). Honey, fruit syrups, dried fruit and marmalades are also rich in fructose (NIHW 2017). Selected products with the highest content of fructose are presented in Table 2.

Fruit juices and concentrated fruit juices contain from approx. 20 g to 70 g of fructose L^{-1}. Beverages dedicated for sportsmen contain the most fructose in the group of all beverages – even 185 g of fructose L^{-1}. The amount of fructose in sweets varies (gummy candies contain 37 g; candy bars from 0 g to as much as 365 g; caramel – 54 g; cookies from < 1 g to 5 g; biscuits – 21 g of fructose kg^{-1} (NIHW 2017). Fructose content in fresh fruit ranges from below 1 g (avocado) to 75 g (persimmon), while in vegetables it ranges from < 1 g (horseradish) to 30 g (sweet yellow pepper) of fructose kg^{-1} – Table 3.

Nuts and seeds are not rich in fructose. Its small amounts may be found in almonds and hazelnuts – 5 g kg^{-1} and peanuts – 4 g kg^{-1}.

As regards legumes, different types of beans have the highest content of fructose (on average 8 g kg^{-1} of dry seeds). The lowest fructose content is found in green lentils, broad beans and green peas – 1 g kg^{-1} of dry seeds.

The richest fructose sources in the group of cereal products are muesli with dried fruit (approx. 40-60 g kg^{-1}) and cornflakes (23 g kg^{-1}). Unprocessed cereal products are low in fructose. Its highest content is found in wheat germ – 6 g kg^{-1} and wheat bran – 5 g kg^{-1}.

Milk products, such as drinking, flavoured and sweetened yoghurts, contain up to 45 g of fructose L^{-1}. Soy yoghurts contain from 1 g to 18 g

Table 2

Selected products with the highest fructose content (NIHW 2017)

Product	Content of fructose (g kg^{-1})
Fructose	998
Honey	414
Apples, dried	317
Apple crisps	311
Raisins	289
Fruit syrups	217
Figs, dried	201
Wild rose, dried fruit	195
Raisins in chocolate	168
Dried fruits, mix	166
Dried figs (with peel)	161
Orange marmalade	147
Blueberry marmalade	147
Dried plums marmalade	146
Dried dates, with seeds	145
Papaya, dried	132
Carrot, dried	130
Mango, dried	128
Dried plums, with seeds	121
Onion, dried	110
Tomatoes, dried in olive oil	105
Apricot, dried	100
Licorice	98
Mix of salted nuts and dried fruits	80

of fructose L^{-1}, while yoghurt ice cream – 18 g of fructose L^{-1}. Natural milk products contain no fructose.

As regards spices added to food, the highest fructose content may be found in dried pepper – 67 g of fructose kg^{-1} and dried oregano (11 g) and basil (8 g). It is also worth paying attention to other additives with high-fructose content. They include (1 kg^{-1}): ketchup – 38 g, mustard – 30 g, soy sauce – 10 g.

Fat products, such as edible oils, olive oils, margarine and butter contain no fructose. Mayonnaise and dressings contain less than 1 g of fructose. Eggs, fresh fish and unprocessed meat contain no fructose. Vienna sausages have the highest fructose content as regards meat products (5 g kg^{-1}). Other

Table 3

The content of fructose in selected fresh fruits and vegetables (NIHW 2017)

Fruits	Content of fructose (g kg^{-1})	Vegetables	Content of fructose (g kg^{-1})
Kaki	75	sweet yellow pepper	30
Green or red grapes	74	sweet red pepper	21
Cherry	48	tomato	20
Blueberry	46	white cabbage	17
Black and red currant	44	green raw chilli, onion	15
Pear	42	zucchini, kohlrabi, turnip, pepper, carrot	14
Apple	35	eggplant	13
Guava	32	white radish, red cabbage	12
Kiwi	31	asparagus, chicory	11
Apricot	28	leek	10
Raspberry	22	chinese cabbage, kale, cauliflower, chives	9
Orange	19	cucumber, broccoli, brussels sprout, sprouts	7
Banana	18	pumpkin, radish, garlic	6
Watermelon	13	lettuce	5
Cranberry, plum	12	artichoke	4
Peach, nectarine, mandarin, cantaloupe melon	11	celery, corn, parsley, rhubarb	3
Honey melon	08	spinach, red beetroot	1
Pineapple	07	horseradish	< 1
Lemon	05		
Avocado	< 1		

processed meat products, such as blood sausage, sausage, liver sausage, contain less than 1 g of fructose kg^{-1}.

Alcoholic drinks may deliver as much as 61 g of fructose L^{-1} (liqueurs). As regards this group of products, high fructose content may be found in sweet and dry fruit wines, Sherry, Vermouth, Madeira – 48 g L^{-1} (NIHW 2017).

Fructose is also used as a substance which preserves and protects food from drying (it is highly hygroscopic). It is also used as a sweetener in products for diabetics. Apart from sweetening juices, it is used for sweetening ice cream, yoghurt, various desserts, candied fruit and alcoholic drinks (Sadowska, Rygielska 2014).

SUMMARY

Fructose is a common carbohydrate. Its main source in most unprocessed products is sucrose. It is suggested that sucrose and fructose influence metabolic processes similarly. Therefore, similar disorders are caused by high intake of table sugar and corn syrup with high fructose content. High fructose intake is recognized as the main cause of the development of metabolic diseases such as obesity, non-alcoholic fatty liver disease, type 2 diabetes, kidney disease and cardiovascular disease. Despite strong ties between fructose and disease, the metabolic fate of fructose in mammals remains incompletely understood.

Over the last two centuries, dietary fructose intake per capita has risen many times. There are no detailed population guidelines for the consumption of fructose. It is recommended only to limit simple sugars to 10% of the energy value of the daily diet.

The latest research on the metabolism and hepatotoxicity of fructose is consistent with old recommendations stating that sweets should be consumed in moderation and after meals. In addition, carbonated drinks and juices, once considered to be beneficial for health (because they do not provide fat), may play a significant role in the development of fatty liver. It is also recommended to avoid eating between meals. Sweet taste supresses satiety and reinforces overall food consumption.

There is little information to determine whether the form in which added sugar is consumed, as beverage or as solid food, affects its potential to promote weight gain. Clinical diet studies needed to address those evidence gaps, especially at the levels of added sugar that are commonly consumed, are difficult to conduct. However, filling these evidence gaps may be necessary for supporting changes that will help to turn food environment into one that does not promote the development of obesity and metabolic disease.

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