Fructose: role in the formation of intestinal symptoms

Abstract. Fructose is a simple sugar that is present in fruit and honey, but is also a major component in the two most commonly used sweeteners, sucrose (table sugar), and high fructose corn syrup (HFCS). Intake of fructose has increased markedly over the last several hundred years, and currently the intake of added sugars approaches 15 percent of overall energy intake in the average western diet. The prevalence of fructose malabsorption is relatively high in healthy adults (~34%) and is even greater in patients with functional gastrointestinal disorders. Symptoms following fructose ingestion, or fructose intolerance, are common in patients with irritable bowel syndrome (IBS). Due to the paucity of targeted therapy for IBS, many patients turn to dietary modifications for symptom management. In recent years the low-FODMAPs diet for treatment of IBS has gained increasing popularity. The acronym FODMAP stands for “fermentable oligosaccharides, disaccharides, monosaccharides, and polyol” and includes foods with fructose in excess of glucose, oligosaccharides including fructans, galacto-oligosaccharide and sugar polyols such as sorbitol and mannitol and lactose. The composition of FODMAPs diets and their mechanisms of action in IBS have been intensively studied in the past decade, but since this link is not specific to fructose. Dietary FODMAP might exacerbate intestinal symptoms by increasing small intestinal water volume, colonic gas production, and intestinal motility. Dietary FODMAPs restriction is associated with reduced fermentation and significant symptom improvement in some IBS patients.

Keywords: fructose; malabsorption; FODMAP; irritable bowel syndrome

Introduction

In recent years, the focus of consumers and nutritionists has been on the consumption of sugars, primarily added sugars, including fructose. Many concerns about the detrimental effects of sugars are associated with their current excessive consumption. At the present time sugars provide about 15% of overall energy intake in the average western diet, with higher intakes among children and young adults (adolescents and adults under the age of twenty) and thus significantly exceed the maximum recommended amounts [1, 2]. Over the past three decades the number of sugar sources has also changed. Fructose, as a paradigmatic example of this type of carbohydrate, deserves special attention.

In the field of gastroenterology and hepatology, the attention to fructose is focused on its role in the development of clinical manifestations of irritable bowel syndrome (IBS) and fat accumulation in the liver with the development of non-alcoholic fatty liver disease (in 2020 an international expert group suggested changing the name of the disease “non-alcoholic fatty liver disease” for a more accurate reflection of its pathogenesis — “metabolic dysfunction-associated fatty liver disease” [3].

General characteristics of fructose

Fructose (levulose, fruit sugar) is an isomer of glucose, a monosaccharide, it is classified as a simple sugar. It is one of the most widespread sugars in nature, it can be found both as a monosaccharide and as a part of the disaccharide sucrose (which along with D-fructose includes D-glucose moiety on a 50/50 ratio) and as fructans. Fructans are polymers of fructose (fructose polysaccharides and fructo-oligosaccharides) [4]. Each fructan molecule consists of many β-D-fructose molecules and one α-D-glucose molecule (non-reducing polysaccharides). Fructo-oligosaccharides are short-chain fructans. An example of fructans is inulin.
Fructose is the sweetest of natural sugars: crystalline fructose is 1.8 times sweeter than crystalline sucrose. Nevertheless, fructose has a lower GI (15 or 39 according to different data) compared to sucrose and glucose (65 and 100, respectively) [1].

Fructose is widely used in the food industry as a sweetener. Due to its increased sweetness, it is possible to add less sugar to products, due to which fructose is often used in low-calorie food. Fructose sweetness in solutions depends on temperature, pH and concentration: it increases when the solution is cooled and when it is acidified. Fructose can enhance fruit flavors. It is used in alcohol-free beverages (carbonated, sports, low-calorie, etc.), frozen desserts, bakery food, canned fruit, chocolate, candies and dairy products, jams, marshmallows, candies, cakes; due to good solubility in ethanol it is used in sweet liqueurs [5].

**Sources of fructose**

Fructose is obtained almost exclusively from the diet (it has recently been demonstrated that fructose is also produced endogenously from glucose and sorbitol) [5]. It is naturally found in a variety of food products, such as honey and fruit. In honey fructose is present in the form of a monosaccharide, in table sugar (sucrose) it is connected with glucose, in fruit, berries and vegetables it is found both as a monosaccharide and disaccharide and polysaccharides of fructans. Fructose is most abundant in honey, dates, raisins and prunes [2, 6] (Table 1).

Fructans are found in such food products as agave, artichoke, asparagus, leeks, garlic, onions (including shallots), in grain varieties, as well as in asteraceae (inulin and inulin-type fructans); inulin is obtained from chicory roots (15–20 % of total weight) and artichoke tubers [1, 2, 7].

Natural food is mostly characterized by small amounts of fructose and due to its slow absorption upon fruit or vegetables consumption, its increase in serum after a meal is small.

Honey is defined in the legislation of the European Communities as “the natural sweet substance produced by Apis mellifera bees from the nectar of plants or from secretions of living parts of plants or excretions of plant-sucking insects on the living parts of plants, which the bees collect, transform by combining with specific substances of their own, deposit, dehydrate, store and leave in honeycombs to ripen and mature” [8]. Honey contains a large number of substances, including sugars, mainly fructose and glucose, water and a wide range of other components such as organic acids, proteins, amino acids, minerals, polyphenols, vitamins, aromatic compounds and about 500 enzymes [9].

Fructose and glucose in honey are derived from the chemical conversion of disaccharides in floral nectar by bee-secreted enzymes, where fructose is the highest proportion of sugars (up to 45 %) in almost every honey type. Sugars in honey provide an energy value of 300 kcal/100 g honey, which is equivalent to 15 % of recommended daily intake of energy. The chemical composition of honey varies depending on floral origin, environment and geographical conditions. Sugars dominate honey composition and they are accountable for sensory and physicochemical properties.

### Table 1 — Fructose content in different food products* [2]

| Food products   | Glucose, g/100 g | Fructose, g/100 g |
|-----------------|------------------|-------------------|
| Honey           | 35.2             | 40.2              |
| Dates           | 34.0             | 31.0              |
| Raisins         | 31.2             | 31.6              |
| Sweet cherry    | 13               | 12                |
| Pomegranate     | 7.9              | 8.5               |
| Grapes          | 7.3              | 8.2               |
| Mango           | 5.5              | 8.2               |
| Banana          | 7.4              | 7.2               |
| Pear            | 2.8              | 7.2               |
| Apples          | 3.4              | 6.9               |
| Cola            | 6.0              | 6.0               |
| Figs            | 7.2              | 5.9               |
| Plums           | 3.9              | 5.2               |
| Kiwi fruit      | 5.1              | 5.2               |
| Pineapple       | 4.7              | 5.1               |
| Orange          | 4.3              | 4.4               |
| Honey melon     | 3.9              | 4.2               |
| Peach           | 4.1              | 4.0               |
| Corn            | 3.1              | 2.9               |
| Carrot          | 2.5              | 2.4               |
| Red currant     | 3.5              | 3.8               |
| Pea             | 3.1              | 3.1               |
| Raspberry       | 2.5              | 3.1               |
| Papaya          | 2.9              | 3.0               |
| Paprika         | 2.7              | 3.0               |
| Kohlrabi        | 1.9              | 1.8               |
| Artichoke       | 0.8              | 1.7               |
| Tomatoes        | 1.2              | 1.5               |
| Sunflower seeds | 1.4              | 1.4               |
| Peanuts         | 1.3              | 1.3               |
| Broccoli        | 0.7              | 1.1               |
| Lemon           | 1.2              | 1.1               |
| Lentil          | 0.7              | 1.0               |
| Cauliflower     | 1.0              | 0.9               |
| Chicory         | 1.5              | 0.9               |
| Cucumber        | 0.8              | 0.9               |
| Pumpkin         | 1.1              | 0.8               |
| Cranberry       | 3.3              | 0.7               |
| Lettuce         | 0.5              | 0.6               |
| Oat             | 0.4              | 0.4               |
| Potato          | 0.5              | 0.4               |
| Rice            | 0.1              | 0.1               |
| Pasta           | 0.1              | 0.1               |

*Note. * — The total fructose and glucose content is specified, which always includes free fructose and half the sucrose value; the values in this table are calculated from the average values.
Non-sugar components, although they are present in small quantities, are the major contributors to the health benefits of honey [10].

Worldwide, sucrose is the main source of fructose and it constitutes 90% of the sweeteners used. In addition, with the advent of isomerization, separation and crystallization technologies, it became possible to produce crystalline fructose and high fructose corn syrup (HFCS) [1, 5, 11]. HFCS is a mixture of fructose and glucose in different concentrations: it can contain up to 90% fructose, but the dominant concentrations are 42% or 55% in commercial products. Fructose as a component of sucrose and HFCS as sweeteners play an important role in the food industry.

In the past, fructose intake was between 16 and 20 g/day and was provided by fresh fruit. Modern societies, especially those with a high prevalence of obesity, are characterized by high fructose intake, most of which comes from added sugars [12]. At the present time populations in industrialized countries consume fructose — according to different data — in amounts up to 60–150 g/day — mainly from sucrose (about 7–20% of total energy intake) [13–15]. In some countries (the USA and Japan) HFCS is its important source [16–18]. Common food products providing fructose intake in Europe are alcohol-free fructose-containing and sugar-containing beverages, fruit juices, fruit, cakes and dairy products. Children and adolescents, as a rule, tend to consume the highest amount of fructose per kilogram of body weight [19].

**Absorption of fructose**

Fructose enters the gastrointestinal tract as pure fructose or sucrose. Absorption of fructose does not require digestive enzymes; the disaccharide sucrose must first be split by small intestinal α-glucosidase into a glucose molecule and a fructose molecule.

Fructose is absorbed into enterocytes via glucose transporter 5 (GLUT 5) and enters the portal blood via the glucose transporter (GLUT 2), then, like glucose, is transported to hepatocytes (GLUT 2) [20].

Absorption of fructose in the intestine is incomplete. The fructose absorption capacity at a single intake is limited to an amount in the range of about 5 to 50 g before diarrhea and flatulence occur in some people. When fructose is consumed with glucose, its absorption rate increases [19, 21]. Several factors affect fructose absorption capacity (age and health), but out of the nutritional factors presence of glucose can improve the digestibility of fructose and prevent malabsorption when fructose intake is restricted [19].

Malabsorption of fructose

Fructose absorption in the intestine can be limited by the capacity of the intestinal transporter system. Fructose malabsorption is a digestive disorder in which fructose absorption is impaired due to a deficiency of the fructose transporter in the enterocytes of the small intestine. This leads to an increased fructose concentration in the small intestine with an increased osmotic load. Upon further entry into the large intestine, unabsorbed fructose is rapidly fermented by intestinal bacteria and form short-chain fatty acids (acetate, propionate and butyrate) and gases (hydrogen, methane and carbon dioxide). The presence of unabsorbed fructose in the small intestine and bacterial fermentation products in the large intestine can lead to changes in motility of gastrointestinal tract and intestinal microbiota. Clinically, fructose malabsorption can cause gastrointestinal symptoms such as abdominal pain, flatulence and diarrhea, the significance of these effects depends on the response of intestine to them [19].

The prevalence and severity of fructose malabsorption are directly proportional to fructose levels in the diet and inversely proportional to age [22, 23]. The incidence of fructose malabsorption is relatively high in healthy adults (~34%) and even higher in patients with functional gastrointestinal tract disorders [24, 25].

When fructose is consumed as a single oral dose, the maximum absorption capacity has been shown to vary with great individual variability, from 5 to 50 g [19]. It has been found that 80% of healthy adults have incomplete absorption of fructose at intake of 50 g that is why many people suffer from flatulence and diarrhea. Refusal of fructose consumption and/or fructans allows relief of symptoms in many patients with functional intestine disorders [26].

The diagnosis of fructose malabsorption is similar to that of lactose intolerance using the hydrogen breath test, which is used as a diagnostic test. Nevertheless, some researchers believe that this test is not appropriate for diagnosis because a negative result does not exclude the possibility of improvement when fructose intake is restricted [19].

The simultaneous consumption of fructose together with glucose can improve the digestibility of fructose and prevent the manifestation of malabsorption symptoms. Thus, the peculiarities of the composition of sugars result in different tolerances of fruit. For example, some people may tolerate bananas or grapefruit well because they contain similar amounts of fructose and glucose, while at the same time do not tolerate apples, which have more fructose than glucose. Based on the properties of carbohydrates the FODMAP diet is composed.

| Food products type | Food products |
|--------------------|---------------|
| Grain varieties    | Wheat, barley, rye |
| Bean family        | Lentils, beans, chickpeas, soybeans, peas |
| Vegetables         | Artichokes, asparagus, cauliflower, garlic, leeks, mushrooms, onions, shallots |
| Fruit              | Apples, apricots, Asian pears, blackberries, cherries, figs, mangoes, nectarines, peaches, pears, persimmons, plums, prunes, watermelon, white peaches, grapes |
| Dairy products     | Milk, ice-cream, soft-ripened cheese, yoghurt |

*Table 2 — Food products “high-FODMAP” [30]*
**FODMAP** is an acronym that defines components of food products and stands for “Fermentable Oligosaccharides, Disaccharides, and Monosaccharides and Polyols”. The FODMAPs diet hypothesis was published in 2005 in one of the world’s leading journals, *Alimentary Pharmacology Therapy* (P.R. Gibson and S.J. Shepherd; Monash University, Australia) [27].

FODMAP products contain nutrients, the molecules of which are composed of 1–10 carbohydrates, are osmotically active, are poorly absorbed, remain in the intestinal lumen, and become substrates for bacterial fermentation and production of short-chain fatty acids and gases [28–30]. Such nutrients include fructose (monosaccharide), sucrose and lactose (disaccharides), oligosaccharides, fructans, polyols (mannitol, xylitol, sorbitol, isomalt, etc.). Food products high in FODMAPs have been recognized as trigger agents for intestinal symptoms such as pain and abdominal distention. Fructose is considered to be a FODMAP only when it is in excess compared to glucose, because under these conditions, fructose excess could potentially be the target of fermentation by intestinal bacteria.

After the initial publication of the “FODMAP hypothesis”, intensive research on food components began and considerable effort was made to determine the FODMAP content of products, to develop cut-off levels for defining what “low-FODMAP” is. According to the available data, food products are classified into “low-FODMAP” and “high-FODMAP”. Table 2 shows the “high-FODMAP” food products [30].

Clinical research in this area is focused on the use of low-FODMAP diet in a variety of clinical situations: functional digestive disorders, primarily irritable bowel syndrome (IBS), inflammatory bowel disease, celiac disease and non-celiac gluten hypersensitivity and others, and the identification of potential side effects [31, 32].

Specialists suggest that physicians use an individualized, personalized, “bottom-up” tactic for patients from a low-FODMAP diet to normal consumption [32, 33]. In practice, following a low-FODMAP diet involves three stages [32]:

- **I phase** (first 2–6 weeks) — strict low FODMAP diet;
- **II phase** (next 6–8 weeks) — reintroduction;
- **III phase** (during longer periods of time) — the individualized diet, which implies the consumption of well-tolerated food.

Current trend is the use of health- and nutrition-related apps for smartphones. According to data of L.M. Yamashita et al.; FODMAP project (2021) the Monash University Low FODMAPs diet app is one of the most recommended by nutritionists or self-initiated by patients [34]. However, experts point out: even if software applications are simple and user-friendly, they should be considered only as a useful tool for following a nutritionist’s recommendations, but not as a substitute for a doctor’s consultation.

Although some of the mechanisms of symptom generation by FODMAP components are understandable, further research is required to understand which patients will respond to specific FODMAP subgroup limitations.

Extensive research on the human microbiome has been performed over the past two decades, providing new data on the intestinal microbiota. For the FODMAP diet researchers have focused on features of the intestinal microbiota that influence response to the diet and its predictability. So, a microbiota with fructolytic activity has been proposed as a biomarker of a good response to FODMAP restriction [35].

The authors of the publications note many gaps with regard to the implementation of the FODMAP diet in clinical practice [32, 34, 36]:

- shortage of information on the composition of food products;
- lack of detailed lists of food products low in FODMAP; for example, people with intestinal symptoms are advised to limit intake of fructans to 0.5 g per meal, and products containing inulin and fructooligosaccharides should be avoided, but information on fructans is difficult to find in food products composition databases;
- shortage of FODMAP-free food products, making it difficult to follow a low-FODMAP diet;
- food products labeling defects;
- difficulties in establishing the exact consumption of FODMAP.

Worries about the health effects of long-term use of the FODMAP diet concern:

- restrictions of a number of products and reductions of dietary diversity;
- impact on quality of life and overall human health.

These worries primarily concern children, who have been little researched with regard to FODMAP diet and its components [34].

**Irritable bowel syndrome**

As defined by the Rome Consensus IV (2016), IBS is a functional bowel disorder in which recurrent abdominal pain is associated with defecation or stool changes [26, 37].

A considerable part of adults and children with functional gastrointestinal disorders associate them with food. Fructose malabsorption is symptomatically similar to IBS and when diagnosed in adult patients, the Rome Consensus prescribes exclusion of fructose tolerance disorder.

Due to the limited possibilities in pharmacotherapy of IBS, attention has turned to dietary modifications to correct symptoms. In recent years, attention has been focused on fructose restriction, gluten restriction, and the FODMAP diet. The Rome Consensus IV (2016) warns that excessive consumption of fructose, artificial sweeteners like sorbitol, xylitol, consumption of milk may cause diarrhea, bloating, pain or flatulence with a note on the sharp increase in the consumption of fructose (food supplements, especially in beverages) over the last 30 years. Thus, C. Melchior et al. (2020) noted fructose malabsorption (hydrogen and/or methane tests) in 22% of patients after an intake of 25 g of fructose [38].

Since 2005, numerous studies have been published regarding the effectiveness of a low-FODMAP diet compared with a conventional diet in correcting IBS symptoms in adults. In pediatric patients, the association of fructose malabsorption with unexplained abdominal pain (pain of...
a constant nature that was considered to be caused by a functional bowel disorder) was determined and the role of fructose dose and symptom severity was evaluated. Patients randomly received 1, 15, or 45 g of fructose followed by a hydrogen breath test. The authors concluded that fructose malabsorption may be a significant problem in children, and control over fructose intake may be effective in reducing gastrointestinal symptoms [39]. Another study in children with functional gastrointestinal disorders found fructose intolerance in 35–55 %, and fructose intake reduction yielded significant improvement in 77–82 % [40]. Experts note that a low-fructose diet is integrated into the treatment of IBS, but the mechanisms of its effects are poorly understood and may not be related to fructose malabsorption, and osmotic loading, bacterial fermentation, microbiota disruption, and hypersensitivity are discussed as possible [38, 41].

In practice, it is not always easy for patients, especially children, to identify intolerances to certain food products. So, a cross-sectional study examined the relation between self-defined food intolerance and severity of IBS symptoms (154 children meeting the Rome Consensus III criteria for pediatric IBS and 32 healthy controls aged 7–18 years) [42]. The authors concluded that children with IBS compared with healthy controls identified at least one food product and avoided more food products, but that these self-reported intolerances were poorly associated with the frequency and severity of abdominal pain, somatization, anxiety, and decreased quality of life.

Again referring to the Rome Consensus IV (2016), it is worth emphasizing that its authors state: special diets with gluten restriction, “low-FODMAP” in patients who have not previously used dietary manipulation, compared with the traditional diet, led to improvement. On the other hand, the effectiveness of specific recommendations (including the gluten-free diet, “low-FODMAP”) and standard dietary recommendations are similar. A diet limiting FODMAPs may be an effective dietary intervention for reducing IBS symptoms in adults. In children, there are promising data, although there is only one randomized, double-blind study that demonstrated the efficacy of a “low-FOODMAP” diet in children with IBS for all types of IBS.

At the same time, researchers express concern that a low-FOODMAP diet, particularly a fructose-restricted diet, with encouraging evidence of therapeutic effectiveness in control over abdominal symptoms in most adults and children diagnosed with IBS, contains severe restriction of large amounts of food products, which may reduce calcium and dietary fiber intake, particularly for children. Additional efforts are required to better clarify the role of the FODMAP diet and restricted fructose in IBS, to suggest the most effective way of dietary restriction based on patient tolerance and/or to identify potential biomarkers for selecting patients for the use of low-FODMAP diets, provided adequate nutrition is maintained and dietary compliance style is facilitated by FODMAP labeling [15, 30].

Conclusions

A number of data have been obtained on the effects of fructose, especially in high doses, relevant to the development of gastrointestinal symptoms, which, at known high levels of its consumption, requires educational measures to regulate/reduce the consumption of added sugars among the population, especially children and adolescents. Further study of the problem will provide a better understanding of how overconsumption of fructose can cause diseases and how it can be reduced, identification of fructose doses that affect health, as well as populations that are particularly responsive to or protected from the adverse health effects associated with high fructose intake.

References

1. Jensen T, Abdelmalek MF, Sullivan S, et al. Fructose and sugar: A major mediator of non-alcoholic fatty liver disease. J Hepatol. 2018 May;68(5):1063-1075. doi: 10.1016/j.jhep.2018.01.019.
2. Roeb E, Weiskirchen R. Fructose and Non-Alcoholic Steatohepatitis. Front Pharmacol. 2021 Feb 8;12:634344. doi: 10.3389/fphar.2021.634344.
3. Estlam M, Newsome PN, Sarin SK, et al. A new definition for metabolic dysfunction-associated fatty liver disease: An international expert consensus statement. J Hepatol. 2020 Jul;73(1):202-209. doi: 10.1016/j.jhep.2020.03.039.
4. Miranda J, Vázquez-Polo M, Pérez-Junquera G, et al. FODMAP Intake in Spanish Population: Open Approach for Risk Assessment. Int J Environ Res Public Health. 2020 Aug 13;17(16):5882. doi: 10.3390/ijerph17165882.
5. Dornas WC, de Lima WG, Pedrosa ML, Silva ME. Health implications of high-fructose intake and current research. Adv Nutr. 2015 Nov 13;6(6):729-37. doi: 10.3945/an.114.008144.
6. Kolderup A, Sivisah B. Fructose Metabolism and Relation to Atherosclerosis, Type 2 Diabetes, and Obesity. J Nutr Metab. 2015;2015:823081. doi: 10.1155/2015/823081.
7. Peredeyri VG, Sizenko AK, Tkach SM. Modern approaches to the diagnosis and correction of fructose malabsorption. Modern gastroenterology. (2010);53:113-120.
8. Council Directive 2001/110/EC of 20 December 2001 relating to honey. Off J Eur Commun. 2002(10):47-52. Available from: https://eur-lex.europa.eu/legal-content/EN/TXT/?uri=celex:32001L0110.
9. Bogdanov S, Jurendic T, Sieber R, Gallmann P. Honey for nutrition and health: a review. J Am Coll Nutr. 2008 Dec;27(6):677-89. doi: 10.1080/07315724.2008.1071947.
10. Nguyen HTL, Panyoyai N, Kasapis S, Pung E, Mantri N. Honey and Its Role in Relieving Multiple Facets of Atherosclerosis. Nutrients. 2019 Jan 14;11(1):167. doi: 10.3390/nu11010167.
11. Hengist A, Koanaman F, Gonzalez JT. Fructose and metabolic health: governed by hepatic glycogen status? J Physiol. 2019 Jul;597(14):3573-3585. doi: 10.1113/JP277767.
12. Hills SP, Mitchell P, Wells C, Russell M. Honey Supplementation and Exercise: A Systematic Review. Nutrients. 2019 Jul 12;11(7):1586. doi: 10.3390/nu11071586.
13. Caravaggio F, Borlido C, Hahn M, et al. Reduced insulin sensitivity is related to less endogenous dopamine at D2/D3 receptors in the ventral striatum of healthy nonobese humans. Int J Neuropsychopharmacol. 2015 Feb 25;18(7):pyv014. doi: 10.1093/ijnp/pyv014.
14. Federico A, Rosato V, Masarone M, et al. The Role of Fructose in Non-Alcoholic Steatohepatitis: Old Relationship and New Insights. Nutrients. 2021 Apr 16;13(4):1314. doi: 10.3390/nu13041314.
15. Persabene L, Salvatore S, Turco R, et al. Low FODMAP diet for functional abdominal pain disorders in children: critical review of current knowledge. J Pediatr (Rio J). 2019 Nov-Dec;95(6):642-656.
Фруктоза: роль у формуванні кишкових симптомів

Резюме. Фруктоза — це простий цукор, присутній у фруктах та меду, також вона є основним компонентом двох найбільш часто використовуваних підслоджувачів — сахарози (столового цукру) та кукурудзяного сиропу з високим умістом фруктози. Споживання фруктози помітно зросло за останні кілька сотень років, і в даний час споживання додаткового цукру наближається до 15 відсотків загального споживання енергії в середньостатистичному європейському раціоні. Поширеність мальабсорбції фруктози відносно висока у здорових дорослих (~ 34 %), і цей показник більше у пацієнтів з функціональними розладами шлунково-кишкового тракту. Симптоми, що розвиваються після прийому фруктози у разі непереносимості фруктози, є поширенними у пацієнтів із синдромом подразнення кишечника (СПК). Через недо статність таргетної терапії СПК багато пацієнтів для лікування симптомів вдаються до модифікації дієти. В останні роки дієта з низьким вмістом FODMAP для лікування СПК набуває все більшої популярності. Абревіатура FODMAP означає «ферментовані олігосахариди, дисахариди, моносахариди та поліол» і включає продукти з умістом фруктози більше, ніж глюкози, олігосахариди, включаючи фруктанди, галактозоолігосахариди та цукрові поліоли, такі як сорбітол та бета-кетоглюкоза. Склад дієти FODMAP та її механізми дії при СПК були інтенсивно вивчені у останнє десятиліття, але їх зв’язок з фруктозою не є визначеним. Оскільки FODMAP може посилювати кишкові симптоми через збільшення об’єму води в тонкому кишечнику, утворення газів у товстому кишечнику і ферментации, дієтичні обмеження FODMAP пов’язані зі зниженням ферментации та значним покращенням симптомів у деяких пацієнтів із СПК.

Ключові слова: фруктоза; порушення всмоктування; FODMAP; синдром подразнення кишечника.