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CLINICAL REVIEWS

Dietary Fructose and Gastrointestinal Symptoms: A Review

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It has been proposed that fructose may cause or aggravate symptoms in patients with functional gastrointestinal disorders. Fructose is commonly used to sweeten processed foods, and the prevalence of incomplete fructose absorption (25 g, 10%) in healthy subjects is as high as 50%. The only controlled study that has been performed did not demonstrate a higher prevalence of fructose-induced gastrointestinal symptoms or incomplete fructose absorption in patients with functional gastrointestinal disorders. The amount and concentration of fructose used to evaluate absorption by breath testing has varied among studies. Moreover, dietary sources of fructose usually contain glucose, which increases fructose absorption in healthy subjects. Thus, breath testing with fructose alone may not reflect fructose ingestion under normal circumstances. Given these limitations, we suggest that a practical, empirical approach to testing in patients with suspected incomplete fructose absorption is to restrict fructose ingestion. Additional controlled studies are needed to clarify the relation between incomplete fructose absorption and symptoms, assess the effects of coingestion of other sugars on fructose absorption, and evaluate the effects of eliminating sugars from the diet on gastrointestinal symptoms.

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INTRODUCTION

Fructose, a naturally occurring sugar, has increasingly been used as a sweetener since the introduction of high-fructose corn syrups in the 1960s. Fructose is incompletely absorbed even in healthy subjects. Recent studies suggest that fructose intolerance may partly explain gastrointestinal symptoms in patients with functional bowel disorders. This article reviews mechanisms for, and factors affecting fructose absorption, evaluates the evidence implicating fructose intolerance as a cause for symptoms in functional bowel disorders, and outlines an approach for evaluating fructose intolerance in clinical practice.

DIETARY SUGARS

Fructose, glucose, and galactose are the three major dietary monosaccharides. Sucrose (glucose–fructose), lactose (glucose–galactose), and maltose (glucose–glucose) are the major disaccharides. Dietary fructose, therefore, occurs in two forms: monosaccharide or disaccharide. Fructose monosaccharide is the sweetest of all natural sugars (1). Honey and fruits (Table 1) were the main dietary sources of fructose monosaccharide prior to the introduction of high-fructose corn syrups in the 1960s. These syrups were developed to provide a cheaper alternative to sucrose (table sugar) in food processing. Since their introduction, the use of high-fructose corn syrups has become widespread, dramatically increasing the amount of fructose monosaccharide in our food supply (2). High-fructose corn syrups are now found in beverages, dairy products, and canned, baked, or processed foods worldwide (3).

PRINCIPLES OF SUGAR ABSORPTION

Only monosaccharides are absorbed across the intestinal epithelium. Therefore, disaccharides must be enzymatically cleaved into their monosaccharide components to be absorbed. Glucose and galactose share a transporter and are efficiently absorbed across the intestinal epithelium against a concentration gradient by an energy-dependent process referred to as active transport. Mannose, another monosaccharide, crosses the epithelium slowly, moving from high to low concentration by passive diffusion. The rate of fructose absorption is between that of mannose and glucose (4). Fructose monosaccharide is absorbed by carrier-mediated facilitated diffusion, an energy-independent process. The fructose carrier is a member of the glucose transport (GLUT) family of genes encoding for facilitative sugar transporters and is referred to as GLUT 5. GLUT 5 is a high-affinity fructose transporter found on the luminal surface of small intestine epithelial cells (5).

Sucrose is cleaved to glucose and fructose by sucrase, an enzyme located in the brush border of small intestine enterocytes. For unclear reasons, the absorptive capacity for fructose derived from sucrose exceeds that of fructose monosaccharide in healthy individuals (6, 7).

Table 1. Carbohydrate Composition of Common Foods

Per 100 g Edible Portion	Fructose (g)	Glucose (g)	Sorbitol (g)
Apples	Up to 6.0	1.7	Up to 1.0
Pears	Up to 8.9	2.5	Up to 4.5
Bananas	Up to 3.8	4.5	0
Sweet cherry	Up to 7.2	4.7	Up to 12.6
Strawberries	Up to 2.5	2.6	0
Grapes	Up to 10.5	Up to 8.2	0
Plums	Up to 4.0	Up to 5.5	Up to 2.8
Prunes	Up to 23	Up to 30	Up to 15
Dates	Up to 31	Up to 24.9	
Peaches	Up to 1.5	1.5	Up to 1.3
Apple juice	6–8	1–4	0.3–1.0
Pear juice	5–9	1–2	1.1–2.6
Orange juice	Up to 5.3	2.4	0
Honey (100 g or 3 tblsp)	35	29	0
Sugar-free gum and mints	–	–	1.3–2.2 per piece

Compiled from References (16, 25, 37–39).

TESTING FOR INCOMPLETE FRUCTOSE ABSORPTION

Similar to other carbohydrates, incomplete fructose absorption can be identified noninvasively by measuring breath hydrogen following a fructose load. Colonic bacterial fermentation of carbohydrates, which are not absorbed in the small bowel, results in the production of short-chain fatty acids, hydrogen, carbon dioxide, and trace gases. Hydrogen is not metabolized by humans and must be excreted in breath and flatus and/or consumed by colonic bacteria, producing methane or sulfide.

In general, a rise in breath hydrogen of greater than 20 parts per million (ppm) peaking 2–3 h following substrate ingestion reflects incomplete absorption (8). An earlier peak in breath hydrogen (*i.e.*, within 1 h of substrate ingestion) may reflect rapid small bowel transit, small bowel bacterial overgrowth, or substrate fermentation by oral flora. An antibacterial mouth rinse prior to testing can eliminate the confounding effects of oral flora. Increased fasting breath hydrogen levels may represent small bowel bacterial overgrowth or colonic fermentation of carbohydrates contained in a previous meal (8). There are two potential pitfalls to assessing incomplete fructose absorption by breath hydrogen testing. First, a breath hydrogen response requires that hydrogen-producing bacteria are present and they outnumber hydrogen-consuming bacteria. Individuals who cannot generate a breath hydrogen response to lactulose, a nonabsorbable synthetic disaccharide, probably lack the flora necessary to generate a breath hydrogen response to any saccharide. In these individuals, breath testing for methane may identify carbohydrate malabsorption (9). Second, the appropriate dose and concentration of fructose for breath testing is disputed (10). Incomplete absorption after a 50 g fructose load was observed in 37.5% (10% solution) and 71% (20% solution) of healthy people (7). A 10% fructose concentration approximates more closely than a 20% concentration to the fructose concentration of soft drinks (*e.g.*, (12%). A lower fructose load may be more specific for recognizing genuine fructose intolerance, since subjects with the lowest absorptive capacity are more likely to overwhelm absorptive capacity by dietary fructose content.

FRUCTOSE ABSORPTIVE CAPACITY IN HEALTHY SUBJECTS

In healthy individuals, fructose absorptive capacity has been estimated by determining breath hydrogen response to varying doses and concentrations of fructose in the fasting state. The absorptive capacity for fructose ranged from less than 5 g to greater than 50 g (6), was unrelated to age or sex (11, 12), and was dose and concentration dependent (6, 7, 12, 13). When the fructose dose was increased from 25 to 50 g (10% solution), the prevalence of incomplete absorption increased, *e.g.*, from 0 to 37.5%, 11 to 58%, and 50 to 80%, respectively, in three different studies (6, 7, 12). Similarly, when the fructose concentration was doubled from 10 to 20%, the prevalence of incomplete absorption of 50 g fructose increased from 37.5 to 71.4% (7). These data suggest that for a 10% solution, the threshold for fructose absorption in most healthy individuals lies between 25 and 50 g.

Gastrointestinal symptoms after fructose ingestion may not correlate with the degree of breath hydrogen response. Gastrointestinal symptoms (*e.g.*, flatulence, abdominal discomfort, and/or diarrhea) were reported by 46% of healthy subjects who incompletely absorbed 25 g fructose (14) and 50–83% of those who incompletely absorbed 50 g (7, 12). Another study found mild or no symptoms associated with incomplete absorption of 50 g fructose (6). Thus, relying on breath hydrogen response alone to diagnose fructose intolerance will incorrectly label some "tolerant" incomplete absorbers as fructose intolerant and miss the diagnosis in others.

FRUCTOSE ABSORPTIVE CAPACITY IN FUNCTIONAL BOWEL DISORDERS

In uncontrolled studies, the prevalence of incomplete fructose absorption (25 g) was higher in patients with functional bowel disorders, *i.e.*, 36–75% (13, 15–17) compared to the prevalence (*i.e.*, 0–50%) reported in healthy subjects (6, 7, 12). In the only controlled study, the frequency of incomplete fructose absorption and gastrointestinal symptoms in patients was not significantly different from controls (16). Another study demonstrated that IBS patients with incomplete absorption had significantly higher total symptom scores compared to IBS patients who absorbed fructose (15).

SYMPTOMATIC INCOMPLETE ABSORPTION

The mechanism by which incompletely absorbed fructose causes gastrointestinal symptoms is not fully understood.

Conceivably, the osmotic effects of fructose may cause symptoms by intestinal distention. While IBS patients have an exaggerated perception of bowel distention, symptoms after fructose–sorbitol ingestion were not associated with increased sensitivity to jejunal balloon distension (18). Alternatively, symptoms may be caused by colonic distention resulting from colonic bacterial fermentation of incompletely absorbed sugars. The relationship of symptoms after fructose ingestion to colonic sensitivity is unknown. The colonic anaerobic bacterial flora may also contribute to symptoms after fructose ingestion (14) by fermenting incompletely absorbed fructose to short-chain fatty acids, which stimulate colonic motility (19).

DIETARY FACTORS THAT FACILITATE FRUCTOSE ABSORPTION

In previous studies, glucose, galactose (6, 11, 12, 20), and certain amino acids (21) increased fructose absorption. The extent to which glucose increases fructose absorption depends on the proportion of glucose relative to fructose (6). An equimolar dose of glucose prevented incomplete fructose absorption in healthy subjects (6, 11) and glucose at one-half the fructose dose decreased the prevalence of incomplete absorption by over 50% (6). Glucose and certain amino acids (*e.g.*, alanine, proline, and glutamine) (21) facilitated fructose absorption, presumably by solvent drag and passive diffusion (20–23). In addition, it is conceivable that glucose delayed gastric emptying, thereby facilitating fructose absorption (24).

SORBITOL IMPEDES FRUCTOSE ABSORPTION

Sorbitol is one of several sugar alcohols found naturally (Table 1) and used as "sugar-free" sweeteners by food industry, primarily because they do not promote tooth decay or cause hyperglycemia. For these reasons, sugar alcohols are used in "sugarless" gums and candies, and in ketogenic and diabetic foods. Sorbitol is incompletely absorbed (25). Moreover, doses of sorbitol and fructose that are absorbed when ingested separately may be incompletely absorbed when taken together (26). Incomplete absorption of fructose and sorbitol may cause gastrointestinal symptoms in IBS. Thus, 40–75% of healthy subjects (27, 28) incompletely absorbed a mixture of fructose (25 g) and sorbitol (5 g); most were asymptomatic (27) and breath hydrogen levels did not correlate with symptoms (27, 28). By comparison, 30–100% of IBS patients (15–18, 27, 28) incompletely absorbed this mixture. Moreover, in contrast to fructose alone (16), the prevalence of gastrointestinal symptoms after fructose–sorbitol ingestion was higher in IBS patients compared to healthy subjects (16, 27, 28). This interaction between fructose and sorbitol absorption is critical to understanding the gastrointestinal effects of sweeteners since it is likely that these sweeteners will be consumed together as fruits/juices (Table 1) or as a result of combining processed foods. The interaction between fructose and sorbitol absorption may reflect competition for the same receptor (26) or acceleration of transit by a synergistic osmotic effect (29). The effects of glucose on absorption of a fructose–sorbitol mixture are unknown. Since glucose enhanced the absorption of sorbitol (30–32) and fructose administered separately (6, 11, 20), it is conceivable that glucose will also facilitate absorption of a fructose–sorbitol mixture.

EFFECT OF FRUCTOSE AND FRUCTOSE–SORBITOL RESTRICTED DIETS IN IBS

Functional gastrointestinal symptoms resolved after dietary fructose restriction in an unblinded, uncontrolled report of four patients (33). Forty percent (16) to 56% (34) of IBS patients reported substantial improvement in symptoms after restriction of one or more of the following (*i.e.*, lactose, fructose, sorbitol) incompletely absorbed sugar(s). Patient compliance with fructose-restricted diets has been variable (35, 36). Thus, only 54% of subjects instructed in a fructose-restricted diet complied with dietary fructose restriction for more than 50% of the time from 6 to 18 months after beginning the diet (36). Patient compliance is influenced, among other factors, by the widespread distribution of fructose in food products. Gastrointestinal symptoms improved significantly in patients who complied, but not in patients who did not comply with a fructose-restricted diet.

DIETARY IMPLICATIONS

These studies can be used to guide dietary recommendations for patients who may be fructose intolerant. Assuming that an equimolar amount of glucose prevents incomplete fructose absorption in functional bowel disease as it does in healthy subjects, then only sources containing more fructose than glucose, (*i.e.*, excess fructose) need to be restricted. Fruits and juices that provide glucose in equal (or greater) amounts as fructose (*i.e.*, bananas, strawberries) may be well tolerated. Fruits and juices containing excess fructose (*i.e.*, honey, dates, oranges) or excess fructose plus sorbitol (*i.e.*, cherries, apples, pears) may be poorly tolerated, particularly if ingested in excess and/or without other food or drink.

The main source of fructose in processed foods is high-fructose corn syrups (HFCS). These are mixtures containing either approximately 42 or 55% fructose as defined in Title 21, Chapter I, Part 184, Sec. 184.1866 in the Code of Federal Regulations. In contrast to HFCS, corn syrup mainly contains glucose derivatives (3). Although the name suggests otherwise, glucose is the predominant sugar present in HFCS-42 (53% glucose, 42% fructose, 5% oligosaccharide) (1). HFCS-55 (42% glucose, 55% fructose, 3% oligosaccharide) contains a small excess of fructose (1). Foods sweetened with HFCS contain one of these two fractions. While fractions approaching pure fructose are available, their use is limited to

specialty items such as nutritional bars and "lite" or reduced calorie snacks and beverages.

Fructose absorption after ingestion of fructose alone has not been compared to absorption of fructose contained in HFCS. HFCS-42 contains glucose in excess of fructose and should be well absorbed. The small amount of excess fructose in HFCS-55 may be incompletely absorbed, particularly in individuals with a low absorptive capacity for fructose. Since ingredient labels do not specify which fraction they contain, a conservative approach is to treat all HFCS products as though they contain HFCS-55. A 12-ounce cola sweetened with HFCS-55 contains about 40 g of the sweetener, *i.e.*, 22 grams of fructose and 17 grams of glucose, representing a fructose excess of 5 g per can. Only 1 of 10 healthy individuals incompletely absorbed 10 g (possibly even 5 g) of fructose and had no symptoms (6).

Unless the absorptive capacity for fructose in functional bowel disease differs from healthy subjects, most people will absorb the excess fructose (*i.e.*, fructose in excess of glucose) present in fruit, juice, or processed foods when ingested in recommended serving sizes. One approach to the patient who may be fructose intolerant is empiric dietary fructose restriction. Products listing "fructose" or "crystalline fructose" (not "high-fructose corn syrup") as an ingredient should be eliminated. Juices/fruits containing excess fructose and products listing "high-fructose corn syrup" as an ingredient should be consumed in their recommended serving size (nondiet fountain drinks limited to 12 ounces), with a meal, and without sugar alcohols (unless naturally occurring) or lactose (if lactose intolerant). Perhaps, it is reasonable to initially limit ingestion to a maximum of two servings of all foods and beverages containing excess fructose per meal. If symptoms persist despite these restrictions and fructose intolerance is still suspected, a trial eliminating all HFCS items, as well as natural sources of excess fructose would be appropriate.

CLINICAL IMPLICATIONS

Up to 80% of healthy subjects incompletely absorb 50 g of fructose. For beverages, total sugar concentrations approximate 10%. Therefore, breath hydrogen testing conducted with 10% solutions of a smaller fructose dose (*e.g.*, 25 g) may be more specific for detecting fructose intolerance. There are two options for dealing with clinically suspected fructose intolerance, *i.e.*, breath testing or empirical restriction of dietary fructose. In theory, breath testing is useful for documenting intolerance of a specific dose and concentration of fructose in the fasting state, but does not predict whether the individual will tolerate a lower dose or whether the presence of other nutrients will improve tolerance. Moreover, since incomplete fructose absorption is relatively common, incomplete absorption does not necessarily imply causation of symptoms. Lastly, a negative hydrogen breath test does not exclude the possibility of incomplete absorption. A false negative result may occur when there are too few hydrogen-producing bacteria, too many hydrogen-consuming bacteria, or inadequate bacterial sugar fermentation. These limitations of hydrogen breath testing also apply to other carbohydrates such as lactose.

An alternative approach for evaluating patients with suspected fructose intolerance is to empirically restrict dietary fructose without a breath test. Assuming subjects comply with dietary recommendations, lack of improvement suggests that symptoms cannot be attributed to incomplete fructose absorption.

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REFERENCES

1. Hanover LM, White JS. Manufacturing, composition, and applications of fructose. Am J Clin Nutr 1993; 58:724S–32S.
2. Park YK, Yetley EA. Intakes and food sources of fructose in the United States. Am J Clin Nutr 1993; 58:737S–47S.
3. Vuilleumier S. Worldwide production of high-fructose syrup and crystalline fructose. Am J Clin Nutr 1993; 58:733S–6S.
4. Holdsworth CD, Dawson AM. Absorption of fructose in man. Proc Soc Exp Biol Med 1965; 118:142–5.
5. Wasserman D, Hoekstra JH, Tolia V, et al Molecular analysis of the fructose transporter gene (GLUT5) in isolated fructose malabsorption. J Clin Invest 1996; 98:2398–402.
6. Rumessen JJ, Gudmand-Hoyer E. Absorption capacity of fructose in healthy adults. Comparison with sucrose and its constituent monosaccharides. Gut 1986; 27:1161–8.
7. Ravich WJ, Bayless TM, Thomas M. Fructose: Incomplete intestinal absorption in humans. Gastroenterology 1983; 84:26–9.
8. Romagnuolo J, Schiller D, Bailey RJ. Using breath tests wisely in a gastroenterology practice: An evidence-based review of indications and pitfalls in interpretation. Am J Gastroenterol 2002; 97:1113–26.
9. Strocchi A, Levitt MD. Factors affecting hydrogen production and consumption by human fecal flora. The critical roles of hydrogen tension and methanogenesis. J Clin Invest 1992; 89:1304–11.
10. Doma S, Gaddipati K, Fernandez A, et al Results of the fructose breath test in healthy controls using different doses of fructose: Which dose is best? Am J Gastroenterol 2003; 98:S265. (Abstract.)
11. Kneepkens CM, Vonk RJ, Fernandes J. Incomplete intestinal absorption of fructose. Arch Dis Child 1984; 59:735–8.
12. Truswell AS, Seach JM, Thorburn AW. Incomplete absorption of pure fructose in healthy subjects and the facilitating effect of glucose. Am J Clin Nutr 1988; 48:1424–30.
13. Choi YK, Johlin FC, Summers RW, et al Fructose intolerance: An under-recognized problem. Am J Gastroenterol 2003; 98:1348–53.

14. Born P, Zech J, Lehn H, et al Colonic bacterial activity determines the symptoms in people with fructose-malabsorption. Hepatogastroenterology 1995; 42:778–85.
15. Rumessen JJ, Gudmand-Hoyer E. Functional bowel disease: Malabsorption and abdominal distress after ingestion of fructose, sorbitol, and fructose-sorbitol mixtures. Gastroenterology 1988; 95:694–700.
16. Fernandez-Banares F, Esteve-Pardo M, de Leon R, et al Sugar malabsorption in functional bowel disease: Clinical implications. Am J Gastroenterol 1993; 88:2044–50.
17. Fernandez-Banares F, Esteve-Pardo M, Humbert P, et al Role of fructose-sorbitol malabsorption in the irritable bowel syndrome. Gastroenterology 1991; 101:1453–4. (Comment)
18. Evans PR, Piesse C, Bak YT, et al Fructose-sorbitol malabsorption and symptom provocation in irritable bowel syndrome: Relationship to enteric hypersensitivity and dysmotility. Scand J Gastroenterol 1998; 33:1158–63.
19. Cherbut C. Effects of short-chain fatty acids on gastrointestinal motility. In: Cummings JH, Rombeau J, Sadata T, eds. Physiological and clinical aspects of short chain fatty acids. Cambridge, UK: Cambridge University Press, 1995:191–207.
20. Holdsworth CD, Dawson Am. The absorption of monosaccharides in man. Clin Sci 1964; 27:371–9.
21. Hoekstra JH, van den Aker JH. Facilitating effect of amino acids on fructose and sorbitol absorption in children. J Pediatr Gastroenterol Nutr 1996; 23:118–24.
22. Shi X, Schedl HP, Summers RM, et al Fructose transport mechanisms in humans. Gastroenterology 1997; 113:1171–9.
23. Fine KD, Santa Ana CA, Porter JL, et al Mechanism by which glucose stimulates the passive absorption of small solutes by the human jejunum in vivo. Gastroenterology 1994; 107:389–95.
24. Elias E, Gibson GJ, Greenwood LF, et al The slowing of gastric emptying by monosaccharides and disaccharides in test meals. J Physiol 1968; 194:317–26.
25. Hyams JS. Sorbitol intolerance: An unappreciated cause of functional gastrointestinal complaints. Gastroenterology 1983; 84:30–3.
26. Rumessen JJ, Gudmand-Hoyer E. Malabsorption of fructose-sorbitol mixtures. Interactions causing abdominal distress. Scand J Gastroenterol 1987; 22:431–6.
27. Nelis GF, Vermeeren MA, Jansen W. Role of fructose-sorbitol malabsorption in the irritable bowel syndrome. Gastroenterology 1990; 99:1016–20. (Comment.)
28. Symons P, Jones MP, Kellow JE. Symptom provocation in irritable bowel syndrome. Effects of differing doses of fructose-sorbitol. Scand J Gastroenterol 1992; 27:940–4.
29. Summers RW, Johlin FC. Fructose intolerance is due to rapid orocecal transit and not small bowel bacterial overgrowth. Gastroenterology 2001; 120:A1369. (Abstract.)
30. Beaugerie L, Flourie B, Lemann M, et al Sorbitol absorption in the healthy human small intestine is increased by the concomitant ingestion of glucose or lipids. Eur J Gastroenterol Hepatol 1995; 7:125–8.
31. Beaugerie L, Lemann M, Jian R, et al Effect of glucose and lipids on intestinal absorption of sorbitol: role of gastric emptying. Neurogastroenterol Motil 1996; 8:235–9.
32. Beaugerie L, Flourie B, Pernet P, et al Glucose does not facilitate the absorption of sorbitol perfused in situ in the human small intestine. J Nutr 1997; 127:341–4.
33. Andersson DE, Nygren A. Four cases of long-standing diarrhoea and colic pains cured by fructose-free diet: A pathogenetic discussion. Acta Med Scand 1978; 203:87–92.
34. Goldstein R, Braverman D, Stankiewicz H. Carbohydrate malabsorption and the effect of dietary restriction on symptoms of irritable bowel syndrome and functional bowel complaints. Isr Med Assoc J 2000; 2:583–7.
35. Johlin FC, Panther M, Kraft N. A fructose restricted diet and dietary counseling in patients with dietary fructose intolerance demonstrates significant reduction in symptoms and an improvement in quality of life proportionate to the amount of fructose eliminated. Gastroenterology 2001; 120:A1374. (Abstract.)
36. Choi YK, Kraft N, Summers R, et al How useful is fructose restricted diet in patients with IBS and fructose intolerance? Am J Gastroenterol 2003; 98:S266. (Abstract.)
37. Hardinge MG, Swarner JB, Crooks H. Carbohydrates in Foods. J Am Diet Assoc 1964; 46:197–204.
38. Ladas SD, Haritos DN, Raptis SA. Honey may have a laxative effect on normal subjects because of incomplete fructose absorption. Am J Clin Nutr 1995; 62:1212–5.
39. Rumessen JJ. Fructose and related food carbohydrates. Sources, intake, absorption, and clinical implications. Scand J Gastroenterol 1992; 27:819–28.