

# Food intake and symptoms in FGID: Short-chain carbohydrates

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## **Abstract**

Carbohydrates occur across a range of foods regularly consumed including grains such as wheat and rye, vegetables, fruits and legumes. Short-chain carbohydrates with chains of up to ten sugars vary in their digestibility and subsequent absorption. Those that are poorly absorbed exert osmotic effects in the intestinal lumen increasing its water volume, and are rapidly fermented by bacteria with consequent gas production. These two effects alone may underlie most of the induction of gastrointestinal symptoms after they are ingested in moderate amounts via luminal distension in patients with visceral hypersensitivity. This has been the basis of the use of lactose-free diets in those with lactose malabsorption and of fructose-reduced diets for fructose malabsorption. However, application of such dietary approaches in patients with functional bowel disorders has been restricted to observational studies with uncertain efficacy. Since all dietary poorly absorbed short-chain carbohydrates have similar and additive effects in the intestine, a concept has been developed to regard them collectively as FODMAPs (Fermentable Oligosaccharides, Disaccharides, Monosaccharides And Polyols) and to evaluate a dietary approach that restricts them all. In patients with irritable bowel syndrome, there is now an accumulating body of evidence, based on observational and comparative studies, and on randomised controlled trials that supports the notion that FODMAPs trigger gastrointestinal symptoms in patients with functional bowel disorders and that a diet low in FODMAPs offers considerable symptom relief in the majority of patients who use it.

### **The spectrum of carbohydrates in the diet**

Carbohydrates are classified according to the degree of polymerization (the number of component molecules) and the characteristics of the majority of carbohydrates found in the diet are described in Table 1. The type of bond that exists between the component molecules is either  $\alpha$  or  $\beta$ , and these bonds are important as they affect structure and/or digestibility (1). As discussed below, digestibility and subsequent small intestinal absorption of monosaccharides are important, since nearly all short-chain carbohydrates are readily fermented by intestinal bacteria and some have prebiotic effects, which has led to their putative nutraceutical use as food additives. Polysaccharides are discussed in another technical review (2).

### **Altering carbohydrate intake as a treatment for functional gastrointestinal disorders**

Carbohydrates have been the major target of dietary modification for functional gut symptoms. Historically and anecdotally, carbohydrate-rich foods have been associated with worsening of symptoms in patients with functional gastrointestinal disorders (FGID), ranging from the specific induction of abdominal symptoms following sugar challenges (lactose or fructose), sorbitol, and oligosaccharides (fructans) alone or in combination (3-17) to observation with whole foods and diets (6,18). There has been a profusion of diets published in the lay press, in medical literature or on the internet that principally target carbohydrate intake in its amount and or type. Some key characteristics for the best known diets as published are described in Table 2. It should be noted that only some of these dietary approaches were specifically targeted to functional gut symptoms in their initial descriptions, though most have been applied in patients with FGID by medical and alternative health practitioners, and by the individuals themselves as their descriptions are readily available on the internet and in other published sources. The rationale for some of the diets, such as the specific carbohydrate, paleolithic and anti-candida diets is largely non-evidence based, predominantly relying on anecdotal experience. The major issues with these diets are that they markedly interfere with food choice, altering not only carbohydrate intake, and potentially can impair nutritional

adequacy, although this aspect has not been specifically studied (19). However, they appear to be poor long-term options for a chronic condition such as FGID and are not widely supported in standard clinical practice.

A more pathogenically-oriented approach has been to target specific short-chain carbohydrates, alone or in various combinations. Thus, lactose alone, fructose (alone or in combination with sorbitol), and combinations of multiple, a diet low in Fermentable Oligosaccharides, Disaccharides, Monosaccharides And Polyols (FODMAPs) have been restricted in patients with IBS, as discussed in detail below. The basis of such dietary approaches is the observation that, in a proportion of patients, ingestion of these short-chain carbohydrates induces IBS-like symptoms and their dietary restriction can improve IBS symptoms. One physiological feature common to these molecules is their potential to be poorly absorbed in the small intestine.

#### **Small intestinal absorption of short-chain carbohydrates**

Only monosaccharides (with the chemical structure of a hexose) can be absorbed across the small intestinal epithelium. Thus, disaccharides and oligosaccharides must be hydrolysed to their constituent hexoses for absorption to occur. As shown in **Table 1**, the main dietary **disaccharides**, sucrose, lactose, maltose, isomaltose and trehalose, are all hydrolysed by the activity of their respective hydrolases expressed by small intestinal epithelial cells to yield hexoses, glucose, galactose and fructose. Lactase is the only disaccharidase that is commonly deficient in its activity with the subsequent maldigestion and delivery of lactose to the microbiota of the distal small and proximal large intestine. In contrast, of the dietary **oligosaccharides**, only malto-oligosaccharides ( $\alpha$ -glucans, for example, starch) are hydrolysed in the small intestine. The non  $\alpha$ -glucan oligosaccharides are also referred to as non-digestible oligosaccharides (NDOs) because the mammalian intestine does not synthesise hydrolases for galacto-oligosaccharides (GOS), fructo-oligosaccharides (FOS), isomalto- (soybean) oligosaccharides (IMO) and xylo-oligosaccharides (XOS).

Hence, NDOs are not digested and pass unaltered to the microbiota of the distal small intestine and proximal large intestine.

Dietary *monosaccharides* vary in their absorption. Glucose and galactose each have specific transporter-mediated uptake mechanisms which are efficient and rapid, such that complete absorption occurs in the proximal small intestine. As recently reviewed (20), fructose has two absorptive pathways – a low-capacity glucose-independent facilitated transport via the GLUT5 transporter and a high-capacity glucose-dependent fructose co-transport via the GLUT2 transporter. This dual system implies that fructose is absorbed with different efficiencies depending upon the concurrent presence of glucose. Thus, in the presence of at least equimolar concentrations of glucose, fructose is rapidly absorbed in the proximal small intestine, but fructose in excess of glucose (also referred to as ‘free fructose’) is slowly absorbed right along the small intestine. If there is rapid small intestinal transit or if the capacity of the GLUT5 transporter is low, the fructose is malabsorbed, a physiological phenomenon that is present in about one in three adults. Two other dietary hexoses are xylose and arabinose, both of which are passively absorbed (21). The implication of this is that only a proportion of ingested xylose (and presumably arabinose) will be absorbed and this will be reduced if small intestinal transit is fast.

*Polyols* are also believed to be absorbed by passive diffusion. The rate of absorption relates to the molecular size (22); diffusion occurs across pores in the epithelium and the size of the pores varies along the small intestine, with larger pores being present proximally (23). Transit time and dose will influence the degree of absorption, as will the presence of mucosal disease, such as celiac disease where pore size is reduced. The individual variation seen in the absorption of polyols is due to the variability in all of these factors (24,25).

The ability of an individual to absorb an individual carbohydrate can be tested by breath hydrogen testing, the principle of which is that hydrogen will be produced and a rise is detected in the breath if the tested carbohydrate reaches fermenting bacteria before it can be digested/absorbed. Most commonly, lactose and fructose are tested, but the methodologies applied, including the dose used, are not standardised. This is reviewed elsewhere (26).

### **Mechanisms by which poorly absorbed short-chain carbohydrates induce gut symptoms**

Short-chain carbohydrates are relatively small molecules and as such will be osmotically active in the intestinal lumen. This is of little consequence if rapidly absorbed as for glucose or rapidly digested then absorbed as for sucrose and lactose (where there is adequate lactase activity), but will increase small intestinal luminal water volume if poorly or slowly absorbed. This has been demonstrated by the ability of short-chain carbohydrates to increase small intestinal luminal water volume (27,28) and for a diet increased in poorly absorbed short-chain carbohydrates to increase ileal output in patients with a stoma (29). They are also rapidly fermented (30), which leads to the production of the hydrogen, carbon dioxide and methane gases. A high intake of them also preferentially favors the production of hydrogen over methane, the effect of which is to increase the gas volume per sugar molecule fermented (31). The other major fermentation products are short-chain fatty acids (SCFAs), which themselves promote sodium and water absorption, and promote motility (32-34).

The significance of these two actions – increased luminal water retention and gas production - is that they will lead to luminal distension. A major pathway of afferent input to the enteric nervous system is via stretch receptors. The presence of visceral hypersensitivity in the majority of patients with FGID sets the stage for such afferent input to lead to efferent output, via change in motility and/or sensations of bloating and pain. Limiting the degree of luminal distension and, therefore, the magnitude of the afferent input from the stretch receptors should reduce gastrointestinal symptoms

in patients with FGID. As outlined in **Fig 1**, this provides a rational mechanism for their modulating effects on gastrointestinal symptoms.

Poorly absorbed short-chain carbohydrates also have other physiological effects, the mechanisms of which are yet to be explained. Ingestion of FOS alters oesophageal motility with an increase in gastroesophageal reflux (35). Diets enriched in these short-chain carbohydrates have consistently induced tiredness in patients with IBS but not in healthy controls (30) and at least fructose is associated with depression in young women (36,37). Such observations raise questions about mechanisms of action, which may include effects on gut permeability, production of toxic metabolites in colon (38) and/or altered tryptophan levels (39).

#### **Efficacy of restriction of poorly absorbed short-chain carbohydrates in FGID**

Historically, carbohydrate malabsorption has been focussed mainly on lactose. Patients having gut symptoms temporally related to ingestion of milk are assessed by breath hydrogen response or rise in blood glucose after a large load of lactose (usually 50 g, equivalent of one liter of milk). Those with positive tests have been variably called as having lactose malabsorption or lactose maldigestion. The latter term may be less accurate as lactose can be malabsorbed in association with small bowel bacterial overgrowth (40) without apparent reduced activity of lactase. Those with malabsorption were then classified as 'intolerant' if symptoms developed during testing and placed on a lactose-free diet. However, the specificity to lactose malabsorption of symptoms induced during such testing appears to be low (41). The prevalence of lactose malabsorption in patients with FGID, or IBS specifically, is similar to that in the healthy population. In a large study of non-hospital-based Caucasian patients, 25% of 201 patients with FGID and 20% of 94 with IBS fulfilled breath hydrogen criteria of lactose malabsorption compared with 18% of 71 healthy controls (42). Lactose-free diet in patients with IBS and positive tests for lactose malabsorption has had variable efficacy, but all studies are uncontrolled observations. For example, 100% of 17 Finnish patients had marked

symptomatic relief with lactose restriction and this continued for five years in 14 (43), while 57% of 33 German patients (44), 44% of 110 compliant Italian patients (45) and 39% of 23 British patients had marked improvement in symptoms (46). There are no randomised controlled trials of lactose restriction in patients with IBS. Hence, the dilemma of whether lactose intolerance is the major basis for gut symptoms in a proportion of patients erroneously diagnosed with IBS, while supported by observational data, has yet to be definitively answered.

A similar story has emerged for fructose. Fructose malabsorption has also been implicated as a potential trigger for symptoms in patients with FGID following the initial observation of improved diarrhea in four patients with fructose malabsorption following dietary restriction of fructose (5). Malabsorption of pure fructose has been well documented in variable but similar proportions of healthy and IBS populations, depending upon the dose of fructose and breath hydrogen test criteria used (20,42). For example, 45% of 201 patients with FGID and 45% of 94 with IBS had fructose malabsorption after a 35 g load compared with 34% of 71 healthy controls (42). Co-ingestion with sorbitol has an additive effect on breath hydrogen production, but fructose is completely absorbed when co-ingested with glucose at equimolar concentrations (12,47,48). Observational studies have reported impressive symptom improvement when patients with fructose malabsorption were treated with a 'fructose-free diet'. For example, 50% of 94 patients with IBS and fructose malabsorption markedly improved with a 'fructose-free' diet (6), 58% of patients with functional gut symptoms had durable benefit over 5 years (49), and 67% of 26 patients with fructose and/or lactose malabsorption and functional bloating were completely or partly better after 12 months of a sugar-restricted diet (although data on those with fructose malabsorption alone was not provided) (50). Major limitations of the studies were that none were controlled and the details of the diet used were very limited. Further, it was uncertain whether the test diets were truly free of fructose, an almost impossible task, or if only foods in which fructose was present in excess of glucose were



restricted. Both factors probably contributed to the poor general uptake of this approach in clinical practice.

The most common NDO in a Western diet, short-chain fructans (FOS), also induces functional gut symptoms in challenge experiments (51). A longer term observational study of restricting free fructose and fructans in 62 consecutive patients with IBS and fructose (but not lactose) malabsorption showed durable symptomatic benefit across all symptoms of IBS in three out of four patients and a high adherence rate (77%) (52). Efficacy was observed across both IBS-D and IBS-C. This diet, then coined the 'fructose malabsorption diet', was fully described and was based upon knowledge of food composition and physiological principles. A subsequent double-blinded randomized, quadruple arm, placebo-controlled rechallenge trial was performed in 25 patients with IBS and fructose malabsorption who had responded to this diet. All food (low FODMAP) was provided to participants for 22 weeks (53). They consumed four test substances, fructose, fructans, fructose and fructans, in amounts estimated to be equal to an average Australian intake, or the control carbohydrate, glucose, for two weeks in a randomized order with a washout in between each test substance. The percentage of patients who reported negatively to a global symptom severity question was 77% for fructans, 70% for fructose, 79% for fructose and fructans, all greater than 14% for glucose ( $p < 0.002$ ). Thus, there was little doubt that the efficacy of the dietary restriction resided in the reduction/elimination of fructose and fructans.

Based upon the concepts that all short-chain carbohydrates induced the same effects in the distal small and proximal large intestine and that their effects are additive (supported by combination challenge studies (17,51,54,55)), this diet, using the same principles, was extended to include restriction of all classes of poorly absorbed short-chain carbohydrates, which were formally grouped together by the acronym, FODMAPs (56). Since then, the evidence-base for relief of functional gut

symptoms by this more comprehensive dietary restriction, termed the 'low FODMAP diet', has accumulated and will be described in more detail.

### **Evidence-base for symptom provocation and resolution by dietary FODMAPs**

Randomized controlled evidence for the efficacy of the low FODMAP diet in unselected patients with IBS (i.e., not restricted to those with fructose malabsorption) is accumulating. A randomized single-blinded crossover food-based FODMAP challenge study was carried out in 15 IBS patients and 15 healthy subjects (31). The only gastrointestinal symptom induced in the healthy subjects during the high compared with low FODMAP dietary arm was flatulence ( $p=0.007$ ), whereas the patients with IBS had increased severity of IBS symptoms ( $p<0.01$  for all), in addition to worsening heartburn ( $p=0.025$ ) and tiredness ( $p=0.012$ ). In a comparative, non-randomized study in the UK, the low FODMAP diet was more effective than dietary guidelines from the UK National Institute for Health and Clinical Excellence (NICE) (57) for symptom control in a series of consecutive patients referred for dietary management of their functional gut symptoms (58). Of the 43 patients receiving the low FODMAP dietary advice, 76% reported satisfaction with their symptom response compared with 54% of 39 patients who received standard (NICE guideline) advice ( $p=0.038$ ). The low FODMAP diet also provided greater improvements in individual symptoms, including bloating, abdominal pain and flatulence, compared with standard advice. Of importance, a randomized controlled trial of a low FODMAP diet, with habitual diet as the comparator, confirmed that four weeks of a low FODMAP diet improves symptoms in 68% (13/19) of patients with IBS compared with 23% (5/22;  $p=0.005$ ) (59). Significantly more patients in the low FODMAP group compared to the habitual diet group experienced a reduction in scores for bloating, borborygmi, urgency and overall symptoms and reported lower stool frequency and a greater proportion of stools with normal consistency at four weeks.

There have also been observational studies that have positively reported the clinical effectiveness of a low FODMAP diet in non-IBS patients who have functional gut symptoms. This has included patients with quiescent inflammatory bowel disease where more than 50% of 72 patients described a clinically significant benefit across all bowel symptoms except constipation (60). The frequency of pouch emptying or bowel actions in patients with ileorectal anastomosis were reduced in 15 patients without a colon (61). Likewise, the volume of ileostomy output was consistently reduced, albeit in a small cohort who were not troubled by large volume output (29).

### **Predictors of response to the low FODMAP diet**

In observational studies, adherence to the diet is a strong predictor of response. In a retrospective study (52), in which patient response was evaluated via a structured telephone interview 2–40 (median 14) months after implementing the low FODMAP diet, patients reported a high adherence rate to the diet (77%), which is much greater than rates previously reported (26-56%) for dietary approaches restricting one or more malabsorbed carbohydrates (5,50). Overall response was reported by 85% of patients adherent to the diet compared with 36% of those who were not. It could be suggested that the high adherence rate is related to the ongoing efficacy of the diet. Similar association of adherence to response was observed in patients with quiescent IBD (60). Several other factors, including higher education, working fewer hours, and using recommended cookbooks were identified as predictors of response to the low FODMAP diet, all consistent with better understanding of and greater effort in adhering to the diet. The pattern of bowel habits, whether diarrhea- or constipation-predominant or mixed, has not been a predictor of response, but this has not been prospectively examined. Similarly, no studies have systematically or prospectively examined methods of teaching (one-on-one versus group versus literature-based teaching, dietician versus physician) to predict response.

### **Safety of dietary FODMAP restriction**

Altering food intake with the purpose of reducing fermentable substrates to the distal small intestine and colon potentially has implications for the health-promoting effects of specific substrates or fermentation in general. Some FODMAPs have well established prebiotic activity (for example, FOS and GOS) that leads to the preferential growth of, for example, bifidobacteria (62). Since prebiotics are putatively associated with health-promoting effects (63), a reduction of intake of FODMAPs might have longer term adverse consequences. A reduction of the proportion and concentration of bifidobacteria in the faeces has recently been demonstrated after four weeks of a dietician-taught low FODMAP diet compared with that of patients continuing their habitual diet in a randomized study (59). There is accumulating evidence that the gut microbiome is altered in IBS patients compared to healthy controls, with lower levels of bifidobacteria (63-66). Exaggeration of this effect with the low FODMAP diet might aggravate the adverse health effects attributed to such a relative deficiency of bifidobacteria. However, there are no clinical outcome data regarding whether such a deficiency has any real detrimental effects. This aspect of safety clearly warrants prospective studies with clinical outcomes as end points.

Reducing fermentable substrates delivered to the colon in general may, at least theoretically, have implications for colorectal carcinogenesis and colonic inflammation. Factors associated with the colonic luminal microenvironment are almost certainly involved with carcinogenesis, For example, the exposure to butyrate is protective, phenols and cresols are potentially pro-carcinogenic and a lower luminal pH is believed to be protective [67,68]. On the other hand, excessive fermentation in the proximal colon can be detrimental, at least in animal models, in terms of epithelial injury and susceptibility to inflammation [69-72]. To date, one randomized controlled trial in patients with IBS shows that a low FODMAP diet compared to habitual diet does not alter fecal pH or content of short chain fatty acids after 4 weeks of intervention (59). There is no information on the effects of the low FODMAP diet on other components of the colonic microenvironment.

Modifying the intake of FODMAPs excludes a wide variety of foods and a change in food choice invariably affects the nutritional composition of the diet. The nutritional adequacy of such a restrictive diet has therefore been investigated. A randomized controlled trial in patients with IBS showed that the dietary intake of carbohydrates, starch and total sugars were lower following four weeks of FODMAP restriction versus habitual diet (59). To some extent, this was expected due to the low FODMAP group being advised to reduce certain sources of carbohydrate-rich foods. Total energy, protein, fat and non-starch polysaccharide intakes were not different between groups. Calcium intake was lower in the FODMAP restricted group which could be explained by a reduction in the intake of dairy products or measurement error due to the lack of comprehensive nutritional composition data for some lactose-free products. To ensure nutritional adequacy of a low FODMAP diet, dietary advice should be tailored to ensure that calcium intake meets nutritional requirements and alternative sources of carbohydrate rich foods are made. These findings support delivery of this complex dietary approach by a dietician experienced in teaching the low FODMAP diet.

### **Principles of dietary management**

The low FODMAP diet is relatively complex and patients benefit from being provided with an explanation of the mechanism of action (52,73,74). It involves the reduction, not complete avoidance, of FODMAPs in the diet. Foods have been classified into high and low FODMAP content, and, therefore, knowledge of the FODMAP status of foods is an important skill for patient education. Low FODMAP foods that are suitable alternatives to foods high in FODMAPs are encouraged; for example, rather than completely restricting fruit, intake of high FODMAP fruit is reduced and intake of low FODMAP fruit is encouraged.

The low FODMAP dietary education has two key components. First, all known or suspected types of FODMAP groups are strictly restricted from the patient's diet, according to background food habits for a period of approximately 6-8 weeks (52,73). This allows the patient and provider to determine

whether FODMAP restriction is beneficial. Though most low FODMAP responders improve within a period of several weeks, extension of the broad exclusion period helps to build patient and provider confidence that symptom relief is from the diet rather than coincidence. Secondly, patients are then taught to reintroduce individual FODMAPs to test their individual tolerance of each FODMAP via a series of food challenges (73,74) in order to avoid unnecessary over-restriction. It is important to note that the evidence-base for the low FODMAP diet has derived from experienced specialist gastrointestinal dieticians delivering the education in a one-to-one consultation. It is as yet uncertain whether equivalent efficacy can be achieved by self-teaching or other less structured methods. The potential differences between the two learning methods are summarized in Table 4.

Breath hydrogen testing can be helpful to identify patients who completely absorb a moderate load of specific sugars. This is particularly applicable to fructose and lactose where large variance of the absorptive capacity across individuals is evident. This information can then be helpful to indicate that it is less important to restrict dietary intake of these particular saccharides, potentially rendering the diet less restrictive.

### **Gaps in understanding**

The low FODMAP diet has a growing body of evidence supporting its efficacy, although randomized controlled trials over longer periods of time and across broader populations are needed. Research to date has largely focused on functional bowel disorders. The role of FODMAPs in upper gut functional problems has had scant attention, although the observation that FOS increases gastroesophageal reflux (35) and the increase in heartburn with a high compared with low FODMAP diet in patients with IBS in a short-term study (31) provide evidence that such a question is reasonable to ask. Whether the diet is purely one that avoids triggering symptoms or whether it might have beneficial or detrimental effects on visceral hypersensitivity and other abnormalities of the ENS has not been addressed. The dietary intake of FODMAPs in patients with FGID has only been

addressed in one study (46). The Monash CNAQ (Complete Nutritional Assessment Questionnaire) has been developed and validated to enable semi-quantitative evaluation of the intake of FODMAPs on a population basis to be measured (75). The application of such a tool may provide the missing information on whether excessive intake of FODMAPs might underlie symptom genesis in some or many patients with FGID.

The low FODMAP diet is not a panacea for IBS or other FGIDs. Identification of patient profiles that predict dietary response is clinically important and needed. The implementation of the low FODMAP diet using individual one to one dietician teaching is impractical in parts of the world where there are few trained dieticians and is a relatively time-inefficient way of delivering a therapy to a large population. The comparative efficacy of delivering the diet via, for example, group education, internet-based resources or printed material requires evaluation. Further studies are required to assess the longer term safety of the low FODMAP diet as outlined above. Finally, a very important factor in the use of the low FODMAP diet is to ensure accurate knowledge of local food composition. While there are data on FODMAP composition of foods from many countries worldwide (12, 47, 48, 76-85), much is incomplete and methodologies are inconsistent. This will require ongoing work evaluating indigenous foods specific to different international locales. Fortunately, methods for the evaluation using enzymatic and high performance liquid chromatography techniques are well described (86-89).

## **Conclusions**

The FODMAP concept has been supported by numerous data that provide a significant link between food intake and symptoms in patients with FGID. While a complex concept for patients to grasp, the mechanisms of action are well understood and reduced FODMAP diets have resulted in symptom relief in the majority of adherent patients when taught by a trained dietician. Its application

amongst patients with FGID is justified. Further research studies are warranted to expand our knowledge of applications and implications of its use.



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**Table 1. Classification of the main carbohydrates present in the diet and their small intestinal absorption or hydrolysis**

Class	Subgroup	DP	Name	Constituent Monosaccharide	Absorption/hydrolysis in small intestine
Sugars	Monosaccharides	1	Glucose	n/a	Active absorption (rapid)
			Fructose	n/a	Active absorption (slow in excess of glucose)
			Galactose	n/a	Active absorption (rapid)
			Xylose	n/a	Passive absorption
			Arabinose	n/a	Passive absorption
	Disaccharides	2	Sucrose	Glucose + fructose	Hydrolysed
			Lactose	Glucose + galactose	Hydrolysed (if lactase activity)
			Maltose, isomaltose	Glucose + glucose	Hydrolyzed
Trehalose			$\alpha$ -glucose + $\alpha$ -glucose	Hydrolyzed	
Polyols	Polyols (sugar alcohols)	1	Sorbitol, mannitol, lactitol, xylitol, erythritol, maltitol, isomalt	n/a	Passive absorption
Oligosaccharides	Malto-oligosaccharides ( $\alpha$ -glucans)	3-9	Maltodextrin	Glucose	Hydrolyzed
		3-4	Galacto-oligosaccharides (GOS) <ul style="list-style-type: none"> <li>• Raffinose</li> <li>• Stachyose</li> </ul>	One fructose, one glucose and one galactose One fructose, one glucose and two galactose	No hydrolysis
	Non- $\alpha$ -glucan (non-digestible) oligosaccharides (NDO)	4	Fructo-oligosaccharides (FOS)	Fructose polymer (glucose terminal end)	No hydrolysis
		<10	Isomalto- (soybean) oligosaccharides (IMO)	Glucose with other monosaccharides	Partly hydrolysed only

		2-4	Xylo-oligosaccharides (XOS)	Xylose	No hydrolysis
Polysaccharides	Starch ( $\alpha$ -glucan)	>10	Amylose, amylopectin, modified starches	Glucose polymer linked with $\alpha$ -glycosidic bonds	Hydrolysis except for resistant starch
	Non-starch polysaccharides	>10	Cellulose (eg. $\beta$ -glucan)	Glucose polymers linked with $\beta$ -glycosidic bonds	No hydrolysis
		>10	Hemicellulose (e.g., arabinoxylans)	Always xylose and can include glucose, mannose, galactose, rhamnose, and L-arabinose)	No hydrolysis
		>10	Pectin	Rhamnose with L-arabinose, galactose, xylose	No hydrolysis
		>10	Fructans (e.g., inulin)	Fructose polymer (glucose terminal end)	No hydrolysis
		>10	Plant gums and mucilages	hexose, methyl pentose and pentose sugars joined by glycosidic linkages to uronic acid residues	No hydrolysis
		>10	Hydrocolloids (e.g., xanthan gum, gum Arabic, guar gum)	<i>Xanthan Gum</i> : Fermentation of glucose, sucrose, or lactose by bacteria <i>Gum Arabic</i> : polysaccharides + glycoproteins <i>Guar gum</i> : galactose and mannose	No hydrolysis
	12	Polydextrose	Glucose polymer + sorbitol (10%)	Hydrolyzed	

**Table 2. Comparison of rationale evidence and dietary restrictions in published diets aimed at alteration of carbohydrate intake**

	<b>Specific Carbohydrate</b>	<b>Anti-candida</b>	<b>Paleolithic</b>	<b>Low carbohydrate</b>	<b>Lactose-free</b>	<b>Fructose-free</b>	<b>Low FODMAP</b>
<b>Target audience</b>	IBS & IBD	All ill-health	IBD & FGID	Weight, body fat & glucose control, cardiovascular disease, other	Diarrhea, IBS	Functional gut symptoms, IBS	Functional gut symptoms, IBS
<b>Rationale</b>	Some carbohydrates lead to overgrowth of harmful bacteria (& waste and irritants they produce) in the gut so that di- & poly-saccharides cannot be digested properly & the gut cannot heal	Overgrowth of various species of candida, leads to 'bowel troubles'	Modern diet creates altered patterns of gene expression that lead to disease.	Eating too many carbohydrates (especially refined) leads to blood sugar imbalances, weight gain and cardiovascular problems	Osmotic effect of malabsorbed lactose in people with presumed or measured lactase insufficiency induces diarrhea or IBS symptoms	Poorly absorbed fructose exerts an osmotic effect and is fermented leading to diarrhea and bloating	All osmotically active poorly absorbed substrates that are rapidly fermentable will lead to luminal distension in distal small bowel and colon with subsequent induction of pain, bloating and/or altered bowel habit
<b>Evidence-base in FGID</b>	Nil	Nil	Nil	Observational evidence only of very low CHO diet in IBS-D (78)	Efficacy in lactose-induced diarrhea but not IBS	Observational evidence only of efficacy in IBS & functional bloating	Efficacy in observational, comparative & RCT rechallenge studies
<b>Dietary principle</b>	Restricts the use of complex carbohydrates & eliminates refined sugar, all grains, legumes & starches	Prohibits intake of sugar and most other sweeteners, sweet fruits, yeast-containing baked foods, mushrooms, fermented products	Based on presumed ancient diet of wild plants and animals habitually consumed during the Paleolithic era with development of agriculture	Limits foods high in carbohydrates. Replaces with foods with higher % protein & fat. Degree of carbohydrate restriction varies ('low', 'very low', 'ultra-low')	Restrict dietary intake to less than 4 g lactose per serve, as small amounts of lactose (less than 4g) usually well tolerated	Apparently completely restrict all sources of fructose	Replace with foods containing more than threshold amount of poorly absorbed short-chain carbohydrates with suitable alternatives containing well absorbed short-chain carbohydrates

<b>Published dietary detail</b>	Comprehensively described	Comprehensively described	Comprehensively described	Comprehensively described	Comprehensively described	Not defined - 'fructose-free'	Comprehensively described
<b>Sugar (sucrose) permitted</b>	No	No	No (honey allowed)	No	Yes	Unlikely	Yes
<b>Refined carbohydrates permitted</b>	No	No	No	No	Yes	Unclear	Not wheat, barley or rye
<b>Wholegrain carbohydrates permitted</b>	No	Not if contains yeast	No	No	Yes	Unclear	Not wheat, barley or rye
<b>Legumes permitted</b>	No	Yes	No	No	Yes	Unclear	No
<b>Fruit permitted</b>	Yes	Not dried, juices or sweet fruits	Berries and wild fruits	Berries, citrus	Yes	Unlikely	Many but not stone fruit, apples, pears, watermelon, mangoes
<b>Starchy vegetables permitted</b>	No	Yes	Yes (roots and tubers)	No	Yes	Unclear	Yes (sweet potato in moderation)
<b>Non-starchy vegetables permitted</b>	Yes	Yes	Yes (plant leaves, seaweed, sea grasses)	Yes	Yes	Unclear	Some but not onion, leek, garlic, artichoke, spring onion, mushrooms cauliflower
<b>Dairy foods permitted</b>	Only hard cheeses & homemade yoghurt	None except for live yoghurt	No	Hard cheeses, others vary	All except lactose-containing	Likely	All except lactose-containing

**Table 3. Characteristics and sources of common FODMAPs**

<b>F Fermentable</b>		By colonic bacteria		
<b>O Oligosaccharides</b>	Fructans, Galacto-Oligosaccharides	No absorption (no small intestinal hydrolases)	Wheat, barley, rye, onion, leek, white part of spring onion, garlic, shallots, artichokes, beetroot, fennel, peas, chicory, pistachio, cashews, legumes, lentils, chickpeas	
<b>D Disaccharides</b>	Lactose	↓ digestion, therefore ↓ absorption in 10-95%	Milk, custard, ice cream, yoghurt	
<b>M Monosaccharides</b>	'Free fructose' (fructose in excess of glucose)	Slow, active absorption - poor in ~1 in 3	Apples, pears, mangoes, cherries, watermelon, asparagus, sugar snap peas, honey, high fructose corn syrup,	
<b>A And</b>				
<b>P Polyols</b>	Sorbitol, mannitol, maltitol, xylitol	Slow passive absorption	Apples, pears, apricots, cherries, nectarines, peaches, plums, watermelon, mushrooms, cauliflower, artificially sweetened chewing gum and confectionery.	

**Table 4. A hypothetical comparison of education in the low FODMAP diet as delivered by a trained dietician compared to that as a self-taught option via instruction sheets, books or the internet**

	<b>Dietician-delivered</b>	<b>Self-taught</b>
<b>Structure of education</b>	Complex, requires detailed explanation	Haphazard unchecked access to information
<b>Suitability of foods explained comprehensively</b>	Focus on suitable foods not just problematic foods	Potential misinformation – lists on internet, out-of-date information
<b>Risk of unnecessary over-restriction</b>	Minimises risk	Increases risk (e.g., failure to rechallenge)
<b>Ability to attain nutritional adequacy</b>	Ensures nutritional adequacy	Not monitored for nutritional adequacy
<b>Personalized advice</b>	Individualized	Not individualized

**Figure 1.**

Features of the mechanisms of action of poorly absorbed short-chain carbohydrates (FODMAPs) for the development of gastrointestinal symptoms in FGID. a) FODMAPs are poorly absorbed in the small intestine and arrive into the colon, populated with microbiota. b) Their osmotic activity leads to increased water retention within the lumen of the small and large bowel. c) FODMAPs are substrates for colonic bacterial fermentation, resulting in the rapid production of gas and subsequent luminal distension.