

# Diet and the Gut

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## Review Team

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- The following authors reported that they have no conflicts of interest in relation to the development of this guideline: Govind Makharia, Julio Bai, Sheila Crowe, Tarkan Karakan, Yeong Yeh Lee, Nevin Oruc, Eamonn Quigley, David Sanders, Cihan Yurdaydin.
- Peter Gibson has written a recipe/education book on the low FODMAP diet. Monash University sells a digital application, booklets, and on-line education on the low FODMAP diet.
- Caroline Tuck, Lyndal McNamara, and Jane Muir reported that Monash University sells a digital application and booklets on the low FODMAP diet, and an online course to train dietitians in how to implement the low FODMAP diet. Funds raised contribute to research in the Department of Gastroenterology at Monash University. The authors receive no personal remuneration.
- Anton LeMair acts as guidelines development consultant for WGO.

## Contents

|          |   |           |
|----------|---|-----------|
| <b>1</b> | <b>Key recommendations and cascades .....</b>                                     | <b>4</b>  |
| 1.1      | Introduction .....  | 4         |
| 1.2      | Cascades of diet options and alternatives .....                                   | 5         |
| <b>2</b> | <b>Fiber in the diet .....</b>  | <b>7</b>  |
| 2.1      | Introduction .....  | 7         |
| 2.1.1    | Types of dietary fiber .....  | 8         |
| 2.1.2    | Beneficial effects of dietary fibers .....  | 8         |
| 2.1.3    | Interaction of dietary fibers with the gut microbiota .....                       | 10        |
| 2.2      | Role of dietary fiber in gastrointestinal conditions .....                        | 10        |
| 2.2.1    | Constipation .....  | 10        |
| 2.2.2    | Irritable bowel syndrome (IBS) .....  | 11        |
| 2.2.3    | Inflammatory bowel disease (IBD) .....  | 11        |
| 2.2.4    | Diverticular disease .....  | 11        |
| 2.2.5    | Colorectal cancer .....   | 12        |
| 2.2.6    | Clinical indications for a low-fiber diet .....                                   | 12        |
| 2.3      | Fiber intake and recommendations .....  | 13        |
| 2.4      | Addressing the “fiber gap” .....  | 14        |
| 2.5      | Cascade guidelines—dietary fiber .....  | 16        |
| <b>3</b> | <b>The low FODMAP diet .....</b>  | <b>16</b> |
| 3.1      | Introduction .....  | 16        |
| 3.1.1    | What is the low FODMAP diet? .....  | 16        |
| 3.1.2    | How to implement a low FODMAP diet: reintroduction and problems/limitations ..... | 17        |
| 3.2      | Gastrointestinal conditions and the low FODMAP diet .....                         | 20        |
| 3.2.1    | Indications for low FODMAP diet .....   | 20        |
| 3.2.2    | The low FODMAP diet, functional dyspepsia (FD), and IBS .....                     | 21        |
| 3.3      | Cascade guidelines—FODMAP .....   | 21        |
| <b>4</b> | <b>Carbohydrate intolerance .....</b>   | <b>21</b> |
| 4.1      | Lactose intolerance / lactase deficiency .....                                    | 21        |
| 4.1.1    | Definition of terms .....   | 21        |
| 4.1.2    | Lactose intolerance in perspective: when is it relevant? .....                    | 21        |
| 4.1.3    | Symptoms of lactose intolerance .....   | 22        |
| 4.1.4    | How to diagnose: in resource-limited and well-resourced settings .....            | 22        |
| 4.1.5    | How to treat .....  | 23        |
| 4.2      | Fructose intolerance .....  | 24        |
| 4.2.1    | Definition of terms .....   | 24        |
| 4.2.2    | Proposed mechanisms .....   | 25        |
| 4.2.3    | Intake of fructose .....  | 25        |
| 4.2.4    | Use of dietary modification .....   | 25        |
| 4.2.5    | Recommendations .....   | 25        |
| 4.3      | Sucrase–isomaltase deficiency .....   | 26        |
| 4.3.1    | Congenital sucrase–isomaltase deficiency .....                                    | 26        |
| 4.3.2    | Secondary or acquired sucrase–isomaltase deficiency .....                         | 26        |
| 4.3.3    | Diagnosis .....   | 26        |
| 4.3.4    | Treatment .....   | 27        |

|          |  |           |
|----------|--|-----------|
| 4.4      | Cascade guidelines .....                           | 27        |
| <b>5</b> | <b>Other dietary interventions in IBS.....</b>     | <b>27</b> |
| <b>6</b> | <b>Appendix .....</b>                              | <b>29</b> |
| 6.1      | Abbreviations .....                                | 29        |
| 6.2      | Organizations publishing relevant guidelines ..... | 30        |
| 6.3      | References.....                                    | 30        |

### List of tables

|          |  |    |
|----------|--|----|
| Table 1  | Cascade of guidelines on dietary fiber .....                           | 5  |
| Table 2  | Cascade of recommendations on FODMAP .....                             | 5  |
| Table 3  | Cascade of recommendations on carbohydrate intolerance.....            | 6  |
| Table 4  | Definitions .....  | 8  |
| Table 5  | Summary of the physiological effects of different types of fiber ..... | 9  |
| Table 6  | Recommended and actual fiber intake in different countries.....        | 13 |
| Table 7  | Possible barriers to dietary change and solutions .....                | 15 |
| Table 8  | Clinical management flowchart for using the low FODMAP diet .....      | 18 |
| Table 9  | Three-step process for implementing the low FODMAP diet.....           | 19 |
| Table 10 | Food sources of FODMAPs and suitable alternatives .....                | 19 |
| Table 11 | Clinical symptoms and pathophysiology of lactose intolerance.....      | 22 |
| Table 12 | Food items that are restricted or allowed in lactose intolerance.....  | 24 |
| Table 13 | Abbreviations used in this WGO guideline.....                          | 29 |

### List of figures

|        |  |    |
|--------|--|----|
| Fig. 1 | Dietary intake fiber by region (data from 2010) .....                                  | 14 |
| Fig. 2 | Classification of indigestible and slowly absorbed carbohydrates relevant in IBS ..... | 17 |

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## 1 Key recommendations and cascades

### 1.1 Introduction

This guideline was produced in connection with the World Digestive Health Day (WDHD) held in 2016 on the theme of “Diet and the Gut.” The Guideline Development Review Team consisted of experts on the WDHD theme as well as invited experts, including diet and nutrition specialists, pharmacists, and primary-care physicians.

Given the central role of the digestive tract and its related organs in the processes of digestion and absorption, it should come as no surprise that the food we eat has critical and complex interactions with the gastrointestinal tract and its contents, including the microbiota. The nature of these interactions is influenced not only by the composition of the diet and the integrity of the gastrointestinal tract, but also by psychosocial and cultural factors. The general public—and in particular those who suffer from gastrointestinal ailments—rightly perceive their diet as being a major determinant of such symptoms and seek guidance on optimal dietary regimens. Many medical practitioners, including gastroenterologists, are unfortunately often ill-prepared to deal with such issues. This is a reflection of the lack of education on the topic of diet and nutrition in many curricula.

Dietary changes have the potential to alleviate symptoms, but they may also result in regimens that are nutritionally deficient in one or more respects. It is vital, therefore, that whenever possible the medical practitioner should engage the services of a skilled nutritionist/dietitian to evaluate a given individual’s nutritional status, instruct the patient on new diet plans, and monitor progress. It is also incumbent on gastroenterologists to become educated on modern dietary practices as they relate to gastrointestinal health and disease. We hope that this guideline will become a valuable resource in this regard.

Diet in general is a very broad subject; we have therefore decided to be selective and have focused on certain diets and conditions for which the diet has a real causative or therapeutic role in adults: celiac disease, dietary fibers, FODMAPs (fermentable oligosaccharides, disaccharides, monosaccharides, and polyols), carbohydrate intolerance, and the role of diet in irritable bowel syndrome (IBS). The topic of celiac disease has already been dealt with in the WGO guideline on celiac disease published in 2016, which should be referred to for further details [1,2].

With WGO “cascades,” the intention is to recognize differences in disease epidemiology, sociocultural factors, and health-care provision that exist in different parts of the world and the ways in which they preclude, in most instances, the promulgation of a “one size fits all” or a single gold-standard approach. The Appendix in this guideline lists organizations that produce relevant guidelines. This Global WGO Guideline includes a set of cascades intended to provide context-sensitive and resource-sensitive options for the dietary approach to gastrointestinal conditions. Through the WGO cascades, the WGO Guidelines program aims to provide clinical practice recommendations that are useful in many different environments across the world.

This WGO Guideline on diet and the gut is intended for use by health providers, including family-care and primary-care physicians, gastroenterologists, pharmacists, and nutritionists/dietitians.

The WGO Guidelines are produced through a systematic development process for achieving an expert consensus on the basis of the medical and scientific literature, existing practice guidelines, and regional best-practice standards. All available sources were used to develop this guideline. Monthly high-level evidence literature searches in EMBASE/Medline are delivered to the review team members as alerts, and are scanned by team members to identify new insights and evidence for the next guideline update.

## 1.2 Cascades of diet options and alternatives

Tables 1–3 present cascades of resource-sensitive diet options and alternatives for countries and regions with different levels of resources, access, culture, and epidemiology.

**Table 1** Cascade of guidelines on dietary fiber

|   |
|---|
| <b>Individual strategies</b>  |
| <ul style="list-style-type: none"> <li>• Dietary education and/or behavior-change counseling for individuals with a low-fiber diet or health condition modulated by fiber intake</li> <li>• Increased consumption of high-fiber foods, including fruit, vegetables, legumes, and whole grains</li> <li>• Use of fiber supplementation where recommended intake cannot be met through diet alone, or there is evidence of therapeutic benefit for a specific health condition</li> </ul> |
| <b>Strategies for governments, international partners, civil society, nongovernmental organizations, and the private sector</b>   |
| <ul style="list-style-type: none"> <li>• Shape healthy environments</li> <li>• Make healthier, high-fiber food options affordable and easily accessible to all</li> <li>• Promote high-fiber food options and increase public awareness of the health benefits associated with consumption</li> </ul>   |
| <b>Strategies for the food industry</b>   |
| <ul style="list-style-type: none"> <li>• Increase the fiber content of processed foods through changes in food-processing practices and/or fortification of highly processed foods</li> <li>• Increasingly introduce and promote production of innovative, fiber-rich products</li> </ul>   |

**Table 2** Cascade of recommendations on FODMAP

|  |
|--|
| <b>Extensive resources (gold standard)</b>   |
| <ul style="list-style-type: none"> <li>• Clinical evaluation to confirm the diagnosis of IBS, including exclusion of celiac disease</li> <li>• Dietary interview by an expert dietitian to evaluate dietary intake, meal pattern, level of FODMAP consumption</li> <li>• Dietary instruction with regular follow-up</li> <li>• Initial dietary reduction of FODMAP intake for 2–6 weeks, providing adequate education on foods to be consumed and to be avoided, as well as adaptation to suit the patient's other dietary needs (e.g., other dietary intolerances, food preferences, religious requirements)</li> <li>• Follow-up after 2–6 weeks to evaluate the effect of the low FODMAP diet on symptom control</li> <li>• When good symptom control has been achieved: strategic re-challenge to assess tolerance to individual FODMAP subgroups, followed by long-term management planned on the basis of the response to the re-challenge</li> <li>• When poor symptom control has been achieved: assessment of compliance with dietary recommendations, consideration of other dietary and nondietary management strategies</li> </ul> |

### Medium resources

- Clinical evaluation to confirm the diagnosis of IBS, including exclusion of celiac disease
- Dietary interview by a dietitian to evaluate dietary intake
- Dietary instruction with semi-frequent follow-up
- Initial dietary reduction of FODMAP intake for 2–6 weeks, providing adequate education on foods to be consumed and to be avoided, as well as adaptation to suit the patient's other dietary needs (e.g., other dietary intolerances, food preferences, religious requirements)
- Follow-up assessment within 3 months to evaluate the effect of the low FODMAP diet on symptom control
- When good symptom control has been achieved: strategic re-challenge to assess tolerance to individual FODMAP subgroups, followed by long-term management planned on the basis of the response to the re-challenge
- When poor symptom control has been achieved: assessment of compliance with dietary recommendations, consideration of other dietary and nondietary management strategies

### Limited resources

- Clinical evaluation to confirm the diagnosis of IBS, including exclusion of celiac disease
- Dietary interview by a dietitian, if available, to evaluate dietary intake
- Dietary instruction with semi-frequent follow-up, if possible
- Initial dietary reduction of FODMAP intake for 2–6 weeks
- Follow-up within 6 months to evaluate the effect of the low FODMAP diet on symptom control
- When good symptom control has been achieved: strategic re-challenge to assess tolerance to individual FODMAP subgroups

FODMAP, fermentable oligosaccharides, disaccharides, monosaccharides, and polyols; IBS, irritable bowel syndrome.

**Table 3** Cascade of recommendations on carbohydrate intolerance

| Extensive resources (gold standard) |  |
|-------------------------------------|--|
| Lactose intolerance                 | <ul style="list-style-type: none"> <li>• <i>Diagnosis:</i> challenge with 25 or 50 g lactose and assessment of the hydrogen and methane breath response; <i>or</i> food challenge with symptom monitoring to identify intolerance</li> <li>• <i>Management:</i> assessment and education by a dietitian to ensure suitable intake of calcium-rich foods. Reduction of dietary intake of foods high in lactose, with replacement using lactose-free milk and yoghurt products and/or use of oral <math>\beta</math>-galactosidase</li> </ul>                              |
| Fructose                            | <ul style="list-style-type: none"> <li>• Fructose intolerance should be considered as a component of the low FODMAP diet (see Table 2)</li> </ul>  |
| Sucrase–isomaltase deficiency*      | <ul style="list-style-type: none"> <li>• <i>Diagnosis:</i> the value of enzyme activity assessment in duodenal/jejunal biopsies has been established in children. The value of this test in adults is not yet established</li> <li>• <i>Management:</i> for congenital deficiency, a starch-restricted and sucrose-restricted diet followed by re-challenge, supervised by a dietitian; sacrosidase enzyme supplementation</li> </ul>  |
| Medium resources                    |  |
| Lactose intolerance                 | <ul style="list-style-type: none"> <li>• <i>Diagnosis:</i> challenge with 25 or 50 g lactose and assessment of the hydrogen and methane breath response; <i>or</i> food challenge with symptom monitoring to identify intolerance</li> <li>• <i>Management:</i> Assessment and education by a dietitian, if available, to ensure suitable intake of calcium-rich foods. Reduction of dietary intake of foods high in lactose, with replacement using lactose-free milk and yoghurt products and/or use of oral <math>\beta</math>-galactosidase, if available</li> </ul> |
| Fructose                            | <ul style="list-style-type: none"> <li>• Fructose intolerance should be considered as a component of the low</li> </ul>  |

| FODMAP diet                     |  |
|---------------------------------|--|
| * Sucrase–isomaltase deficiency | <ul style="list-style-type: none"> <li>• <i>Diagnosis:</i> the value of enzyme activity assessment in duodenal/jejunal biopsies has been established in children. The value of this in adults is not yet established</li> <li>• <i>Management:</i> consideration should be given to a starch-restricted and sucrose-restricted diet, followed by re-challenge supervised by a dietitian for congenital enzyme deficiency</li> </ul>  |
| Limited resources               |  |
| Lactose intolerance             | <ul style="list-style-type: none"> <li>• <i>Diagnosis:</i> Food challenge with symptom monitoring to identify intolerance</li> <li>• <i>Management:</i> Dietary education by a health professional, if available, to ensure suitable intake of calcium-rich foods. Reduction of dietary intake of foods high in lactose, with replacement using lactose-free milk and yoghurt products and/or use of oral <math>\beta</math>-galactosidase, if available</li> </ul>                      |
| Fructose                        | <ul style="list-style-type: none"> <li>• Fructose intolerance should be considered as a component of the low FODMAP diet.</li> </ul>   |
| * Sucrase–isomaltase deficiency | <ul style="list-style-type: none"> <li>• <i>Diagnosis:</i> In the absence of clinical testing, consideration should be given to the clinical picture: symptom types and response to treatment in children with suspected congenital deficiency only</li> <li>• <i>Management:</i> Consideration should be given to a starch-restricted and sucrose-restricted diet with instruction from a dietitian, if available, and only in children with suspected congenital deficiency</li> </ul> |

FODMAP, fermentable oligosaccharides, disaccharides, monosaccharides, and polyols.

## 2 Fiber in the diet

### 2.1 Introduction

Dietary fibers are carbohydrates (both natural and synthetic) that resist digestion in the small intestine of humans and convey a physiological health benefit [3,4]. Fiber adds bulk to the diet, reduces energy density in foods, and may improve glycemic control and prevent or reduce constipation [3,5]. In many countries, a large number of individuals do not consume enough dietary fiber to meet recommended targets [3,5]. Good dietary fiber sources include: whole grains, nuts and seeds, vegetables, and fruit [3,5]. A greater intake of dietary fiber has been associated with a lower risk of several chronic diseases, including cardiovascular disease and diabetes, and it may reduce the risk of all-cause mortality [5–9]. Dietary fiber may be included in the nutrition panel on food labels, and it is typically listed as a subset of total carbohydrates.

Table 4 Definitions

| Concept                       | Definition  |
|-------------------------------|---|
| Dietary fiber [10,11]         | <ul style="list-style-type: none"> <li>The edible parts of plants or analogous carbohydrates that are resistant to digestion and absorption in the human small intestine, with complete or partial fermentation in the large intestine. Dietary fiber includes polysaccharides, oligosaccharides, lignin, and associated plant substances. Dietary fibers promote beneficial physiological effects including laxation, and/or blood cholesterol attenuation, and/or blood glucose attenuation.</li> <li>Fibers that are incorporated into foods as additives should demonstrate functional human health outcomes in order to receive a fiber classification.</li> <li>A database has been developed, listing studies that test fiber and physiological health outcomes identified by experts at the Ninth Vahouny Conference [12].</li> </ul> |
| Diet and the gut              | <ul style="list-style-type: none"> <li>Clinical diet, intended to treat (part of) the disease or disorder, or to correct excess or deficiency in relation to selected gastrointestinal disorders</li> </ul>   |
| “Fiber-rich”                  | <ul style="list-style-type: none"> <li>3 g or more of fiber per labeled serving</li> </ul>  |
| “High in fiber” on food label | <ul style="list-style-type: none"> <li>Must contain at least 5 g per serving</li> </ul>   |
| Energy density                | <ul style="list-style-type: none"> <li>Relationship of calories to the weight of food (calories per gram)</li> </ul>  |
| Fiber density                 | <ul style="list-style-type: none"> <li>Fiber content in a specified amount of a food or diet, usually per 100 g of food, and for comparing diets usually per 1000 kcal intake</li> </ul>  |

### 2.1.1 Types of dietary fiber

Food naturally contains a mixture of soluble and insoluble fibers, and both types have important health benefits in the context of a high-fiber diet [3,5]. Although the solubility of fiber was once thought to determine its physiological effect, more recent studies suggest that other properties of fiber, especially fermentability and viscosity, are more important, and plant components (such as antioxidant compounds) associated with dietary fiber may also contribute to reduced disease risk [5,13].

### 2.1.2 Beneficial effects of dietary fibers

For a summary of the physical characteristics and physiological benefits of naturally-occurring fibers, see Table 5. The following is a list of well-established beneficial physiological effects associated with the consumption of a high-fiber diet with whole foods in general [12]:

- Reduction in blood total and/or low-density lipoprotein (LDL) cholesterol
- Reduction in postprandial blood glucose and/or insulin levels
- Increased stool bulk and/or decreased transit time
- Increased production of short-chain fatty acids due to fermentation by colonic microbiota

In addition, the following physiological effects of dietary fibers are considered probable, but require further scientific substantiation [12]:



- Reduced blood pressure
- Increased satiety
- Weight loss/reduction in obesity
- Positive modulation of colonic microbiota

**Table 5** Summary of the physiological effects of different types of fiber (adapted from Eswaran et al. 2013) [14]

| Type                | Oligosaccharides   |  | Fibers  |   |  |
|---------------------|--|--|---|---|--|
|                     | Yes  | Yes  | Intermediate  | No  | No   |
| <i>Soluble</i>      | Yes  | Yes  | Intermediate  | No  | No   |
| <i>Fermentable</i>  | Highly   | Highly   | Yes   | Slowly  | No   |
| Examples            | FOS, GOS   | Resistant starch, inulin, pectin, guar gum   | Psyllium, oat fiber   | Wheat bran, lignin  | Cellulose, sterculia   |
| Chain length        | Short-chain  | Long-chain   | Long-chain  | Long-chain  | Long-chain   |
| Sources             | <ul style="list-style-type: none"> <li>• Legumes/pulses</li> <li>• Wheat, rye</li> <li>• Onions, garlic</li> </ul> | <ul style="list-style-type: none"> <li>• Legumes/pulses</li> <li>• Rye, barley</li> <li>• Firm bananas</li> <li>• Oats, buckwheat groats</li> <li>• Cooked and cooled pasta, rice, potato</li> </ul> | <ul style="list-style-type: none"> <li>• Seed of <i>Plantago ovata</i></li> <li>• Oats</li> </ul> | <ul style="list-style-type: none"> <li>• Some fruits and vegetables</li> <li>• Wheat bran</li> <li>• Whole-grain cereals</li> <li>• Brown rice, wholemeal pasta, quinoa</li> <li>• Linseed, flaxseed</li> </ul> | <ul style="list-style-type: none"> <li>• Whole grains and high-fiber cereals</li> <li>• Nuts, seeds</li> <li>• Skins of fruits and vegetables</li> </ul> |
| Laxative effect     | Weak   | Mild   | Good  | Good  | Good   |
| Transit time effect | None   | None   | Hastens transit time  | Hastens transit time  | Hastens transit time   |
| Prebiotic effects   | Stimulates growth of specific bacteria—e.g., bifidobacteria  | Increases overall bacterial abundance; not selective for bifidobacteria  | Increases overall bacterial abundance; no evidence for selective growth                           | Increases overall bacterial abundance; no evidence for selective growth   | No evidence for selective growth   |
| SCFA production     | Very rapidly fermented in terminal ileum and proximal colon to produce SCFA  | Rapidly fermented in proximal colon to produce SCFA  | Moderately fermented along length of colon to produce SCFA  | Slowly fermented along length of colon to produce SCFA  | Poorly fermented   |
| Gas production      | High   | Moderate   | Moderate  | Moderate–high   | Low  |

FOS, fructo-oligosaccharide; GOS, galacto-oligosaccharide; SCFA, short-chain fatty acid.

### 2.1.3 Interaction of dietary fibers with the gut microbiota

Ingested fiber may influence fecal microbiota profiles, cause changes in the complex gastrointestinal environment, and promote the growth of bacteria in general and potentially beneficial bacteria in particular [14–16]. Oligosaccharides, including fructo-oligosaccharides and galacto-oligosaccharides, inulin, and possibly other soluble fibers, are therefore regarded as prebiotics that may stimulate the preferential growth of lactobacilli, bifidobacteria, and other health-promoting bacteria in the colon [3,14–16]. The gut microbiota are thought to play a crucial role in human health and prevention of disease through a variety of mechanisms, including production of short-chain fatty acids (SCFA), which are important for maintaining gut homeostasis and optimal immune function [3,14–16]. Changes in the gut microenvironment have been associated with many common conditions, including irritable bowel syndrome, obesity, cardiovascular disease, and asthma [15,16]. The relationship between the gut microbiota, dietary fiber, and health outcomes is an area of rapidly growing interest, but well-controlled human trials are required in order to confirm the emerging links noted in animal and epidemiological studies [15,16].

## 2.2 Role of dietary fiber in gastrointestinal conditions

### 2.2.1 Constipation

- Increasing fiber in the diet has long been considered a first-line treatment for constipation [3,17,18].
- Increasing fluid in the diet has long been considered a first-line treatment for constipation [3,17,18]; the evidence to support this is mixed.
- Improvements in bowel movement frequency and consistency may be observed by gradually increasing dietary fiber (or by adding fiber supplements) to a target dose of 20–30 g of total dietary and/or supplementary fiber per day. Fiber should be introduced gradually into the diet over weeks rather than days, to allow the body to adjust [18,19].
- Stool frequency is thought to be improved by soluble fiber through an increase in stool bulk and weight, and by insoluble fiber through the acceleration of intestinal transit time; for both, however, and especially for insoluble fibers, high-quality evidence is lacking [14].
- The best evidence for fiber supplementation is for psyllium in the management of chronic constipation [18].
- Evidence for the efficacy of fiber is particularly lacking for individual constipation subtypes: metabolic, neurological, diet-related, myogenic, drug-related, and pelvic floor dysfunction [14].
- In patients with obstructive diseases of the intestine, a high-fiber diet should be avoided.
- Delayed colon transit or dyssynergic defecation may be present when patients experience marked worsening of their constipation-related symptoms with fiber [17,20,21].

**Conclusion:** A high-fiber diet may be protective against, and therapeutically useful in the treatment of, constipation. A gradual increase in fiber intake through diet and/or supplementation to 20–30 g/day with adequate fluid is recommended. Additionally, psyllium supplementation may be appropriate in the management of chronic constipation. The role of fiber in other forms of constipation is limited, and a high-fiber diet may exacerbate symptoms in some individuals.

### 2.2.2 Irritable bowel syndrome (IBS)

- The effect of fiber on IBS symptoms is variable and specific to fiber type [14,22,23].
- Soluble fiber supplements—including linseed, methylcellulose, partially hydrolyzed guar gum, and psyllium—have demonstrated therapeutic benefit in a number of clinical trials, particularly for patients with constipation-predominant IBS (IBS-C) [3,14,22,23].
- Highly fermentable fibers, such as oligosaccharides, inulin, and wheat bran fiber (by virtue of its oligosaccharide content) may contribute to increased gas production, thus exacerbating symptoms of bloating, flatus, and gastrointestinal discomfort in IBS [14,22,23].
- Reducing the intake of highly fermentable fibers, as part of a low FODMAP dietary approach (see section 3), is recommended for the management of IBS and provides symptomatic relief in approximately 75% of patients [14,22].

**Conclusion:** Highly fermentable fibers, including oligosaccharides and inulin, and also wheat bran may exacerbate symptoms of IBS. The best evidence indicates that reducing the intake of these fermentable fibers as part of a low FODMAP dietary approach (see section 3) is effective in managing symptoms in the majority of IBS patients. Conversely, soluble fiber supplements including psyllium, linseed, and methylcellulose may be of therapeutic benefit, particularly in IBS-C.

### 2.2.3 Inflammatory bowel disease (IBD)

- Evidence for a therapeutic effect of dietary fiber in IBD is lacking. However, due to the potential anti-inflammatory and immune-modulating effects of fiber, this warrants further investigation [24].
- Restriction of dietary fiber in IBD is unnecessary except in the case of significant intestinal stenosis [24].
- Reducing the intake of highly fermentable fibers, as part of a low FODMAP dietary approach, may be useful in symptomatic management of IBD patients who have coexisting IBS (see section 3) [25,26].

**Conclusion:** There is currently a paucity of evidence supporting a therapeutic role of dietary fiber in IBD. Further high-quality studies are therefore required. The intake of dietary fiber should not be restricted in IBD patients, except in the case of intestinal obstruction. IBD patients with coexisting IBS may benefit from reducing their intake of highly fermentable fibers as part of a low FODMAP dietary approach.

### 2.2.4 Diverticular disease

- Higher intakes of dietary fiber may be associated with a reduced risk of diverticular disease [27,28].
- It is not clear whether a high-fiber diet is beneficial in patients with diverticulosis, as its pathogenesis is probably multifactorial and complex. More studies are needed in order to evaluate the role of fiber in the pathogenesis and treatment of diverticular disease [27,28].
- A few poor-quality studies have suggested that fiber may improve symptoms associated with uncomplicated diverticulosis and decrease the risk of diverticulitis. However, high-quality evidence is lacking [27,28].

- During an episode of acute diverticulitis, a low-fiber diet is often recommended in order to minimize bowel irritation [27,28]. However, there is limited evidence to support this strategy.

**Conclusion:** Consumption of a high-fiber diet may be protective against the development of diverticular disease, and the risk of complications (diverticulitis) may be higher in those on a low-fiber diet. Short-term use of a low-fiber diet may be indicated in the case of diverticulitis. However, all of these recommendations are supported by limited evidence and expert opinion only.

### 2.2.5 Colorectal cancer

- Evidence that fiber decreases the risk of colorectal cancers is mixed, and further research is needed [29].
- A 2012 analysis from the European Prospective Investigation into Cancer and Nutrition (EPIC) study showed that total dietary fiber was inversely associated with colorectal cancer risk, with similar results for colon and rectal cancers. Dietary fiber sources from cereals, fruits, and vegetables were similarly associated with a reduced risk of colon cancer [30]. Only cereal fiber was associated with a decreased risk of rectal cancer [30].
- There is no evidence from randomized controlled trials (RCTs) to suggest that increased dietary fiber intake will reduce the incidence or recurrence of adenomatous polyps within a 2–8-year period [31]. Longer-term trials with higher dietary fiber levels are needed in order to evaluate this further [31].

**Conclusion:** Evidence from cohort studies generally indicates a protective effect of a high-fiber diet against colorectal cancer; however, it is not certain whether this relationship is based on cause and effect. Further high-quality studies are required in order to elucidate the relationship and identify potential mechanisms of action.

### 2.2.6 Clinical indications for a low-fiber diet

- Ingestion of low-fiber foods may help decrease diarrhea, gas, and bloating by slowing bowel movements and reducing colonic fermentation [32].
- Short-term use of a low-fiber diet (< 10 g/day) may be recommended for bowel cleansing purposes in the days prior to diagnostic procedures such as colonoscopy, colonography, and laparoscopic gynecological surgery. In comparison with traditional bowel preparation regimens (clear fluid diet with use of cathartic agents), studies have shown that a low-fiber diet approach may be better tolerated, have fewer side effects, and permit a reduction in the dosage of cathartic agents required without compromising the quality of bowel preparation [32,33].
- A low-fiber diet is often recommended temporarily after a flare-up of diverticulitis, Crohn's disease or ulcerative colitis, or following gastrointestinal surgery. However, more studies are required in order to clarify whether this is of any therapeutic benefit [32]. Despite this, short-term use poses little nutritional risk, especially if delivered under the guidance of a dietitian [32] and reintroduction of fiber occurs in the long term.
- Dietary advice regarding a low-fiber diet may include the following: avoiding nuts and seeds, using more refined breads and cereals, reducing the intake of fruits and vegetables where possible, and peeling fruits and vegetables when consumed [32].

- In some cases, fiber is not the only dietary factor to be considered when a low-fiber diet is recommended. Dietary advice for patients with bloating, pain, and other IBS-like symptoms may include avoidance of spicy foods, fatty foods, gut irritants (such as alcohol and caffeinated beverages), and individual foods that are poorly tolerated [19].

**Conclusion:** There is limited evidence to support the therapeutic use of a low-fiber diet in the context of gastrointestinal disease and surgery. However, this is common in clinical practice, and short-term use presents little nutritional risk. A low-fiber diet may be useful in the context of bowel preparation for diagnostic procedures and may improve patient satisfaction and compliance.

### 2.3 Fiber intake and recommendations

Targets for recommended dietary fiber intake vary globally (Table 6). However, guidelines typically recommend an intake of > 20 g/day [4,5]. Actual dietary fiber intake falls below recommendations in many countries worldwide, but it is notably higher in regions with predominantly plant-based diets such as sub-Saharan Africa (Fig. 1) [3–5].

**Table 6** Recommended and actual fiber intake in different countries

| Country/region |        | Recommended fiber intake (g/day) | Median intake (g/day) | Body issuing the requirement  |
|----------------|--------|----------------------------------|-----------------------|---|
| USA and Canada | Male   | 38                               | 16.5–19.4             | The USA and Canada jointly use the Institute of Medicine (IOM) report from the National Academy of Sciences |
|                | Female | 25                               | 12–15                 |   |
| France         | Male   | 30                               | 21                    | <i>Agence française de sécurité sanitaire des aliments</i> (French food safety agency)                      |
|                | Female | 25                               | 17                    |   |
| Germany        | Male   | 30                               | 24                    | <i>Deutsche Gesellschaft für Ernährung</i> (German Nutrition Society)                                       |
|                | Female | 30                               | 21                    |   |
| Japan          | Male   | 30                               | 17                    | Japanese Ministry of Health   |
|                | Female | 25                               | 17                    |   |
| UK             | Male   | 18*                              | 15.2                  | UK Department of Health   |
|                | Female | 18*                              | 12.6                  |   |
|                |        | >25                              |                       | WHO/FAO   |
|                |        | >20                              |                       |   |

\* Lower requirements due to use of the nonstarch polysaccharide (NSP) method. Source: Jones 2014 [4].

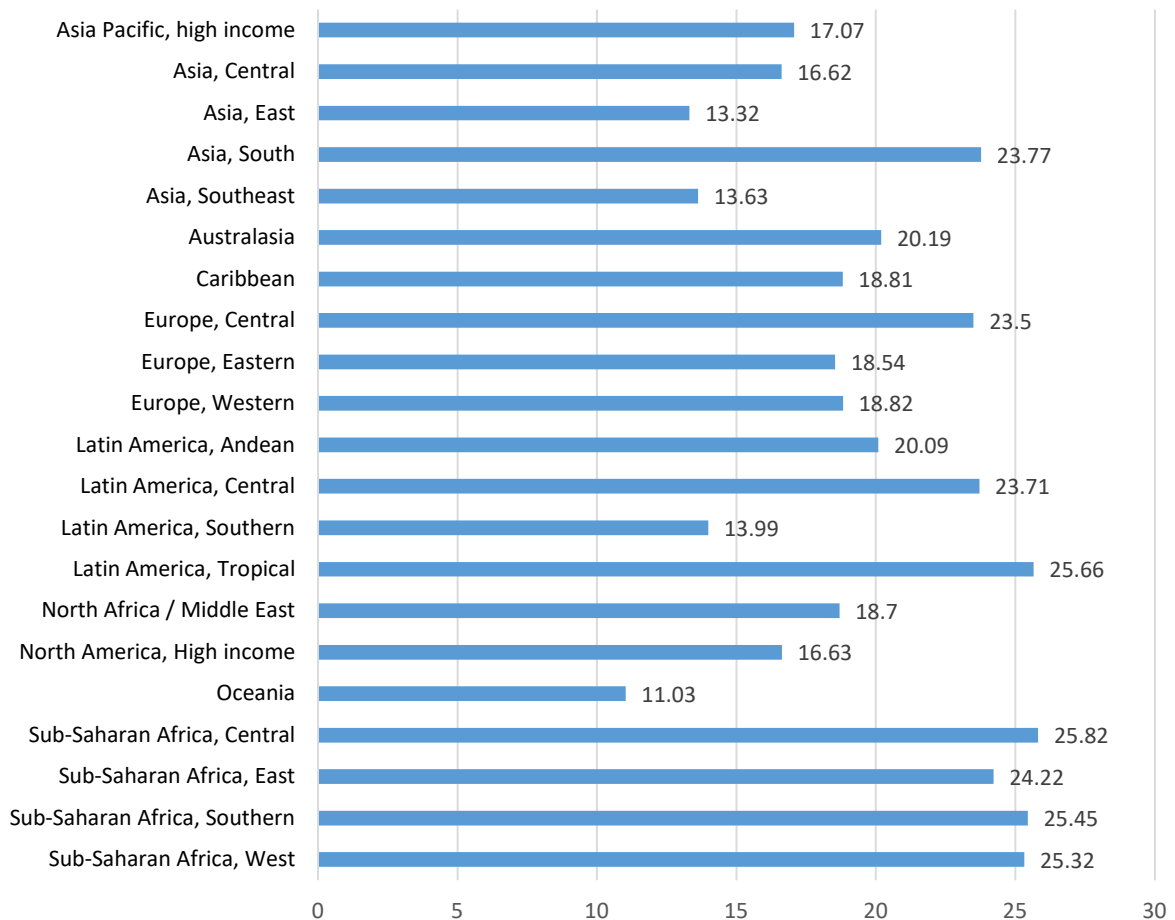


Fig. 1 Dietary intake of fiber by region (data from 2010). Source: Global Nutrition and Policy Consortium/Global Dietary Database [34].

## 2.4 Addressing the “fiber gap”

Adequate dietary fiber intake can be achieved by increasing variety in daily food patterns [5]. Eating at least 400 g or five portions of fruit and vegetables per day reduces the risk of chronic disease and helps ensure an adequate daily intake of dietary fiber [11,35]. Dietary messages about increasing consumption of high-fiber foods such as whole grains, legumes, fruit, and vegetables should be broadly supported by food and nutrition professionals [5,35].

Although consumers are also turning to fiber supplements and bulk laxatives as additional fiber sources, the best advice is to consume fiber in foods. Few fiber supplements have been studied for physiological effectiveness [5]. Increasing fiber in the diet too quickly can lead to symptoms such as gas, bloating, and abdominal cramping, so a gradual increase in intake should always be recommended [5].

Good sources of dietary fiber include: whole-grain products, fruit, vegetables, beans, peas and legumes, and nuts and seeds. Foods labeled “high in fiber” typically contain at least 5 g of fiber per serving. However, food-labeling requirements vary across countries [4,5].

Examples of common high-fiber foods include [36]:

- ½ cup red kidney beans, cooked (6.5 g fiber)
- ½ cup wheat bran cereal (9.1 g fiber)

- 1 cup butternut squash, baked (6.6 g fiber)
- 1 large orange (7.2 g fiber)
- 1 cup raspberries (8.0 g fiber)
- 1 cup whole-wheat spaghetti, cooked (5.9 g fiber)
- 1 cup broccoli, boiled (5.5 g fiber)

Consumers have an interest in increasing fiber intake, but compliance and cost pose a challenge. Dietary change requires alterations in long-term habits and is difficult to achieve, despite the reported benefits. Maintaining dietary change requires motivation, behavioral skills, and a supportive social and also political environment (Table 7) [5,37].

Many factors and complex interactions influence the evolution and shape of individual dietary patterns over time: income, food prices (the availability and affordability of healthy foods), individual preferences and beliefs, cultural traditions, as well as geographical, environmental, social, and economic factors [35].

**Table 7 Possible barriers to dietary change and solutions [35]**

#### **Barriers to dietary and lifestyle change**

- Misperception/lack of awareness of personal behavior
- Optimistic bias—the belief that healthy eating messages are targeted at people more vulnerable than ourselves
- Taste preferences as a barrier to healthier eating
- Giving up favorite foods
- Lack of knowledge on how to prepare food or cook
- Practical aspects: cost, preparation, and poor availability—many factors and complex interactions. Income, food prices (which will affect the availability and affordability of healthy foods), individual preferences and beliefs, cultural traditions, as well as geographical, environmental, social, and economic factors all interact in a complex manner to shape individual dietary patterns. Therefore, promoting a healthy food environment, including food systems that encourage a diversified, balanced, and healthy diet, requires involvement across multiple sectors and stakeholders, including government and the public and private sectors
- Lack of knowledge about the importance of healthy eating—a real but often unnoticed barrier to dietary change
- Difficulties in understanding food-packaging labels—portion size, knowledge about how to balance the diet
- Absence of an environment that facilitates lifestyle changes and complements nutrition messages and education initiatives

#### **Policy solutions for promoting and implementing a healthy diet**

- Promote a healthy food environment, including food systems that encourage a diversified, balanced, and healthy diet. This requires involvement across multiple sectors and stakeholders, including government, and the public and private sector
- Create coherence in national policies and investment plans, including trade, food, and agricultural policies, to promote a healthy diet and protect public health
- Encourage consumer demand for healthy foods and meals
- Promote appropriate infant and young child feeding practices
- Tax unhealthy foods (this has already been proposed for sugar in food)
- Encourage suppliers and vendors to support healthy diets

#### **Solutions for motivating individuals to adopt a balanced diet after experiencing health problems and for preventing lapses in diet**

- Individual education—e.g., promoting awareness of a healthy diet, help on how to exchange habitually bought items for healthier alternatives, encouraging children to adopt and maintain

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a healthy diet, encouraging culinary skills

- Provide nutrition and dietary counseling at primary health-care facilities
  - Promote, protect, and support breastfeeding in health services and the community, including through the WHO’s Baby-friendly Hospital Initiative ([www.who.int/nutrition/topics/bfhi/en/](http://www.who.int/nutrition/topics/bfhi/en/))
  - Provide behavioral therapy to equip individuals with skills that can help prevent lapses in diet
- 

## 2.5 Cascade guidelines—dietary fiber

Please see section 1.2 Cascades, Table 1.

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## 3 The low FODMAP diet

### 3.1 Introduction

#### 3.1.1 What is the low FODMAP diet?

The low FODMAP diet was developed by researchers at Monash University in Melbourne, Australia, to assist patients with irritable bowel syndrome (IBS) [38–40]. Research worldwide has now confirmed that the diet is effective in managing the symptoms of IBS [41–45].

“FODMAP” is an acronym that stands for: *fermentable oligosaccharides, disaccharides, monosaccharides, and polyols*.

The acronym groups together specific types of short-chain carbohydrates that are slowly absorbed or not digested in the small intestine. Because of their small molecular size, they increase the water content of the small intestine through an osmotic effect, and because they are readily fermented by bacteria, their delivery to the large bowel results in gas production [38,39,46]. FODMAPs can thus distend (or stretch) the bowel. In patients with IBS who are hypersensitive to this stretching, symptoms occur such as abdominal pain, bloating, excessive flatulence, and changes in bowel habits (constipation and/or diarrhea) [47]. Figure 2 classifies indigestible and slowly-absorbed carbohydrates according to their functional properties [48].

The low FODMAP diet includes reducing dietary intake of the five main subgroups of carbohydrates:

- Fructose in excess of glucose—e.g., honey, mango
- Lactose (when hypolactasia is present)—e.g., milk, yoghurt
- Sugar polyols (including sorbitol and mannitol)—e.g., avocado, mushrooms
- Fructans—e.g., wheat, onion, garlic
- Galacto-oligosaccharides (GOS)—e.g., legumes, soy milk



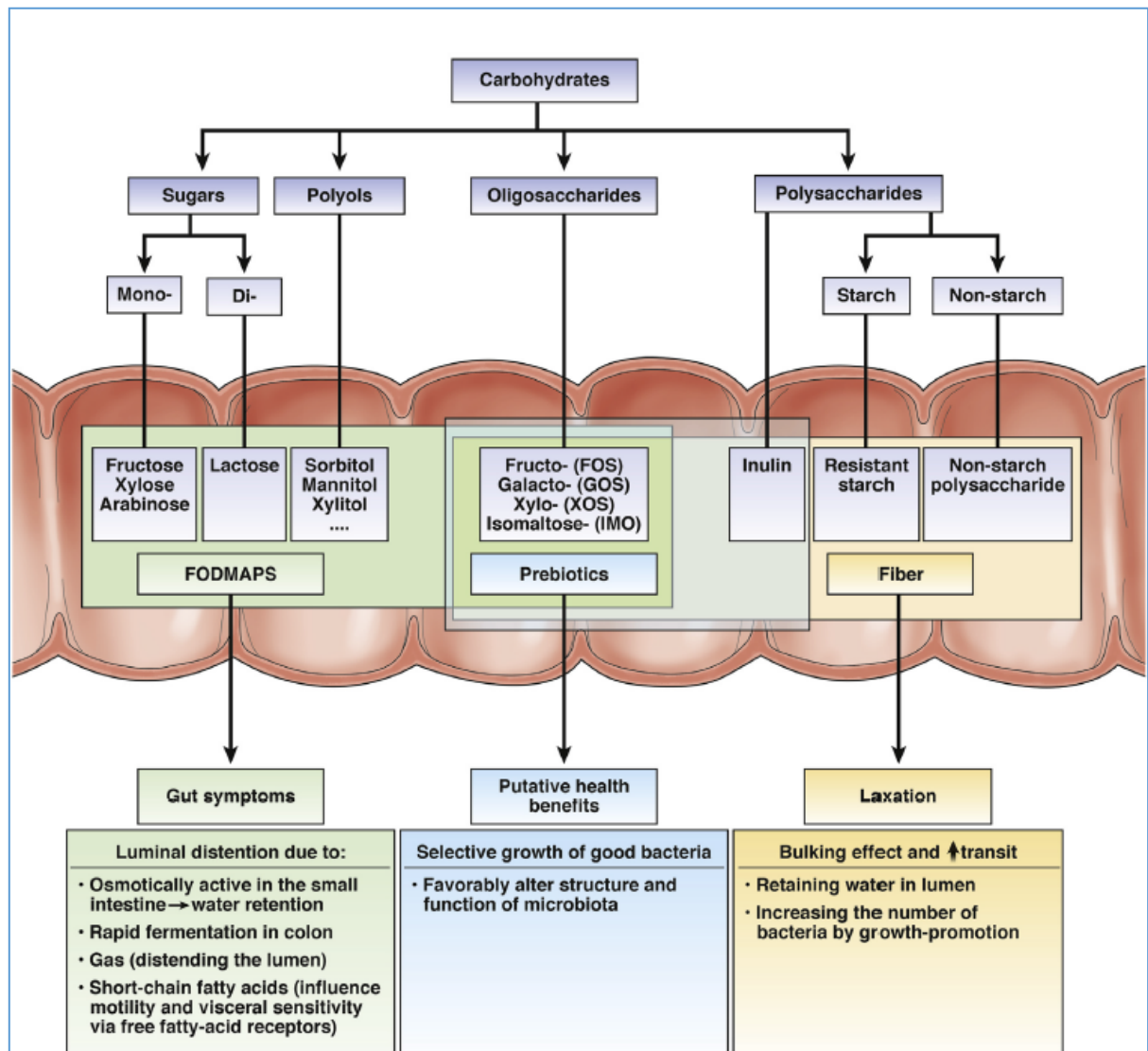


Fig. 2 Classification, according to their functional properties, of indigestible and slowly absorbed carbohydrates that are relevant in IBS. Source: Gibson et al. 2015 [48].

The aims of the low FODMAP diet are to help patients control their symptoms and subsequently to identify specific food triggers. This is done through an initial dietary reduction of all FODMAPs, followed by strategic re-challenges. Patients are then able to follow their own modified version of the diet. It is not suggested that the strict low FODMAP diet should be followed over the longer term.

The FODMAP content of a wide range of foods has been analyzed by the Department of Gastroenterology at Monash University, with findings published in a number of research articles [49–52]. The complete list of the FODMAP content of foods—classified as low, moderate and high—is available to the public through a smartphone application developed by the university [53].

### 3.1.2 How to implement a low FODMAP diet: reintroduction and problems/limitations

The low FODMAP diet is best implemented with the assistance of an experienced dietitian. Table 8 provides a clinical management overview, including the roles of the family physician

(general practitioner) or gastroenterologist and the dietitian. The diet can be implemented in a three-step process (Table 9).

**Table 8** Clinical management flowchart for using the low FODMAP diet

| Family physician (general practitioner) and/or gastroenterologist   |   |
|---|---|
| <ul style="list-style-type: none"> <li>• Medical investigation</li> <li>• Appropriate exclusion of other gastrointestinal disorders</li> <li>• A clinical diagnosis of IBS</li> </ul> | → Referral to dietitian   |
| Dietitian   |   |
|   | → Symptom improvement   |
|   | — Dietary re-challenge  |
|   | — Individual dietary triggers identified: “individual modified low FODMAP diet” established   |
| <ul style="list-style-type: none"> <li>• Assessment of usual diet and symptoms</li> <li>• Low FODMAP diet trial for 2–6 weeks</li> <li>• Assessment of dietary adherence</li> </ul>   | → Poor response   |
|   | — Reintroduce high FODMAP foods   |
|   | — Consider other triggers/therapies:  |
|   | <ul style="list-style-type: none"> <li>• Food chemicals</li> <li>• Stress</li> <li>• Small-intestinal bacterial overgrowth</li> </ul> |

Source: Tuck et al. 2014 [54].

First, patients should be identified as having functional bowel symptoms and should have other conditions such as celiac disease adequately excluded before their diet is changed. This is important, as dietary modifications may affect the accuracy of tests such as those for celiac disease. Patients should then be educated on ways of reducing high FODMAP foods in the diet, usually for a period of 2–6 weeks [55]. The primary aim of this initial phase is to improve symptom control. Patients are best educated by an experienced dietitian on the FODMAP content of foods, to ensure that they understand which foods to avoid—and importantly, which foods to include during the initial phase. No entire food groups should be excluded; instead, modifications should be made to the types of food chosen in each food group. For example, consumption of apples, which have a high FODMAP content, might be changed to intake of oranges, which have a low FODMAP content. This is important for maintaining nutritional adequacy [55]. Patients should understand the mechanisms of FODMAPs and the effect of dose so that they can grasp the dietary process.

The second phase is the re-challenge phase, the aim of which is to identify specific dietary triggers in each individual. It is unlikely that all high FODMAP foods cause symptoms for every individual, and strategic challenges are therefore used to identify tolerance levels for each FODMAP subgroup [56]. Guidance from a dietitian helps the patient test each FODMAP subgroup, including dose response, frequency of consumption, and the additive effect of multiple high FODMAP foods [56]. Individual tolerance for FODMAPs varies widely. Tolerance within an individual can also vary depending on other factors, including stress levels.

The final phase is the maintenance phase. The aim of the maintenance phase is for the patient to reintroduce as many high FODMAP foods back into the diet as tolerated, whilst still maintaining good symptom control. Any foods that are well tolerated should be reintroduced into the diet. Foods that are moderately tolerated may be reintroduced on an occasional basis, whilst foods that are poorly tolerated should continue to be avoided [56]. In the long term, patients are encouraged to continue to challenge themselves with poorly tolerated foods from time to time, in order to reassess their tolerance.

**Table 9** Three-step process for implementing the low FODMAP diet

|                |   |
|----------------|---|
| <b>Phase 1</b> | <ul style="list-style-type: none"> <li>— Initial assessment by a dietitian to evaluate symptoms and FODMAP intake</li> <li>— The dietitian provides education on the low FODMAP diet</li> </ul> |
| ↓              |   |
| <b>Phase 2</b> | <ul style="list-style-type: none"> <li>— The dietitian reviews the patient's response to dietary modification</li> <li>— FODMAP re-challenges are started</li> </ul>                            |
| ↓              |   |
| <b>Phase 3</b> | <ul style="list-style-type: none"> <li>— The dietitian reviews the response to FODMAP challenges</li> <li>— The challenges are interpreted</li> </ul>   |

- *List of high FODMAP foods and low FODMAP alternatives:* due to continuing and rapidly progressing research into the low FODMAP diet, many food lists that are available—both printed and on-line—are unfortunately outdated and hence have inaccuracies [57]. Table 10 lists rich sources of FODMAPs and suitable low FODMAP alternatives [58]. The Monash University smartphone application for the low FODMAP diet is the most useful tool, as it is regularly updated [53]. The app costs US\$ 10 with no other fees, including all updates.

**Table 10** Food sources of FODMAPs and suitable alternatives

| Food group         | Richest sources of FODMAPs  | Suitable alternatives   |
|--------------------|---|---|
| Fruit              | Apples, apricots, cherries, blackberries, boysenberries, mango, nashi pears, nectarines, peaches, pears, persimmon, plums, watermelon | Banana, blueberry, cantaloupe, grapefruit, grapes, lemon, lime, mandarin, orange, passion fruit, raspberry, rhubarb, strawberry       |
| Vegetables         | Artichokes, asparagus, cauliflower, garlic, mushrooms, onion, shallots, snow peas, spring onion (white part)                          | Carrot, chili, chives, cucumber, eggplant, ginger, green beans, lettuce, olives, parsnips, peppers, potato, spinach, tomato, zucchini |
| Protein sources    | Legumes/pulses  | All fresh beef, chicken, lamb, pork, veal   |
|                    | Pistachio nuts  | Macadamia, peanut, walnut, and pine nuts  |
|                    | Cashews   | Eggs, tempeh, tofu  |
| Breads and cereals | Wheat, rye, barley  | Buckwheat, corn, oats, polenta, quinoa, rice, spelt   |

| Food group | Richest sources of FODMAPs  | Suitable alternatives  |
|------------|---|--|
| Dairy      | Condensed or evaporated milk, cottage or ricotta cheese, custard, ice cream, milk, yogurt | Butter, lactose-free milk, lactose-free yogurt, other cheeses, rice milk |
| Other      | Honey, sorbitol or mannitol, high-fructose corn syrup, fructose                           | Golden syrup, maple syrup, regular sugar (sucrose), glucose              |

Source: Barrett 2013 [58].

- Potential adverse effects of the low FODMAP diet:* Any major change to dietary intake carries risks of unwanted effects such as reduced dietary fiber or an increased risk of nutritional inadequacy in general. The effect of dietary modification on quality of life must also be considered. Additionally, what is eaten is a major factor in defining the gastrointestinal microbiota [59]. There is therefore a potential that restricting FODMAPs, including prebiotic fructans and galacto-oligosaccharides, could have marked effects on the composition of the microbiota [60].

To date, there are only limited published data on the nutritional adequacy of patients' intake whilst on the short-term low FODMAP diet or in the long term following reintroduction of FODMAPs for tolerance. However, it is thought that with appropriate dietary counseling, the diet can be applied in a nutritionally adequate way. The effect of the diet on nutritional adequacy when self-implemented is not known [60]. Studies to date suggest that total energy, carbohydrate, and calcium intake may be reduced in the short term, although fiber intake does not appear to be altered. Due to the potential of the diet to cause alterations to nutritional intake, body weight and dietary intake should be monitored throughout the duration of the therapy [60].

A low FODMAP diet can potentially improve or worsen an individual's quality of life; however, most data suggest that the low FODMAP diet does not lead to a deterioration in quality of life and may even improve it [61]. A recently published placebo-controlled study in patients with IBS reported that a low FODMAP diet was associated with adequate symptom relief and significantly reduced symptom scores in comparison with a placebo [62].

Because of these potential detrimental effects of dietary modification, long-term adherence to the low FODMAP diet is recommended only for those who have severe symptoms and require ongoing restriction for symptom control. A program of reintroduction of high FODMAP-containing foods in order to identify the patient's tolerance threshold is therefore encouraged.

## 3.2 Gastrointestinal conditions and the low FODMAP diet

### 3.2.1 Indications for low FODMAP diet

Most of the evidence for the use of the low FODMAP diet is for patients with IBS. As mentioned above, it is important for patients to have received a clinical diagnosis of IBS, with exclusion of other diseases, before a low FODMAP diet is implemented.

There is a small amount of evidence regarding the use of the low FODMAP diet in patients with inflammatory bowel disease (IBD) (Crohn's disease and ulcerative colitis) [26,63]. In patients with IBD, it is common to have IBS-type symptoms. Use of the low FODMAP diet in

IBD is therefore targeted at controlling the IBS-type symptoms, rather than inflammation related to the disease itself. This may also apply to patients with celiac disease.

The use of the low FODMAP diet is now being investigated for other conditions such as endometriosis [64], infantile colic [65], functional dyspepsia, fibromyalgia [66], scleroderma, and chronic fatigue syndrome. However, the evidence for the use of the diet in these conditions is minimal and it is therefore not recommended for use in connection with them at this stage.

### 3.2.2 The low FODMAP diet, functional dyspepsia (FD), and IBS

There is considerable overlap of symptoms between IBS and functional dyspepsia (FD). Functional dyspepsia is characterized by symptoms of bloating, belching, epigastric pain, and discomfort. Many patients experience both FD and IBS simultaneously. Although data are limited for the effect of the low FODMAP diet specifically for FD, there are anecdotal reports that it can be used to manage symptoms [67]. Further data are required in order to assess the effects of the low FODMAP diet in patients with FD.

### 3.3 Cascade guidelines—FODMAP

Please refer to section 1.2 Cascades, Table 2.

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## 4 Carbohydrate intolerance

### 4.1 Lactose intolerance / lactase deficiency

#### 4.1.1 Definition of terms

- *Lactose*: A disaccharide that is commonly found in dairy products, with the highest concentrations in milk and yoghurt.
- *Lactase*: A brush-border enzyme required for cleaving lactose (a disaccharide) into the monosaccharides glucose and galactose.
- *Lactase deficiency*: brush-border lactase activity that is markedly reduced relative to the activity observed in infants.
- *Lactose malabsorption*: Occurs when a substantial amount of lactose is not absorbed in the small intestine.
- *Lactose intolerance*: Occurs when lactose malabsorption induces gastrointestinal symptoms [68].

#### 4.1.2 Lactose intolerance in perspective: when is it relevant?

The expression of lactase is down-regulated in approximately 65–75% of the human population after weaning. Lactose malabsorption is more prevalent in populations in Asia, South America, and Africa [69]. Lactase persistence (continued lactase production in adult life) is a genetically determined trait and occurs most frequently in European and some African, Middle Eastern, and southern Asian populations [70].

The rate at which lactase activity is lost varies depending on ethnicity. Chinese and Japanese lose 80–90% of lactase activity within 3–4 years of weaning, in comparison with 7 years after weaning in Jews and 18–20 years after weaning in Northern Europeans [71].

Secondary lactose intolerance can be caused by damage to the small intestine, as in untreated celiac disease or viral gastroenteritis. Secondary lactose intolerance is usually reversible once the primary condition has been treated [69].

As distinct from lactose intolerance, cow's milk allergy is an inflammatory response to milk proteins. There are overlapping symptoms between lactose intolerance and cow's milk allergy, and misdiagnosis is therefore possible. Cow's milk protein allergy occurs in 2–6% of infants and 0.1–0.5% of adults [72]. In addition to gastrointestinal symptoms, cow's milk protein allergy can lead to skin symptoms (erythema, pruritus) and respiratory system manifestations (wheezing, breathlessness), and even to anaphylaxis [72]. Due to the similarity of some symptoms, it is important for health professionals to be aware of the differences between the two. Cow's milk allergy can also be induced by dairy products with minimal lactose content (such as hard cheeses).

#### 4.1.3 Symptoms of lactose intolerance

Typical symptoms of lactose intolerance include abdominal pain, bloating, flatulence, diarrhea, and borborygmi. It may also result in nausea and vomiting, although these are less frequent [69].

In patients with common adult-type hypolactasia, the amount of ingested lactose required to produce symptoms varies from 12 to 18 g, or 8–12 ounces of milk. Ingestion of small to moderate amounts of lactose usually produces bloating, cramps, and flatulence, but not diarrhea. Ingestion of larger amounts of lactose, faster gastric emptying times, and faster intestinal transit times all contribute to more severe symptoms. Several factors determine the onset of symptoms of lactose intolerance, such as lactose content in the diet, gut transit time, fermentation capacity of the gut microbiome, visceral hypersensitivity [73], and (possibly) neuropsychological factors [74].

**Table 11** Clinical symptoms and pathophysiology of lactose intolerance

| Pathophysiology   | Clinical symptoms   |
|---|---|
| Excessive production of hydrogen and methane in the intestine                               | Bloating, distension of the abdomen, excessive flatulence, nausea, and abdominal pain (nonspecific in nature) |
| Excessive unabsorbed lactose with osmotically driven water, in excess of colonic absorption | Diarrhea in some patients   |

Source: WHO Handbook on Diet and the Gut, 2016 [74].

#### 4.1.4 How to diagnose: in resource-limited and well-resourced settings

The diagnosis of lactose intolerance is based on self-reporting of symptoms after lactose ingestion [54]. Determining the dose of lactose that persons with lactose intolerance can tolerate is critical in determining its implications for health [68]. The presence of malabsorption of lactose is commonly not associated with symptoms. It is only when lactose malabsorption induces symptoms that “lactose intolerance” can be diagnosed.



- *Lactose hydrogen breath testing.* Lactose hydrogen breath testing is currently considered to be the most cost-effective, noninvasive, and reliable measure of lactose malabsorption [69]. The breath test usually involves consumption of 25 g or 50 g of lactose, followed by measurement of breath hydrogen and methane over the following 3–4 hours. Although diagnostic guidelines vary, an increase in breath hydrogen by 20 ppm (parts per million) above baseline or in methane by 10 ppm above baseline suggests lactose malabsorption [69].
- *Lactose “tolerance” test [74].* This is a blood test for lactase deficiency, and the traditional term “tolerance” test is a misnomer. The patient consumes 50 g of lactose dissolved in water. Samples of capillary blood are obtained to test the plasma glucose concentration at –5, 0, 15, 30, 45, and 60 minutes. When lactose malabsorption is present, the blood sugar will not rise after lactose ingestion; normally, plasma glucose should increase by at least 1.4 mmol/L (25.2 mg/dL); its failure to do so indicates lactose malabsorption. The test’s sensitivity and specificity for lactase deficiency are high (both > 90%).
- *Genetic test [74].* The genetic test identifies single nucleotide polymorphisms associated with lactase persistence/nonpersistence. For example, genotype CC correlates with hypolactasia, while the TT genotype correlates with lactase persistence. Not everyone with the CC genotype will develop symptoms of lactose malabsorption. However, the relevant mutations depend on the ethnicity of the population studied.
- *Intestinal biopsy.* A jejunal biopsy can be used to assess lactase activity, but it is less sensitive and more invasive than the lactose breath test. This test is not recommended in clinical practice.
- *Food challenge.* A dietary challenge may be sufficient to identify lactose malabsorption in many cases. The food used for the challenge should be relevant to the individual’s needs and food preferences. A standard challenge involves consuming 1 cup (250 mL) of skimmed milk\* in one sitting, with individual monitoring of the symptom response. However, this should be adapted to the individual. For example, if a particular person rarely consumes this quantity of milk and would rather challenge with 200 g of yoghurt, then skimmed-milk yoghurt\* should be used for the food challenge [56].

\* Since some persons are intolerant of dairy fat (triacylglycerol).

#### 4.1.5 How to treat

There are two key ways of treating lactose intolerance: either through dietary avoidance of foods that contain significant amounts of lactose (Table 12), or by using  $\beta$ -galactosidase to hydrolyze the lactose content of foods.

- *Dietary modification—reduction of large quantities of lactose.* Most individuals with lactose intolerance can tolerate 12–15 g of lactose without gastrointestinal symptoms being triggered [68]. An average dairy-based meal contains approximately 12 g of lactose [75], hence small quantities of lactose are likely to be well tolerated even in those with lactose intolerance. Consideration of the dosage of lactose consumption is imperative for the management of lactose intolerance. Dairy products such as hard cheese, an excellent source of calcium, contain < 1 g of lactose and should therefore be included in the diet of those with lactose intolerance. Appropriate education is imperative to ensure adequate intake of calcium-rich foods.

Consideration should be given to reducing the intake of large quantities of lactose. This can be done by reducing the intake of products that are high in lactose, and/or by using lactose-free products. Dairy products such as cow's milk and yoghurt can be preincubated with  $\beta$ -galactosidase to hydrolyze the lactose content. There is an increasing demand for lactose-free products in some countries, resulting in the availability of lactose-free milk, yoghurt, cheese, cream and ice-cream. However, the need for products such as lactose-free cheese and cream is questionable, in view of their minimal lactose content [54].

**Table 12** Food items that are restricted or allowed in individuals with lactose intolerance

| Food items to be avoided  | Food items that are allowed  |
|---|--|
| <ul style="list-style-type: none"> <li>All kinds of milk: whole, low-fat, nonfat, cream, powdered, condensed, and evaporated</li> <li>Chocolate containing milk</li> <li>Cottage cheese, ice cream, creamy/cheesy sauces, cream cheeses, soft cheese, and mozzarella</li> <li>Milk and creamer</li> </ul> | <ul style="list-style-type: none"> <li>All fruits, vegetables, legumes</li> <li>All cereals</li> <li>All meat, fish, and eggs</li> <li>All vegetable fats</li> <li>Lactose-free milk and soy milk</li> <li>Hard cheeses and other lactose-free dairy products</li> <li>Yogurts, unless unfermented milk is added back in</li> <li>Kefir</li> </ul> |

Source: WGO Handbook on Diet and the Gut, 2016 [74].

- Enzyme supplementation.* An alternative treatment for lactose intolerance is supplementation with lactase ( $\beta$ -galactosidase) enzyme, which can be taken orally together with food. Studies have shown that this is effective in reducing breath hydrogen and in achieving symptomatic improvement [76–78], although higher doses of lactase, such as 50 g, appeared to overwhelm the enzyme's capacity [77].

## 4.2 Fructose intolerance

### 4.2.1 Definition of terms

- Fructose:* A monosaccharide commonly found in honey, mango, apple, pear, and high-fructose corn syrup.
- Excess fructose:* Fructose and glucose commonly coexist in foods; excess fructose is defined as the presence of fructose in excess of glucose (also termed “free fructose”). The absorption of excess fructose relies on low-capacity absorptive pathways that appear to be present along the length of the small intestine. In this situation, fructose molecules are likely to remain in the small-intestinal lumen for longer and exert their osmotic effects over much of its length, with or without “spill-over” into the colon (i.e., fructose malabsorption) [46,54]. The fructose that reaches the large intestine is then available for colonic fermentation, creating by-products of hydrogen and methane that can be measured in expired air.
- Fructose malabsorption:* Incomplete absorption of a given dose of fructose in the small intestine, resulting in “spill-over” into the large intestine. Fructose malabsorption has been shown to be a normal phenomenon, occurring in approximately 35% of healthy individuals [79].



- *Fructose intolerance*: This occurs when consumption of fructose induces gastrointestinal symptoms.

#### 4.2.2 Proposed mechanisms

- Fructose has been shown to have an osmotic effect, increasing luminal water content in the small intestine and leading to distension (stretching) of the intestinal wall. If this is marked, or if visceral hypersensitivity is present, it may result in abdominal pain, bloating, and sometimes diarrhea. This effect has been found to occur regardless of the degree of fructose absorption in the small intestine [46]. It is therefore not the presence of malabsorption, but rather the presence of visceral hypersensitivity such as that seen in functional gastrointestinal disorders, that is likely to cause the symptoms after fructose consumption [47].
- Breath hydrogen testing used to be a popular tool to help in targeting dietary therapy; it was thought that patients who had a negative fructose breath test did not require fructose restriction. However, symptoms may occur regardless of the presence of malabsorption based on breath hydrogen or methane production, due to the effects of fructose on the small intestine. Breath hydrogen testing is therefore no longer recommended for assessing fructose absorption [80].

#### 4.2.3 Intake of fructose

Estimates of fructose consumption suggest that total fructose intake has increased in recent years, largely due to the increased use of high-fructose corn syrups. A study in the United States that compared intake in the periods 1977–1978 and 1999–2004 found only a 1% increase in fructose consumption as a percentage of energy intake, in comparison with a 41% increase in total carbohydrate intake [74,81]—suggesting that the increase in fructose consumption is not as significant as thought. Fruit and fruit products were the main source of dietary fructose in 1999–2004 [81].

#### 4.2.4 Use of dietary modification

Early studies investigating the effect of excess fructose on gastrointestinal symptoms focused on fructose alone, or fructose in combination with sorbitol. However, these fructose-restricted diets were poorly described. Since excess fructose is often consumed together with other short-chain carbohydrates that have similar effects on the bowel (i.e., FODMAPs), it is the combined role of these specific carbohydrates in the pathogenesis of gastrointestinal symptoms, as opposed to their effects individually [49–51], that results in symptoms. The grouping of these fermentable carbohydrates as part of the low FODMAP diet has been associated with symptom improvement in up to three-quarters of patients with functional gastrointestinal disorders [40,41,44,82].

#### 4.2.5 Recommendations

- Fructose ingestion (in excess of glucose) is modified as a component of the low FODMAP diet. Reduction of all dietary FODMAPs, rather than fructose alone, has a more beneficial effect on reducing gastrointestinal symptoms.

- “Fructose malabsorption” is neither a diagnosis nor a condition. Rather, dietary fructose may trigger IBS-like gastrointestinal symptoms, and its effects should be considered along with the effects of the other dietary FODMAPs (see section 3).
- The clinical value of breath tests for identifying fructose malabsorption is limited.
- For food items that should be avoided, please refer to the FODMAP composition table (Table 10).

### 4.3 Sucrase–isomaltase deficiency

Sucrase–isomaltase deficiency (also known as sucrose intolerance) usually manifests early in life and can result in carbohydrate malabsorption, causing symptoms of diarrhea, bloating, and abdominal pain, similar to the symptoms of diarrhea-predominant IBS. The cause of sucrase–isomaltase deficiency is reduced small-intestinal activity of an enzyme known as glucosidase. The enzyme is normally involved in the digestion of starch and sugars. With reduced glucosidase activity, carbohydrates—particularly sucrose—then behave as FODMAPs, with increased osmotic activity and fermentation in the bowel, potentially leading to symptoms of IBS [83]. A role for sucrase–isomaltase deficiency in later-onset IBS is poorly established.

#### 4.3.1 Congenital sucrase–isomaltase deficiency

In congenital sucrase–isomaltase deficiency, mutations in the sucrase–isomaltase (*SI*) gene lead to severe symptoms. This is a rare condition. However, recent studies have identified multiple variations of the *SI* gene with reduced function. About 2–9% of persons of North American and European descent may be affected, suggesting that it is possibly an under-recognized condition [84]. However, it has yet to be shown whether it is pathogenetically involved with symptom induction in patients with functional gastrointestinal disorders.

#### 4.3.2 Secondary or acquired sucrase–isomaltase deficiency

Secondary or acquired sucrase–isomaltase deficiency can also theoretically occur, but it is usually transient. Animal studies have shown that villous atrophy, such as that occurring in untreated celiac disease, may result in sucrase–isomaltase deficiency. This should be reversible with healing of the villous atrophy [84].

#### 4.3.3 Diagnosis

A diagnosis of sucrase–isomaltase deficiency can be established using duodenal or jejunal biopsies in children, for assessment of sucrase, lactase, isomaltase, and maltase activity [84]. However, the biopsy samples must be immediately frozen, and due to the complex freeze/thaw process required for sample analysis, inaccurate results may occur [85]. Other methods of diagnosis are available, such as sucrose breath testing, but performing hydrogen breath testing in young children is problematic [85]. More recently, genetic sequencing has become available to identify forms of congenital sucrase–isomaltase deficiency [84], although these results should be considered in combination with the clinical picture. There are few data on the value of performing such tests in adults.

#### 4.3.4 Treatment

Limited evidence is available for the treatment of sucrase–isomaltase deficiency. Treatment options include dietary restriction of sugars and starch, although this has been poorly studied. Patients undertake an initial restrictive phase, followed by gradual reintroduction in order to determine tolerance. However, such dietary restrictions are difficult, and patients are often noncompliant [85]. An alternative to dietary modification is enzyme replacement with sacrosidase, which has shown good effect in studies with small sample sizes [85,86]. However, enzyme supplementation is costly and may not be available worldwide.

#### 4.4 Cascade guidelines

Please refer to section 1.2 Cascades, Table 3.

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## 5 Other dietary interventions in IBS

Certain food items trigger the symptoms experienced by IBS patients, including foods that are rich in FODMAPs. Although many IBS patients believe that they are intolerant of certain foods [87], this often cannot be reproduced on a blinded re-challenge with the offending foodstuff [88,89]. IBS patients often institute dietary changes themselves, in an attempt to alleviate symptoms [88,90].

Safe, reliable, and affordable tests for the diagnosis of food intolerance are lacking. Clinicians cannot therefore easily confirm the offending food component(s) in patients who report food-induced symptoms [91]. General principles are as follows:

- Celiac disease should be considered, especially in patients with persistent symptoms of gas, bloating, and diarrhea, and in patients with a positive family history [91]. Symptoms of IBS are common in patients with celiac disease.
- Commercial tests that falsely claim to diagnose food intolerance on the basis of analyses of blood, stool, hair, or fingernails should be avoided [91].
- Lactose, fructose, excess fiber, resistant starch, fructans, and galacto-oligosaccharides may exacerbate/induce IBS symptoms. One approach in the absence of a dietitian is to consider individual elimination trials to improve symptoms [91].
- A low FODMAP diet should be considered for patients in whom individual dietary interventions have failed, or who have ready access to a dietitian with expertise. Dietary education in most studies demonstrating efficacy for this diet has been dietitian-led (or nurse-led). Four weeks of adherence is sufficient to determine the benefit of the diet. The diet is easily taught by experienced practitioners, the important reintroduction phase can be supervised, and additional attention to maintaining nutritional adequacy is provided.
- Dietary changes will disturb the gut microbiome; little is known about the clinical significance of changes to the microbiome associated with any IBS-specific diet [91]. Limited data suggest that there are no significant changes in the microbiome during the reintroduction phase of the low FODMAP diet [92].
- If the low FODMAP diet is unhelpful, it should be discontinued. Introduction of other dietary restrictions should be associated with careful consideration of nutritional adequacy, particularly if they are being added to current dietary restrictions.

Among other dietary approaches, few have good-quality evidence of efficacy, safety, and nutritional adequacy. The exception is the *gluten-free diet* (GFD), which is widely initiated by IBS sufferers in the United States without any input from health-care professionals.

Reference may also be made to the following WGO Global Guidelines [93]:

- *Irritable Bowel Syndrome: a Global Perspective* (2015), for clinical recommendations on IBS diagnosis and management, including WGO cascade options—since neither the epidemiology nor the clinical presentation of the condition, nor the availability of diagnostic and therapeutic resources, are sufficiently uniform throughout the world to support the provision of a single, gold-standard approach [94].
- *WGO Global Guidelines: Celiac Disease* (2016) [1]. The 2017 update of this WGO Global Guideline has been published in the *Journal of Clinical Gastroenterology* [2].

Several uncontrolled studies have shown that a proportion of patients who meet the criteria for IBS will respond to a GFD [95–98]. The controversy lies in whether the offending food components are gluten, nongluten wheat proteins, or fructans. There is a cohort of patients with IBS or other functional gut symptoms, often with extraintestinal symptoms, who self-report that they are gluten-sensitive. However, gluten has yet to be implicated as the causative molecule in such patients. A subgroup who have an increased density of intraepithelial lymphocytes and eosinophils in the small-bowel and often large-bowel mucosae have been shown to develop gastrointestinal symptoms after double-blind placebo-controlled challenges to wheat and other proteins [99]. Dietary restriction guided by the results of such challenges has led to long-term symptomatic benefits in these patients [100]. Further research in other centers is required in order to assess the generalizability of these findings.

In the majority of individuals who do not have the above-mentioned histopathological changes, GFD may be effective, but whether the patient needs to be gluten-free or whether gluten is a marker for other molecules contained in wheat, such as fructans, remains controversial. A recent study in Norway provided evidence that fructans, but not gluten or wheat protein, were the culprits in patients with self-reported gluten sensitivity [101].

- So-called “nonceliac” gluten sensitivity (NCGS) is likely to represent a heterogeneous group of patients, often with IBS, and may include a proportion with non-immunoglobulin E-mediated wheat protein sensitivity. The majority of these patients will have no evidence that they are intolerant of gluten itself—as consistently shown in RCTs using double-blind placebo-controlled cross-over food challenges [102].
- There are currently no biomarkers of gluten sensitivity. Determining HLA-DQ2/8 as a predictive marker for wheat sensitivity cannot be recommended [97].
- In the absence of biomarkers for NCGS, a double-blind placebo-controlled trial (DBPCT) to assess gluten-induced symptoms has been considered in clinical practice to be the best method of detecting NCGS after excluding celiac disease and wheat allergy. This approach has been utilized in some areas of Europe, but has been seldom used in the rest of the world. However, a recent European study [103] revealed the limitations of this test, suggesting that gluten is not the source of these patients’ symptoms, that a DBPCT is inadequate to determine whether gluten is causing the symptoms, and that the DBPCT is not a satisfactory method of detecting gluten-induced symptoms in individual patients without celiac disease. However, a standardized and rigorous methodology for assessing NCGS, once developed, may have merit.

- Many patients who suffer from IBS-like symptoms self-report “gluten sensitivity,” and their symptoms may improve on a gluten-free diet [98]. According to a recently published study in Norway [101], this improvement is probably due to a reduction in fructans, rather than gluten or nongluten wheat proteins.
- The benefits of a low FODMAP diet may outweigh the benefits of a GFD [91].
- The ultimate role of diet in different IBS subtypes needs further research [87].

Despite uncertainty regarding the role of gluten, specifically, in the genesis of symptoms in IBS, a trial of GFD is a reasonable intervention for people who feel that their symptoms become worse with gluten-containing foods.

- IBS patients with pain or bloating as the predominant symptom [89] and patients with IBS-D or mixed IBS may benefit most.
- A GFD is difficult to implement and maintain, and it is not inexpensive [104].
- The involvement of a trained registered dietitian is recommended.

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## 6 Appendix

### 6.1 Abbreviations

**Table 13** Abbreviations used in this WGO guideline

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|        |   |
|--------|---|
| DBPCT  | double-blind placebo-controlled trial                                     |
| FAO    | Food and Agriculture Organization   |
| FD     | functional dyspepsia  |
| FODMAP | fermentable oligosaccharides, disaccharides, monosaccharides, and polyols |
| FOS    | fructo-oligosaccharide  |
| GFD    | gluten-free diet  |
| GOS    | galacto-oligosaccharide   |
| IBD    | inflammatory bowel disease  |
| IBS    | irritable bowel syndrome  |
| IBS-C  | constipation-predominant irritable bowel syndrome                         |
| IBS-D  | diarrhea-predominant irritable bowel syndrome                             |
| LDL    | low-density lipoprotein   |
| NCGS   | nonceliac gluten sensitivity  |
| NSP    | nonstarch polysaccharide  |
| RCT    | randomized controlled trial   |
| SCFA   | short-chain fatty acid  |
| WA     | wheat allergy   |
| WDHD   | World Digestive Health Day  |
| WGO    | World Gastroenterology Organisation                                       |
| WHO    | World Health Organization   |

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## 6.2 Organizations publishing relevant guidelines

- World Health Organization (WHO) guidelines on nutrition [105]  
[www.who.int/publications/guidelines/nutrition/en/](http://www.who.int/publications/guidelines/nutrition/en/)
- American College of Gastroenterology (ACG) guidelines (e.g., nutrition therapy in the adult hospitalized patient) [106]  
<https://doi.org/10.1038/ajg.2016.28>
- British Society of Gastroenterology (BSG) guidelines (e.g., celiac disease) [107]  
<https://www.bsg.org.uk/clinical/bsg-guidelines.html>  
<http://gut.bmj.com/content/63/8/1210>
- National Institute for Health and Care Excellence (NICE) guidelines (e.g., diet, nutrition and obesity) [108]  
[www.nice.org.uk/sharedlearning/lifestyle-and-wellbeing/diet--nutrition-and-obesity](http://www.nice.org.uk/sharedlearning/lifestyle-and-wellbeing/diet--nutrition-and-obesity)
- North American/European Societies for Pediatric Gastroenterology, Hepatology, and Nutrition (NASPGHAN/ESPGHAN) guidelines (for children) [109]  
[www.naspghan.org/content/55/en/Nutrition-and-Obesity](http://www.naspghan.org/content/55/en/Nutrition-and-Obesity)

World Gastroenterology Organisation (WGO) guideline: Coping with common gastrointestinal symptoms (Hunt et al., 2014) [19]  
[https://journals.lww.com/jcge/Fulltext/2014/08000/Coping\\_With\\_Common\\_Gastrointestinal\\_Symptoms\\_in.4.aspx](https://journals.lww.com/jcge/Fulltext/2014/08000/Coping_With_Common_Gastrointestinal_Symptoms_in.4.aspx)  
<http://www.worldgastroenterology.org/guidelines/global-guidelines/common-gi-symptoms>

- World Gastroenterology Organisation (WGO) guideline on celiac disease (Bai and Ciacci, 2017) [2]  
<http://www.worldgastroenterology.org/guidelines/global-guidelines/celiac-disease>
- British Dietetic Association (BDA): Dietary management of irritable bowel syndrome in adults (McKenzie et al., 2016) [22]  
<https://doi.org/10.1111/jhn.12385>

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