Diet Interventions for Irritable Bowel Syndrome

Separating the Wheat from the Chafe

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KEYWORDS

• Irritable bowel syndrome • Diet therapy • Nutrition • Gluten • Carbohydrates
• Histamine • Low FODMAP diet • Sucrase-isomaltase deficiency

KEY POINTS

- As a heterogeneous condition, irritable bowel syndrome (IBS)-based diet therapy requires individualization.
- Consider IBS masqueraders or overlapping conditions that may prompt food intolerance in patients with IBS who are unresponsive to traditional therapies.
- The mechanisms responsible for food-related symptom induction in patients with IBS is multifactorial and, as yet, incompletely defined.

INTRODUCTION

Individuals living with irritable bowel syndrome (IBS) have frequently identified food as a trigger to their digestive distress. In one survey study of nearly 200 patients with IBS, 84% perceived that eating any food would induce gastrointestinal (GI) symptoms. Carbohydrates were the most common food group identified as problematic in 70% of those surveyed, including dairy products (49%), beans/lentils (36%), apple (28%), flour (24%), and plum (23%), all potential fermentable, oligo-,di-mono-saccharide and polyol (FODMAP) carbohydrate sources. Interestingly, 58% of this cohort identified foods rich in histamine, such as wine/beer (31%), salami (22%), and cheese (20%), as prompting symptoms.[1]

Food can impact a variety of physiologic factors that are relevant to the pathogenesis of IBS, such as motility, visceral sensation, brain–gut interactions, microbiome, gut permeability, immune activation, and neuro-endocrine function.[2] To comprehend the intricacy of the relationship between food and gut symptoms in patients with IBS, it

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is imperative to understand that food, in and of itself, is complex. Different components in a food can vary depending on a number of factors, including the ripeness and processing of food. These variables can impact the role of food intolerance. To further elucidate this concept, mechanically processing or separating fibers from grains will alter the particle size of the fiber. Larger particle size in insoluble fibers has been shown to enhance colonic mechanical irritation and laxation.[3] Ripeness can alter the nature of the carbohydrates in food; for example, unripe bananas are rich in resistant starch, whereas some varieties when ripened have greater amounts of fructans, a source of rapidly fermentable oligosaccharides, known to prompt GI distress in patients with IBS.[4,5]

Given that IBS is a heterogeneous condition and that the symptomology present in patients with IBS is common in other GI disorders, one needs to consider other potential overlapping or alternative diagnoses that may incite food intolerance, such as the connection between celiac disease and gluten or bile acid diarrhea and a high-fat diet. Gluten, a protein found in wheat, barley, and rye has been clearly identified as the trigger of immune activation and inflammation in celiac disease, but gluten's role in patients with IBS is less clear. Celiac disease occurs in approximately 1% of the population, but those who present with IBS-like symptoms appear to have an increased risk. A systematic review and meta-analysis showed a higher pooled prevalence of biopsy-proven celiac disease across all subtypes of IBS.[6] Moreover, more than 1/3 of Rome IV IBS-diarrhea or functional diarrhea have been shown to be experiencing primary bile acid diarrhea.[7] Once considered solely a pediatric condition, sucrase-isomaltase deficiency (SID) may be an IBS-D mimicker in adults. Adults presenting with genetic mutations related to congenital SID can present with IBS-D symptomology and have a greater likelihood of not responding to a low FODMAP diet (LFD).[8]

THE COMPLEXITY OF WHEAT INTOLERANCE IN IRRITABLE BOWEL SYNDROME

Wheat, a type of grass plant, is second only to rice as the key food crop consumed by humans. The physiologic effects of wheat bran can be split into nutritional effects, mechanical effects (on the GI tract) and antioxidant effects via phytonutrients in wheat.[9]

Whole wheat provides a rich source of carbohydrates, including insoluble fiber (wheat bran) and soluble fibers such as rapidly fermentable fructans. The various components in wheat have the potential to elicit different food intolerance symptom responses in those with IBS.[10]

Wheat-based fructans have been identified as part of the short-chain, rapidly fermentable carbohydrate family, coined FODMAPs. FODMAPs are found in many everyday foods and can prompt GI symptoms via their osmotic or fermentation effects in the gut.[11] Gluten, amylose trypsin inhibitors (ATIs), and other proteins present in grains, including wheat, barley, and rye, also may incite GI symptoms. ATIs found in higher levels in wheat and other gluten-containing cereals activate an innate immune response in mice and increase intestinal inflammation.[12] The potential role of ATIs in humans with IBS remains to be explored.

The use of novel confocal laser endomicroscopy (CLE), an endoscopic imaging tool that enables a high-resolution assessment of GI mucosal histology at a cellular and subcellular level, allowing identification of changes such as increased intraepithelial immune cells, epithelial leaks/gaps, and widened intervillous spaces that can be observed in real time, has revealed interesting data in patients with IBS. CLE research has uncovered that an atypical allergy to wheat and to a lesser degree with yeast, may be present in some individuals with IBS. Fritscher-Ravens and colleagues[13] used CLE to assess food sensitivity in patients with IBS and found a significant proportion

reacted to wheat and then felt better on a wheat-free diet. Seventy-six (70%) of 109 patients with IBS reacted to 1 of 5 tested food antigen mixtures (CLE+) delivered to the duodenal mucosa: wheat (61%), yeast (20%), milk (9.2%), soy (6.6%), or egg white (4%). CLE+ patients experienced immediate changes in tight junction proteins including increases in expression of claudin-2, decreases in occludin, as well as increased eosinophil degranulation. The researchers postulate these CLE+ patients with IBS have a form of non–immunoglobulin (Ig)E-mediated atypical food allergy that involves enhanced eosinophil and intraepithelial lymphocyte activation. When food antigens that elicited a positive CLE response were excluded from the diet, patients demonstrated a 70% average improvement in Francis IBS severity score after 3 months and a 76% improvement at 6 months. Impressively, 68% of CLE+ patients showed at least an 80% improvement in symptoms, whereas only 4% did not respond at all.[13] This innovative research using CLE highlights that some patients with IBS may be experiencing epithelial dysfunction due to atypical food allergy. Two recent publications of CLE in patients with IBS suggest the potential use of this technology to identify atypical food allergy and guide specific food elimination diets.[13,14]

When all of these data are taken into consideration, it is clear that wheat is complex and contains several compounds that could produce a symptomatic response in patients with IBS, see **Table 1**.

GLUTEN INTOLERANCE IN IRRITABLE BOWEL SYNDROME

Both the popularity of following a gluten-free diet (GFD) and the market for gluten-free products have increased at astounding rates. Kim and colleagues[17] analyzed data from the National Health and Nutrition Examination Surveys (NHANES) 2009 to 2014 and found the prevalence of celiac disease remained steady while the self-reported adherence to a GFD among individuals without celiac disease increased over the same period. Based on their findings, approximately 2.7 million people adhere to a GFD without a celiac disease diagnosis. Self-reported wheat sensitivity is associated with functional gastrointestinal disorders, including IBS.[18] It appears that many individuals are gravitating toward a GFD because they believe it is healthier for them or that it could lessen their GI symptoms. It should be noted there is no evidence that a GFD is healthier than a gluten-containing diet. In fact, many gluten-free products, such as crackers, breads, and snack foods, are devoid of the enrichment of key nutrients, such as iron, thiamin, riboflavin, and folic acid found in their enriched wheat-based counterparts.[19]

The data supporting a GFD for those with IBS are lacking, yet there may be benefit in an unclear proportion of patients. A 2018 systematic review and meta-analysis of randomized controlled trials (RCTs) assessing the impact of a GFD or LFD in improving IBS symptoms found insufficient evidence to recommend a GFD to reduce IBS symptoms.[20] There were only 2 RCTs looking at a GFD that met the inclusion criteria for this meta-analysis. Participants in both trials who had their diets spiked with gluten had increased IBS symptoms compared with those who remained on a GFD. Each study had separately reported a statistically significant result, but the significance was lost when results were pooled due to the marked heterogeneity between individual trial results.[20] A recent prospective study demonstrated patients with IBS who tested positive for deamidated antigliadin antibodies (AGA+), IgG and IgA, experienced greater GI symptom improvement (75%) on a GFD than those who were without the antibodies (AGA−) (38%). After adhering to a GFD for 4 weeks, GI symptoms improved overall in AGA+ patients with IBS, specifically constipation (P = .01), diarrhea

Table 1
The complexity of wheat and GI symptom induction in IBS

Component of Wheat	Definition	Pathomechanism for GI Symptom Induction in IBS
Gluten	A family of proteins found in wheat, as well as other grains including rye and barley. Gluten is composed of gliadins and glutenins in wheat, secalins in rye and hordeins in barley.	Alters bowel barrier function and/or leads to increased stool frequency, particularly in HLA-DQ2/8–positive patients.[15–17]
Fructan	A polymer of fructose molecules found in wheat, as well as other grains including rye and barely.	Microbial fermentation of fructans gives rise to gas in the colon, contributing to luminal distention. Although the gas production is normal, those with IBS may experience an exaggerated symptom response of discomfort, pain, bloating due to visceral hypersensitivity.[11]
Amylase trypsin inhibitors	Proteins found in wheat as well as other grains including rye and barely.	Increases intestinal inflammation via activating toll-like receptor 4 (TLR4) on myeloid cells in the intestine of mice.[15] Studies lacking in humans.
Wheat-germ agglutinin	A protein found in wheat.	May initiate an inflammatory immune reaction in the gut.[16]
Wheat bran	An insoluble fiber found in wheat.	Large particle wheat bran mechanically stimulates/irritates the gut mucosa which increases fecal mass and colonic transit rate. Fermentable fiber. Shown to be ineffective at normalizing bowel habits in patients with IBS as well as increase bloating, gas, and pain.[3]
Wheat protein	Four classes of proteins found in wheat (albumin, globulin, gliadin, glutenin).	IGE allergy or atypical allergy noted via CLE in patients with IBS.[13,14]

Abbreviations: CLE, confocal laser endomicroscopy; GI, gastrointestinal; IBS, irritable bowel syndrome; IGE, Immunoglobulin E.

(P = .001), and abdominal pain (P<.001), whereas AGA− patients with IBS experienced improvements only in abdominal pain (P = .01). There were limitations to this trial, however, as it was a small study and participants were not blinded to the GFD intervention.[21] Larger studies are needed to validate the usefulness of AGA as a potential marker to identify a subgroup of patients with IBS who may benefit from a GFD.

IS IT GLUTEN OR FRUCTAN INTOLERANCE?

When one goes on a GFD, eliminating wheat, rye, and barely, gluten is not the only food component that is removed from the diet. Fructans, also present in these grains, are reduced on a GFD and thus may be responsible for symptom improvement. A double-blind, placebo-controlled (DBPC) study in participants with self-reported gluten sensitivity found fructans were more likely to induce symptoms than gluten. IBS symptom scores, both overall and for bloating, were higher with fructans compared with gluten. Surprisingly, only 13 of 59 participants had their highest symptom score after the gluten challenge, whereas 27 participants had their lowest symptom score after the gluten challenge.[22] These data support that fructans are a more likely culprit than gluten. Research by Biesiekierski and colleagues[23] further substantiates that the presence of fructans in wheat drives IBS symptoms and not gluten. In a placebo-controlled, crossover rechallenge study, patients who habitually consumed a GFD diet experienced symptom relief with FODMAP reduction and did not have specific or dose-dependent reactions with gluten challenges. In this same cohort of patients, elevated biomarkers of intestinal injury were found in patients on a self-selected GFD and improved once they reduced their FODMAP intake. In addition, the reintroduction of gluten at various dosages did not negatively influence markers of intestinal epithelial injury and barrier function.[24]

THE POWER OF THE PLACEBO AND NOCEBO EFFECT

The placebo and nocebo effect cannot be overlooked, especially in a patient population such as IBS that has demonstrated high rates of the placebo effect, ranging from 16.0% to 71.4%, with a pooled placebo response of 40.2%.[25] On social media and in alternative medicine clinics, gluten is often promoted as inflammatory or toxic; this perception could play a role in the nocebo response often found in clinical trials involving patients with IBS who believe they are gluten sensitive. An eye-opening systematic review by Molina-Infante and Carroccio[26] found 80% of patients with suspected non-celiac gluten sensitivity cannot be diagnosed formally after a DBPC crossover gluten challenge and revealed that 40% of patients undergoing a DBPC challenge showed a nocebo response.

LEVERAGING BIOMARKERS TO PREDICT RESPONSE TO DIET IN IRRITABLE BOWEL SYNDROME

Using biomarkers to help predict those who might benefit from the LFD is highly desirable, as this would reduce the overuse of a complex elimination diet while selecting patients who are the most likely to respond to this diet intervention.

A potentially promising area for the development of biomarkers is the gut microbiome and/or metabolome. An exciting exploratory study found fecal volatile organic compounds (VOC) profiling was able to predict response to an LFD and a probiotic intervention.

Rossi and colleagues[27] found 15 VOC profile features that classified response to an LFD with a mean accuracy of 97% (95% confidence interval [CI] 96%–99%). This

study highlighted a noninvasive, cost-effective tool may be a promising method to help predict response to diet interventions in those with IBS. In addition, several studies have identified characteristics of the microbiome or metabolome that predict a greater likelihood of response to the LFD. Children who responded to the LFD were at baseline enriched in *Bacteroides,* Ruminococcaceae*, Faecalibacterium prausnitzii*, taxa with known saccharolytic metabolic capacity.[28] Adult patients with IBS demonstrated distinct differences in bacterial DNA profiles between responders and nonresponders to the LFD.[29] Bennet and colleagues[30] demonstrated fecal bacterial profiles from stools collected before starting the LFD were able to discriminate responders from nonresponders.

Food sensitivity testing using IgG antibodies to various foods in patients with IBS lacks proper validation in methodologically rigorous RCTs.[31,32] Despite their lack of validation, food sensitivity tests are quite popular, often recommended by functional practitioners, and can be costly to patients with limited coverage via health insurance. Several major medical organizations including the American Academy of Allergy, Asthma & Immunology, the Canadian Society of Allergy and Clinical Immunology, and the European Academy of Allergy and Clinical Immunology recommend against using IgG testing to diagnose food allergies or food intolerances/sensitivities due to the lack of evidence to support their use.[33] Furthermore, food-specific serum IgG levels have been proposed to reflect exposure to food components versus an intolerance or hypersensitivity.[34] Patients often present to clinic with questions regarding food sensitivity testing or with results from a test they have previously taken. It is important to have a thorough discussion with patients regarding the lack of evidence behind their use and the risks of "false positives," which ultimately lead to unnecessary food avoidance and escalation of food fears. Studies on IgG-based food sensitivity testing are ongoing and will hopefully shed light on whether these tests offer any benefit to patients with IBS.

An RCT of 58 patients with IBS found that a 4-week exclusion diet guided by leukocyte activation testing (LAT) led to significant global improvement and decreased symptom severity compared with a matched comparison diet. It should be noted that several of the foods (apple, onion, pear, and chickpea) removed based on LAT are high in FODMAPs, which may have contributed to the symptom reduction experienced by those in the intervention arm. Although the results are compelling, a larger trial is needed to assess the usefulness of LAT in guiding elimination diets for those with IBS.[35]

The Lifestyle Eating and Performance Mediator Release Test (LEAP MRT) is another commercially available food sensitivity test that lacks validation. There is currently no published, peer-reviewed research to support the anecdotal claims that MRT can identify potential food sensitivities in those with IBS.

LOW FERMENTABLE, OLIGO-,DI-MONO-SACCHARIDE AND POLYOL DIET AND IRRITABLE BOWEL SYNDROME

For years, patients with IBS have identified food as a trigger for their GI symptoms, but the medical community had little to offer in terms of diet interventions. The LFD emerged in the literature in 2005, and through ongoing research has validated a connection between diet and IBS symptom induction. Pioneered by Monash University researchers, FODMAPs were first *speculated* as a potential link to diet-induced changes in small bowel ecology and injurious effects on the colonic epithelium with increases in intestinal permeability, potentially predisposing one to IBD.[36] The LFD is done in 3 phases and ideally, should be administered by a trained GI dietitian, see **Fig. 1**. Gibson and colleagues[37] continue to hypothesize that hyperfermentation

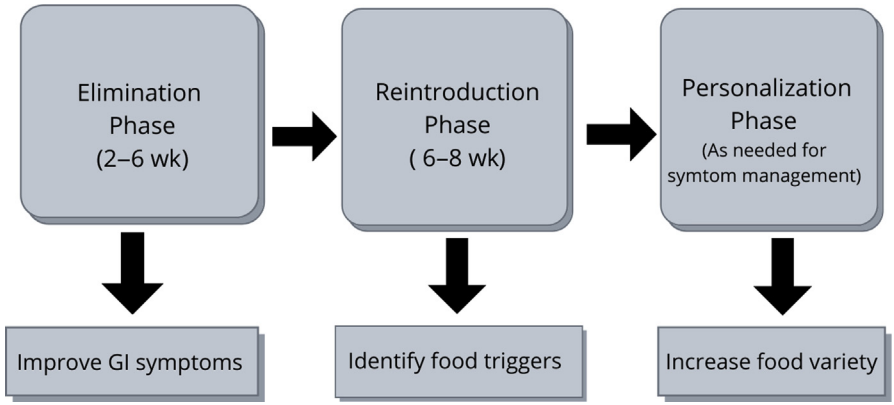

Fig. 1. The low FODMAP diet approach.

related to FODMAPs may be problematic in GI patients, prompting potential inflammatory and intestinal permeability effects as observed in animal studies. FODMAPs are prevalent in the diet in everyday foods, including milk, wheat, onion, garlic, apples, and watermelon, to name a few, and even in popular manufacturing process, such as the addition of fiber additives to our food supply in the form of chicory root extract. Because of their small size, FODMAP carbohydrates are rapidly fermented and osmotically active, prompting luminal distention and gut symptoms in patients with IBS who often have underlying abnormalities in motility and visceral sensation.[11,38,39] The full mechanism for how FODMAPs induce IBS symptoms has yet to be elucidated. Metabolomic research reveals that a high FODMAP diet consumed by patients with IBS increases urinary histamine and an increase in bacterial endotoxin, lipopolysaccharides.[40,41] The dynamics between diet and the gut microbiome, in addition to luminal distention in individuals with IBS appears to play a key role.

Dionne and colleagues,[20] in a systematic review of the LFD, identified 7 RCTs comparing a LFD to a number of different types of control interventions in 397 participants. The LFD was noted to have a reduced relative risk for global symptoms compared with control interventions (Relative risk = 0.69; 95% CI 0.54–0.88). Unfortunately, there was a high degree of heterogenicity in the study designs, limiting the quality of the data.[20] Another systematic review and meta-analysis by Schumann and colleagues[42] found 9 RCTs with a total of 596 subjects. This meta-analysis found significant group differences for LFD compared with other diets with regard to gastrointestinal symptoms (standardized mean difference [SMD] −0.62; 95% CI −0.93 to −0.31; P = .0001), abdominal pain (SMD −0.50; 95% CI −0.77 to −0.22; P = .008), and health-related quality of life (SMD 0.36; 95% CI 0.10–0.62; P = .007).

There is great interest in the LFD in the IBS community. A plethora of food manufacturers are emerging with low FODMAP offerings. In part, the diet has validated the patient's experience that food does impact their GI symptoms. The LFD has metaphorically opened up a Pandora's box for the use of nutritional interventions in patients with IBS.

THE LOW FERMENTABLE, OLIGO-,DI-MONO-SACCHARIDE AND POLYOL DIET AND NUTRIENT ADEQUACY

Removing a number of fruits, vegetables, wheat, and the omnipresent garlic and onion from one's diet can be a challenge on the low FODMAP elimination diet; however, this

dietary intervention does include all food groups and can be nutritionally balanced. The elimination phase of the LFD includes a wide variety of fruits, vegetables, whole grains, nuts, seeds, legumes, lactose-free dairy, and animal proteins. When well planned, the LFD can meet a person's nutrient, both macronutrient and micronutrient, needs. Studies have demonstrated individuals on an LFD have decreased intakes of carbohydrate,[43–46] calcium, [45,46] and energy.[43,44,46] Although other studies have found nutrient intakes to remain similar between an LFD intervention, the habitual diet, or control arm.[47–50] Once adjusting for energy intake, Eswaran and colleagues[46] found the only statistically significant decrease in micronutrient intake observed during a 4-week LFD was for riboflavin. Many patients at baseline were observed to have intakes below the recommended daily intake (DRIs) for several nutrients, including vitamin D, vitamin E, vitamin C, and calcium. A dietitian-led elimination phase can actually enhance micronutrient intake from baseline, as was demonstrated by the increased intake of vitamins A, C, E, K, niacin, B-6, Cu, and Mg on a 4-week LFD, with significant increases in niacin ($P<.05$) and vitamin B-6 ($P<.01$).[46] Moreover, when the LFD was compared with a sham diet, no difference was observed between the 2 groups' total energy, macronutrients, or fiber intake.[47] In this trial, both groups were noted to be consuming approximately 13 g of fiber per day, which is below daily recommended fiber intakes of 25 to 38 g.[51] A recent pilot study demonstrated a dietitian-led LFD in adults older than 65 years did not significantly reduce a participant's nutrient intake from their baseline, but at baseline, most participants consumed less than DRIs of several macronutrients and micronutrients.[49] Long-term nutrient intake in those educated on an LFD appears also to be adequate. Patients who were educated on an LFD and continued some form of restriction, termed 'FODMAP adapted,' were found to be consuming a nutritionally adequate diet up to 18 months after initial education.[50] In addition to improving GI symptoms, any nutrition therapy or diet intervention should be tailored to address and prevent nutrient intake shortfalls.

ROLE OF HISTAMINE INTOLERANCE

Histamine intolerance occurs when there is an imbalance of accumulated histamine and a reduced capacity for histamine degradation.[52] Within the GI tract, exogenous histamine can be impacted by a reduction in diamine oxidase (DAO), the enzyme required to degrade dietary histamine, consumption of a diet rich in histamine content, and the gut microbial metabolism of histidine, which produces histamine. Small bowel inflammation or reduced surface area may reduce DAO production, as it is produced on the apical enterocytes of the upper intestinal villi.[53] Symptoms of histamine intolerance can present with both common IBS symptoms as well as extraintestinal symptoms, including abdominal pain, diarrhea, flushing, urticaria, headaches, vertigo, hypotension, bronchoconstriction, nausea and vomiting.[52] As mentioned previously, one survey study revealed almost 60% of patients with IBS identify histamine-containing foods as a trigger to their digestive distress.[1] The histamine content of foods can vary depending on the microbial composition in the food as well as how the food product is stored and prepared.[54]

Presently, the diagnosis of histamine intolerance is based on the following criteria[55]:

- Presentation of 2 or more histamine-intolerance symptoms
- Improvement with a low-histamine diet
- Improvement with antihistamine medications

Some general recommendations to reduce dietary histamine include reducing high histamine foods, freezing leftover protein-rich foods to retard histamine production,

and consuming fresh, minimally processed foods over ultraprocessed foods. See Table 2 for histamine-rich foods.

The role of a low-histamine diet has not been properly evaluated in patients with IBS but provides another potential area of research in the intersection of diet and IBS.

Table 2
Foods rich in histamine

Food Type	High Histamine Foods
Fruit	Avocado, citrus, strawberries, kiwifruit, papayas, pineapples, dried fruit
Vegetable	Tomatoes, spinach, eggplant
Animal protein	Mackerel, tuna, sardines, anchovies, herring, eggs, aged beef, cured meats, leftover meat or fish
Dairy	Aged cheeses, Kefir, yogurt
Alcohol	All
Other	Nuts, chocolate, vinegar, fermented foods such as kimchi, sauerkraut

Data from Refs.[55–57]

THE SIXTH FERMENTABLE, OLIGO-,DI-MONO-SACCHARIDE AND POLYOL? SUCROSE INTOLERANCE

The LFD does not exclude sucrose, as this disaccharide is typically well digested and absorbed. When there is an absence or reduction in sucrase and isomaltase, however, malabsorption of dietary carbohydrates such as sucrose and starches may prompt GI distress similar to malabsorbed FODMAP carbohydrates due to resultant fermentative and osmotic effects. SID can be either congenital (CSID) or acquired (for example, in association with mucosal injury). SID may be a factor in patients who do not benefit from traditional IBS therapy, such as the LFD. Research revealed patients who carried hypomorphic (pathogenic) sucrase-isomaltase (*SI*) gene variants were significantly less likely to experience adequate symptom relief from the LFD compared with non-carriers (43.5% vs 60.9%; $P = .031$; odds ratio [OR] 4.66).[8]

Although a condition more on the radar for the pediatric gastroenterologists, there may be benefit for assessing for SID in adult patients with IBS who do not benefit from traditional therapy. Symptomology in adults includes frequent, postprandial diarrhea, along with gas and bloating.[58] Patients with CSID tend to have lifelong symptoms. On the other hand, acquired SID may result transiently due to villous atrophy or small intestinal inflammation, such as in untreated celiac disease, Crohn's disease, malnutrition, or in some cases of small intestinal bacterial overgrowth.[59]

Recent studies reveal that heterozygous carriers of *SI* variants experience GI symptoms. One recent study found that CSID genetic mutations were more common in patients with IBS (n = 1031) than asymptomatic controls ($P = .074$; OR 1.84).[60] In a 6-year retrospective study involving disaccharidase assay in nearly 28,000 mucosal biopsy samples in symptomatic children, researchers found that 9.3% of the cohort was deficient in sucrase and maltase.[61] A small study (n=31) in adults with presumed diarrhea-predominant IBS and mixed presentation IBS found SID present in 35% of patients. Among patients with SID, 63.6% had diarrhea, 45.4% had abdominal pain, and 36.4% had bloating.[58] SID (genetic or acquired) should be a consideration, particularly in IBS-D or IBS-M patients who do not respond to an LFD and demonstrate intolerance to foods rich in sucrose, see Table 3.

Table 3
Sample of foods rich in sucrose

Food Type	High-Sucrose Foods[62]
Fruit	Apples, apricots, cantaloupe, dates, mango, nectarines, oranges, peaches, tangerines
Vegetable	Beets, carrots, corn, green peas, sweet potatoes/yams
Sweeteners and ingredients	Sucrose (table sugar), many other types of sugar: brown, cane, beet, coconut, date, and powdered, maple syrup, jam and jelly
Dairy	Products (yogurt, milk, milk shakes) sweetened with the preceding ingredients or containing high-sucrose fruits
Baked and processed foods	Breakfast cereals, baked goods, candy granola bars, pastries, pudding

*Not a complete list.

THE POTENTIAL FOR DISORDERED EATING AND EATING DISORDERS IN IRRITABLE BOWEL SYNDROME

When eating leads to debilitating GI symptoms, there may be a normal adaptive response to avoid food triggers. Determining when eating behavior becomes disordered in a patient with GI symptoms can be challenging. Eating and feeding disorder tools have not been validated in IBS, so one must be cautious when assigning a diagnosis of an eating behavior in this population. A higher prevalence of disordered eating (DE) has been found in patients with GI disorders compared with healthy controls. A systematic review demonstrated 23.43% of general gastroenterology patients engaged in DE behaviors.[63] The 2 most common eating disorders (EDs), anorexia nervosa and bulimia nervosa, can result in digestive distress, and studies reveal 41% to 52% of patients with an ED history have IBS.[64] In addition, in an IBS patient cohort, greater adherence to an LFD was shown to be associated with ED behavior.[65] Recently, research has started to examine Avoidant/Restrictive Food Intake Disorder (ARFID), an ED first included in the *Diagnostic and Statistical Manual of Mental Disorders, 5th Edition*, in the adult gastroenterology population. In general, little research has examined the assessment, treatment, and impact of ARFID in adults. Although the prevalence of ARFID in the adult GI population is not well known, 2 studies found 12.6% to 21.0% of patients met ARFID criteria.[66,67] In addition, a prospective screening study using the 9-item ARFID screen (NIAS) identified a 19.6% positive ARFID screen risk in adult patients with GI disorders.[68] These studies highlight this may be an underrecognized disorder among this population.

When considering the use of diet therapy for a person with IBS, it is essential for clinicians to understand a patient's current dietary habits and history, which includes assessing/screening for maladaptive eating habits and past or present history of an ED. An elimination diet is not appropriate for a patient who has already implemented significant restrictions and/or may be struggling with DE or an active ED. As with all IBS patient care, an integrated care approach is optimal, and for a patient with DE or an ED a mental health provider and dietitian who specializes in ED should be part of their treatment team.

SUMMARY

Diet interventions for patients with IBS are not a "one-size-fits-all" proposition. Depending on the degree of symptomology, initial therapy may simply include adjusting meal

timing, limiting overly processed foods and additives, and adding balance of macronutrients to the diet. In patients with low to moderate symptomology and/or very poor background diet, these slight changes may be all that is warranted. Patients with IBS who were frequently under eating during the day and consuming most of their nutrition in the evening through a larger main meal experienced symptom improvement on a balanced Mediterranean diet. The balanced Mediterranean diet focused mostly on increasing dietary fiber and improving food habits by recommending regular meals and snacks throughout the day. Not surprisingly, this diet approach was preferred by patients over the LFD and GFD.[69] If the patient has failed "cleaning up" the diet, one may consider an LFD intervention as long as there are no contraindications of doing so, such as an active ED or extreme food fears. Details for potential contraindications of the LFD have been reviewed elsewhere.[70] In addition to full diet protocols, such as the LFD for symptom management, one may also consider adding certain foods to offer therapeutic benefit, such as 2 green kiwifruit per day, to aid symptoms of constipation.[71,72] The use of prebiotics combined with diet changes such as the Mediterranean diet are being explored and may provide benefit.[73] Patients with IBS who are interested in diet therapy will benefit from receiving a referral to an experienced GI dietitian. GI dietitians perform a full nutrition assessment, provide detailed instructions, including grocery shopping, menu planning, and label reading, while individualizing the diet intervention to the patient's lifestyle and personal clinical needs.

CLINICS CARE POINTS

- The low FODMAP diet is the 3 phase nutritional approach with the most evidence supporting its efficacy for symptom control in IBS.
- Patients with IBS are at increased risk for maladaptive eating and should be screened for disordered eating and/or eating disorders, particularly prior to prescribing restrictive diet therapies.
- IBS masqueraders such as sucrase isomaltase deficiency, bile acid diarrhea and celiac disease may prompt the necessity of diet therapies other than the low FODMAP diet.
- A GI dietitian referral is recommended for IBS patients to help guide individualized nutritional advice based on lifestyle, symptoms, socio-economics and complete medical history.

DISCLOSURE

Consultant: GI OnDemand (E. Haller). Paid Board Member/Advisory Panel, FODY food company, GI OnDemand Consultant: A2 milk company, Beckon, Gastro Girl. Stock/Shareholder: Fody foods, Epicured, GI OnDemand (K. Scarlata).

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