Irritable bowel syndrome (IBS) is the most common functional gastrointestinal (GI) disorder, affecting approximately 10% to 15% of the worldwide population [1]. Perhaps because it is so prevalent, IBS sometimes is viewed as a trivial disorder; however, in severe cases, the illness can result in significant disability and impairment in patient quality of life [2]. Although gastroenterologists spend a large proportion of their time dealing with IBS, most patients are seen in primary care, and a substantial proportion does not seek health care at all [3].

In spite of growing interest and research into IBS over the last 20 years, the underlying cause of the condition remains unknown. IBS is almost certainly a multi-factorial illness with particular factors being of paramount importance in certain individuals. These include: motility, visceral sensitivity, central processing, genetic factors, psychological factors, inflammation, and dietary factors. From the patient’s perspective, however, the most frequently perceived cause for symptoms is food intolerance. This is perhaps not surprising, given the work by Ragnarsson et al, which suggested that in the region of 50% of patient’s pain episodes worsen in the postprandial period [4]. This study also suggested that pain was exacerbated more frequently by food ingestion than relieved by defecation, the latter characteristic being one of the requirements for a diagnosis of IBS according to the Rome II criteria [5]. Probably as a result of these sorts of observations, approximately 60% of patients with IBS think that they have some form of dietary allergy and hence nearly always want to discuss the role of food in their condition [6].
The importance of the cephalic response to food

Before discussing dietary manipulation, it is important that the patient understands the cephalic response to food, which was demonstrated by Rogers et al in 1993 [7]. These investigators showed that the discussion of food, the smell of food, or even sham feeding led to increased colonic motor activity, thus demonstrating that gut activation can take place even before any food is ingested [8]. Given that increased colonic motor activity has been shown to be associated strongly with abdominal pain in patients with IBS, it is important to realize that the process of eating, irrespective of what is eaten, may exacerbate the symptoms of IBS [8].

Dietary fat, fiber, and other factors influencing gut motility

The most common form of dietary advice offered to patients with IBS is to increase their intake of fiber; indeed a recent survey reported that approximately 95% of general practitioners believe that fiber deficiency is the main cause of IBS [9]. Some years ago, the authors assessed the efficacy of fiber supplementation in treating IBS by recording patients’ overall symptomatic response to several sources of dietary fiber [10]. In this, study cereal fiber made 55% of patients worse, with only 11% claiming an improvement. Other forms of fiber were not so deleterious but nevertheless seldom led to an improvement in symptoms. It should be pointed out that this study was undertaken in secondary care patients; thus it could be argued that patients responding well to dietary fiber in a primary care setting would not need to be referred onwards to secondary care and therefore only patients failing to improve, or being made worse by fiber may have entered the study, resulting in selection bias. The authors recently addressed this issue by examining the response to fiber in 100 consecutive primary care IBS patients and found significant differences compared with patients seen in secondary care. In this second study, only 22% of primary care patients reported symptom deterioration with bran, and 27% reported an improvement (R. Lea, Miller, P.J. Whorwell, unpublished data).

In the secondary care setting, the authors therefore give patients a diet sheet recommending cereal fiber exclusion, and this certainly leads to improvement in a substantial proportion of subjects. It is also worth bearing in mind that coffee may act as a colonic stimulant and can upset some patients; hence a period excluding coffee or caffeine containing products is also worthwhile of consideration [11,12]. Dietary fat is another potent modulator of gut motor function. This has been shown by Serra et al, who conducted a series of studies that demonstrated that in contrast to healthy volunteers, IBS patients exhibited retention of gas that had been infused into the small intestine [13]. Following administration of enteral fat, the volume of retained gas increased from 289 to 505 mL [14]. These studies may help to explain the common clinical experience of patients reporting that meals high
in fat exacerbate symptoms, particularly bloating [11]. This is important, because some patients may erroneously conclude they have intolerance to a food such as milk when in fact it is the fat, rather than the milk protein that is causing problems. In a similar way, mashing potatoes in butter can lead to the conclusion that potatoes are a problem when it is the fat in the butter that is actually causing symptom deterioration. It is also essential to appreciate that other aspects of lifestyle may affect gut motility and therefore be implicated in exacerbation of IBS symptoms. For instance, breakfast is a significant stimulant to the gastro–colonic response, and forgoing this meal can exacerbate constipation. Furthermore missing meals and eating irregularly also seem to aggravate symptoms.

Food intolerance and exclusion diets

In 1982, Alun-Jones et al reported evidence favoring the presence of food intolerance in a large proportion of patients with IBS and claimed that by adopting a strict exclusion diet followed by sequential reintroduction of foods, approximately one third of patients would improve [11]. It should be noted, however, that all these patients had diarrhea-predominant IBS and that other studies have not always been able to confirm such a high response rate with this approach. The literature recently was summarized by Niec et al, who performed a systematic review of clinical trials using food elimination diets followed by rechallenge [15]. Of the seven studies included in their analysis, the response rate varied from 15% to 71%, with the higher figures relating to studies of patients with diarrhea-predominant IBS. Milk, wheat, and eggs were the most frequently implicated foods. Although the principal of food elimination or exclusion appears straightforward, for the patient, it is very demanding and is supervised best by a suitably qualified and enthusiastic dietician, because a major disadvantage of dietary elimination is that it can result in a wide range of foods being excluded. Although this would have to be taken to extremes for significant nutritional problems to develop in previously well-nourished individuals, there is a danger of exacerbating hitherto unrecognized underlying eating disorders, resulting in clinically significant malnutrition. This is particularly important, as some years ago the authors showed that a significant proportion of patients with IBS have a predisposition toward eating disorders.

Food allergy and irritable bowel syndrome

IgE-mediated food allergies are rare in adults and typically occur shortly after the offending food, usually nuts, shellfish, or fish, is ingested [16]. Symptoms of abdominal pain, vomiting, and diarrhea, often associated with extra-GI problems such as urticaria, wheezing, or even anaphylaxis, are characteristic. Because they may be potentially life-threatening the
possibility of an IgE-mediated food allergy, in the appropriate clinical context, should not be overlooked, especially in a patient with a personal or family history of atopic disorders.

True food allergy has not been well studied in the much more common clinical setting of IBS. On the occasions when it has been studied, it predominantly has been investigated using IgE-mediated skin prick or radioallergosorbent test (RAST). Petitpierre et al used these methods in 12 atopic and 12 nonatopic individuals who were given an exclusion diet with subsequent food provocation [17]. Fourteen of the 24 subjects responded to food elimination and suffered typical IBS symptoms on dietary rechallenging, with an IgE-mediated mechanism considered likely in nine of the atopic individuals. Thus although food allergy is unlikely to explain symptoms in most patients with IBS, this mechanism may be important in the subgroup of patients with atopy. Although the obvious treatment for these patients is dietary elimination, in practice it may be difficult to determine the offending food and even more difficult to completely remove all traces of it from the diet. To address one possible solution to this problem, Stefanini et al conducted a 4-week multi-center study comparing the efficacy of the mast cell stabilizing agent sodium cromoglycate at 1500 mg per day with an elimination diet and reported that 67% of patients improved with cromoglycate, compared with 60% using the elimination diet [18]. Thus sodium cromoglycate taken before each and every meal may be worth trying in patients suspected of having food allergy and having difficulty with dietary elimination.

In contrast to IgE, IgG antibodies to food are common in the normal population and often have been considered physiological; therefore their potential role in IBS had not been studied previously. In a recent randomized controlled trial, the authors assessed the effectiveness of a food elimination diet based on the presence of IgG antibodies for treating patients with IBS [19]. One hundred and fifty patients were randomized to receive either a true diet based on the patient’s individual food antibody profile or a sham diet excluding a similar number of foods, but not those to which they had antibodies. Participants remained on the diet for 12 weeks and were then observed during a 4-week food reintroduction phase. As can be seen from Fig. 1, which compares symptom severity scores in the two groups of patients fully adherent to the diet, patients receiving the IgG-determined elimination diet improved significantly more than those receiving the sham diet. Global rating scores also showed significant improvements, and patients on the true diet suffered far greater deterioration in symptoms than the sham diet group when the diet was relaxed. The foods to which patients were most likely to have antibodies were yeast, milk, egg, wheat, cashew nuts, peas, almonds, and barley. The mechanism by which IgG food antibodies could be mediating this detrimental effect in IBS is unclear; however, in view of mounting evidence to support a low-grade inflammatory process in some patients [20], it is tempting to speculate
that IgG antibodies could be involved in this process. Ultimately, whatever the underlying mechanisms, this study suggests that further research in the area is warranted.

**Carbohydrate malabsorption**

The role of carbohydrate intolerance in IBS has been addressed by several investigators [21–28]. One representative study measured breath hydrogen and GI symptoms following oral administration of lactose, fructose, sorbitol, and sucrose in 25 patients with functional bowel disorder and 20 healthy volunteers [21]. An elimination diet based on the results of these tests then was implemented. Malabsorption of at least one sugar occurred in 90% of all subjects, although symptom scores were significantly higher in the IBS patients compared with volunteers, and 40% of the patients improved following elimination of the offending sugar. Therefore, while there is evidence that fructose, lactose, and sorbitol malabsorption are all common in IBS patients, malabsorption of these sugars is similarly prevalent in healthy volunteers [21–23,25,26,29–31]. Nevertheless, restriction of carbohydrates in subjects with IBS does seem to help improve symptoms. Thus this approach is worth considering, especially in view of the increasing use of sugar substitutes by food manufacturers. An interesting example of this issue is the use of small quantities of sorbitol in chewing gum, which is sometimes implicated as a cause for symptoms by patients with IBS. Although this may indeed, in part, be related to the presence of sorbitol in the gum as has been discussed, the act of chewing itself stimulates GI motor activity, and this also could result in exacerbation of symptoms [7].
**Probiotics**

The potential beneficial effects of probiotics are being investigated in a several diseases, and IBS is no exception. The main probiotic bacteria are *Lactobacillus*, *Bifidobacterium*, and some nonpathogenic forms of *Escherichia coli*. The capacity of these bacteria to adhere to the mucosa seems to be therapeutically important [32], and, by definition, they are all living organisms and therefore need to be able to survive the acidic gastric environment following ingestion to confer any benefit. Initial studies in IBS have been conflicting. Two randomized controlled trials that administered *Lactobacillus plantarum* or placebo to patients with IBS reported a reduction in either flatulence or pain and better overall GI functioning [33,34], although another trial examining the efficacy of this organism failed to demonstrate any effect over placebo [35]. A further study, using the patented probiotic formulation (VSL#3), suggested that although there were no differences in global relief scores, there was a tendency for bloating to improve [36]. An additional study that used tablets containing *Lactobacillus GG* also demonstrated little benefit [37]. One potential reason for these contradictory findings is the vast range of probiotic species available, which probably all vary in their therapeutic potential and degree of adherence. Thus failure of one organism to have an effect does not necessarily imply that this approach is doomed to failure. This view is supported by the preliminary report of a newly identified strain of *Bifidobacterium* appearing to have a promising effect in IBS [38].

**Celiac disease**

There has been interest recently in the potential overlap between celiac disease and IBS. The prevalence of celiac disease in the population is approximately 0.2% to 0.3%, and recent reports have indicated that this figure may increase to between 10% and 20% in patients with IBS [39–41]. It is possible, however, that this particular subgroup of IBS patients simply may represent a cohort of misdiagnosed patients with celiac disease. To address this issue, it would be helpful to know the prevalence of IBS in patients diagnosed with celiac disease who are adherent to a gluten-free diet, and also to know whether treating celiac disease improves IBS symptoms [42]. It is also important to remember, as has been discussed, that wheat fiber intolerance has a high prevalence in patients with IBS, and therefore benefit from a gluten-free diet may be caused by wheat fiber exclusion rather than gluten withdrawal. With the role of inflammation in IBS becoming increasingly topical, it is interesting to note that patients with inflammatory bowel disease in remission have a higher prevalence of IBS symptoms [43,44]. Thus it might be anticipated that patients with celiac disease in remission also might be affected similarly. There is now debate surrounding the whole issue of screening patients with IBS for celiac disease [45,46] and
whatever the effect on symptoms of a gluten-free diet, there is benefit to be gained in reducing the risk of complications such as osteoporosis and malignancy in treating IBS patients diagnosed with celiac disease.

Summary

Irritable bowel syndrome patients frequently believe that food intolerances are to blame for many of their symptoms, although not uncommonly this is caused by the nonspecific increase in gut motility that occurs with food ingestion. Nevertheless, dietary manipulation may result in substantial improvement in IBS symptomatology provided it is individualized to the particular patient. By further understanding the mechanisms involved in dietary intolerance, it should be possible to optimize the benefits of this approach to treatment.

References


