The role of food intolerance in functional gastrointestinal disorders in children

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Background

Functional gastrointestinal disorder (FGID) is a common, benign, chronic diagnosis that has a significant negative impact on quality of life. FGIDs that develop in childhood can persist into adulthood. Currently, there is no cure and few treatment options are available.

Objective

This article provides an outline of current research supporting the role of food intolerance in children with FGIDs.

Discussion

Food intolerances have long been reported by patients with FGIDs; however, randomised controlled trials are lacking in this area. Food intolerances that have been investigated include intolerance to food chemicals, lactose, fructose and, more recently, fermentable carbohydrates, termed FODMAPs. The low-FODMAP diet eliminates poorly absorbed short-chain carbohydrates and has a clearly defined mechanism of action. Emerging evidence suggests it alleviates symptoms in adults with irritable bowel syndrome and, potentially, also in children. However, more evidence is required for the efficacy of the diet in children and in other subgroups of FGID. Any dietary restriction in growing children should be undertaken with clinical supervision by a dietitian.

Keywords

paediatrics; food hypersensitivity; gastrointestinal diseases

CPD

Scope of the problem

A functional gastrointestinal disorder (FGID) is defined by the Rome III criteria as a variable combination of chronic or recurrent gastrointestinal symptoms, such as diarrhoea, constipation and abdominal pain, which are not explained by structural or biochemical abnormalities.[1] Prior to the 2006 Rome III criteria, most paediatric studies considered the broader definition of recurrent abdominal pain (RAP)[2] and these studies found RAP was prevalent in 8–25% of school-aged children.[2,3] More recent studies found that RAP accounts for about 5% of childhood consultations in general practice and only approximately 50% of these are attributable to a FGID.[4] The true epidemiology of FGIDs is unknown as only 10–46% of affected individuals seek medical attention.[5]

Although a benign diagnosis, FGIDs have a significant effect on a child's quality of life, with data showing the quality of life of children with a FGID is similar to those with active inflammatory bowel disease.[6] FGIDs have a negative impact on a child's school performance, sports and social activities.[7] This translates to considerable health costs,[8] including increased medical consultations, sick leave and work/school absenteeism. Given that FGIDs are chronic and the burden lifelong, childhood cases are likely to persist into adulthood,[9] thereby affecting quality of life in the long term.

Diagnosis

All FGIDs are diagnosed using validated symptom-based clinical criteria (namely, the Rome III criteria).[1] Simple laboratory and endoscopic tests are used to eliminate organic bowel diseases, including coeliac disease, inflammatory bowel disease, food allergy, eosinophilic oesophagitis, colorectal cancer and gastroenteritis, symptoms of which are similar to FGIDs. It is important to note that the diagnosis of FGID remains a diagnosis of exclusion. This review is concerned with FGID with associated abdominal pain, which includes functional dyspepsia, irritable bowel syndrome (IBS), abdominal migraine and childhood functional abdominal pain.[1]

Despite FGIDs being chronic and debilitating, few treatment options are available and those options have a low and variable success rate. FGIDs present a significant therapeutic challenge because of their wide range of symptomatology, poorly understood aetiology and, hence, a lack of pharmacological targets. However, IBS, the most common FGID,[1] has garnered the most attention with respect to the role of food intolerances in symptom generation, albeit limited attention with respect to controlled trials in children.

Food intolerance

The term food intolerance can be defined as a non-immunologically mediated adverse reaction to food, which can be resolved following dietary elimination of the suspect food and reproduced by the food challenge.[10] There are no known biological markers that confirm food intolerance.[10,11] Food intolerances are not life-threatening and most individuals with food intolerances can tolerate small amounts of the 'trigger' food in their diet without ill effect. The elimination of a suspect food or foods from an individual's diet is not a cure but, rather, a means to providing symptom relief.

Two out of every three adults with IBS report that eating certain foods triggers symptoms.[12] Furthermore, the greater the severity of IBS symptoms, the more food items patients identify as being responsible for the symptoms.[13] Similarly, children have reported that certain foods exacerbate symptoms. Carlson et al[7] studied perceived food intolerance in 25 child–parent pairs through a questionnaire and focus groups. The majority of children participating were classified as either having IBS or abdominal migraine. The median number of foods identified as producing gastrointestinal symptoms was 11; the top three trigger foods were self-identified as spicy foods, pizza and cow's milk. Children identified coping strategies, including eating smaller portions, modifying foods or avoiding the food altogether.

There have been numerous attempts to define a diet that would assist in controlling symptoms in FGIDs. Intolerance to food chemicals (both natural and added) has been linked to FGIDs. Two of the main culprits are believed to be amines and salicylates. Russell et al[14] suggest a diet low in amines is beneficial for reducing the severity and frequency of abdominal migraine in children. They further suggest a 'few-foods' or oligo-antigenic diet, but to our knowledge there is no support in the literature for these recommendations. In Australia a diet regularly used for conditions associated with food intolerance, including IBS, is The Royal Prince Alfred Hospital (RPAH) elimination diet. This diet was adapted from the Feingold Diet,[15] which also focuses on elimination of food chemicals. However, randomised controlled trials for its efficacy in children are lacking, and current evidence for its use in FGIDs is weak.[16] Furthermore, poor dietary compliance and the ad hoc removal of extra whole foods, namely dairy, soy and wheat, has resulted in inconsistent use of the diet and heterogeneity of evidence. In removing these whole foods it becomes unclear whether the diet's effect is indeed due to a food chemical intolerance or other food intolerances, such as protein intolerance or poorly absorbed carbohydrates, as these confounding variables exist within dairy, wheat and soy food groups.[16]

Poorly absorbed carbohydrates

Malabsorption of carbohydrates has been implicated in the pathogenesis of FGIDs and is an emerging area where increasing evidence in studies in adults may be promising for children with FGIDs. However, early work that focused on single carbohydrates is less convincing. Dearlove et al[17] and Lebenthal et al[18] conducted randomised controlled trials of lactose-free diets in children with RAP but there was no clear evidence that lactose elimination was effective. By contrast, the contribution of fructose to functional abdominal pain has been investigated by Gomara et al[19] and Escobar et al[20] with more promising results. Gomara et al[19] randomly gave doses of 1 g, 15 g and 45 g of fructose to 32 children with FGIDs and reported a positive breath hydrogen test in 11 participants. Interestingly, children with and without a positive breath test reported exacerbation of symptoms over the 3-hour period following the fructose load. Further, the 11 patients with a positive breath test were asked to follow a restricted fructose diet and 81% reported immediate symptom improvement. In particular, abdominal pain and bloating continued to be significantly reduced 2 months after the initial breath test. Similarly, in a larger study Escobar et al[20] recruited 121 children with functional abdominal pain and a positive hydrogen breath test for fructose intolerance, instructed them on a low-fructose diet and found 77% reported resolution of symptoms.

In 2005, a therapeutic diet emerged that aimed to minimise poorly absorbed short chain carbohydrates, denoted by the acronym FODMAPs (fermentable oligo-, di-, monosaccharides and polyols).[21] *Table 1* shows the five FODMAP groups, their malabsorption rates and examples of food sources. Interestingly, fructose and lactose are two of the five FODMAP groups highlighted for restriction in this diet. It is important to note, however, that the low-FODMAP diet involves investigation as to which groups prove to be problematic in any individual and therefore restriction is only recommended for carbohydrate groups that elicit symptoms, rather than across all FODMAP groups. This ensures a diet that can be liberalised over time, with challenges to determine an individual's threshold for tolerance.

A low-FODMAP diet alleviates gastrointestinal symptoms by reducing the amount of undigested carbohydrate that presents to colonic bacteria. Less fermentation results in decreased abdominal bloating and pain, as well as less flatulence. Ong et al[22] found that malabsorption of FODMAPs is not more common in people with IBS than those without IBS; however, people with IBS produce more gas and report more symptoms.

Initial studies in adults showed that reducing FODMAPs in an individual's diet led to a significant decrease in symptoms of IBS.[21] Subsequently, a randomised controlled crossover trial by Shepherd et al[23] has shown that dietary FODMAPs triggered IBS symptoms in 25 adult participants. More recently, Halmos et al[24] conducted a double-blind randomised controlled crossover trial that compared a low FODMAP diet with a standard, nutritionally balanced Australian diet in 30 adults with IBS. They found that a low-FODMAP diet significantly improved satisfaction with stool consistency, and decreased bloating, abdominal pain and breath hydrogen. These data from randomised controlled trials support that a low-FODMAP diet warrants

consideration as a management strategy for adults with IBS. More studies are needed to investigate this diet as a therapeutic treatment for other subtypes of FGID.

There is emerging evidence for consideration of a low-FODMAP when treating children with abdominal pain related to FGIDs. Chumpitazi et al[25] conducted a double-blind randomised controlled trial of 54 children with IBS, aged 7–17 years. They compared a low-FODMAP diet to a high-FODMAP diet using a crossover design and found fewer episodes of abdominal pain, less bloating, less nausea and lower breath hydrogen production after only 2 days on the low-FODMAP diet. Further studies in children are needed to confirm these findings and, as with adults, to determine the value of the low-FODMAP diet in other forms of FGID.

Practicalities of food elimination diets

Following any dietary restriction has risks and there is evidence that food elimination diets can result in weight loss, failure to thrive, food aversions,

eating disorders and an increased risk of nutritional deficiency, especially in those who adhere long term to strict dietary eliminations.[12,16] The risk of nutritional deficiency is magnified in growing children, which highlights the importance of clinical supervision to ensure adherence to a balanced diet. Consultation with a dietitian will help ensure maximum liberalisation of an individual's long-term diet.

Challenges of managing FDIG

Table 2 presents typical clinical scenarios of paediatric FGIDs and outlines appropriate management strategies.

Conclusion

Although patients report that certain foods trigger symptoms of FGIDs, there are limited data to support the role of food intolerance in the literature. While there is some evidence suggesting a role for dietary therapies when treating children with FGIDs, much of what we know currently is extrapolated from studies involving adults. There is increasing evidence from randomised controlled trials to support the low-FODMAP diet for adults with abdominal pain related to FGID and, more recently, has emerged for children. It is noted that any elimination diet should be undertaken with clinical supervision, especially in growing children. More research is needed to establish the efficacy of the low-FODMAP diet in children with FGID and which subtypes of FGID are most likely to respond to a low-FODMAP diet. Literature addressing the role of food chemical intolerances is scarce, and further studies are needed.

Key points

- More randomised controlled trials are needed to address the therapeutic role of food intolerance in the treatment of children with FGIDs.
- The low-FODMAP diet is emerging as a successful dietary therapy for adults with IBS, with somewhat less evidence for children.
- More research is needed to establish whether other subtypes of FGID also respond to a low-FODMAP diet, and to investigate other potential food intolerances.
- Care should be taken with dietary restrictions especially in growing children and clinical supervision by a dietitian is strongly recommended.

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Table 1. FODMAPs overview[26]

FODMAP	Malabsorption rates	Food sources
Fructose (in excess of gluten)	30–40% of the population do not absorb fructose efficiently but not all report symptoms	Honey Apple Apricot Avocado
Lactose	Malabsorption is due to the biological absence of the enzyme lactase. Nearly 100% of children are born with this enzyme but a significant proportion of the population have reduced activity after early childhood.	Dairy milk Yogurt Custard Dairy deserts
Fructooligosaccharides (fructans/FOS)	Malabsorption in all people	Wheat products
Galactooligosaccharides (GOS)	Malabsorption in all people	Cabbage Legumes Garlic Leek
Polyols (sugar alcohols)	Malabsorption in 50% of people	Pear Apricot Corn Cauliflower Mushroom

Table 2. Clinical scenarios of paediatric FGIDs

A child aged 3 years has frequent bouts of diarrhoea associated with dairy food consumption and a history of abdominal pain.

- May be lactose or milk protein allergy
- Are there any alarm features for organic disease? Consider past feeding and family histories
- Toddlers have high nutrient requirements, small intakes and are dependent on others for their nutrition. They are also still learning and developing the skills to eat a nutritious balanced diet. Therefore, any elimination diet should be performed under the clinical supervision of an accredited practicing dietitian (APD)* only

An adolescent girl frequently reports debilitating abdominal pain that is affecting her school attendance; she has noted that chewing artificially sweetened gum triggers attacks.

- Consider premenstrual pain or other organic bowel diseases
- FODMAPs? Artificial gum is high in polyols, which are malabsorbed in 50% of all people; some may experience abdominal pain/diarrhoea if eaten in excess
- Does she have abdominal pain when not chewing gum and not premenstrual? If yes, then consider a trial of FODMAP restriction under the supervision of an APD

A boy aged 10 years was recently treated for traveller's diarrhoea. He has had ongoing diarrhoea for 6 months, despite negative stool tests.

- Consider post-gastroenteritis IBS
- Eliminate organic causes before commencing an elimination diet
- Trial dietary FODMAP restriction: some FODMAPs may still be tolerated, therefore, consultation with an APD will help achieve maximum liberalisation of this growing boy's diet.

A girl, 5.5 years of age, presents with poor concentration at school. Blood tests confirm anaemia and a brief dietary history revealed she was avoiding dairy and wheat because of bloating and discomfort.

- Anaemia is an alarm feature for organic disease. It suggests malabsorpiton, and supplementation can be commenced immediately
- Investigate family history of organic bowel diseases and FGIDs
- Test for coeliac disease – patient will need to be eating gluten-containing foods equivalent to 2 slices of bread per day for 6 weeks prior to blood tests and bowel biopsy (see Coeliac Australia www.coeliac.org.au).
- Once the diagnosis of coeliac disease has been confirmed refer to an APD for full dietary assessment and prescription

*To find an APD in your local area, visit the 'Find an APD' section of the Dietetic Association of Australia website at www.daa.asn.au or telephone 1800 812942

References

1. Rasquin A, Di Lorenzo C, Forbes D, et al. Childhood functional gastrointestinal disorders: Child/adolescent. Gastroenterology 2006;130:1527–37.
2. Apley J, Naish N. Recurrent Abdominal Pain (RAP): a field survey of 1000 school children. Arch Dis Child 1958;3:165–70.
3. Chitkara DK, Rawat DJ, Talley NJ. The epidemiology of childhood recurrent abdominal pain in Western countries: a systematic review. Am J Gastroenterol 2005;100:1868–75.
4. Spee LA, Lisman-Van LY, Benninga MA, Bierma-Zeinstra SM, Berger MY. Prevalence, characteristics, and management of childhood functional abdominal pain in general practice. Scand J Prim Health Care 2013;31:197–202.
5. Drossman DA, Li Z, Andruzzi E, et al. U. S. Householder survey of functional gastrointestinal disorders. Dig Dis Sci 1993;38:1569–80.
6. Youssef NN, Murphy TG, Langseder AL, Rosh JR. Quality of life for children with functional abdominal pain: A comparison study of patients' and parents' perceptions. Pediatrics 2006;117:54–59.
7. Carlson MJ, Moore CE, Tsai CM, Shulman RJ, Chumpitazi BP. Child and parent perceived food-induced gastrointestinal symptoms and quality of life in children with functional gastrointestinal disorders. J Acad Nutr Diet 2014;114:403–13.
8. Maxion-Bergemann S, Thielecke F, Abel F, Bergemann R. Costs of irritable bowel syndrome in the UK and US. Pharmacoeconomics 2006;24:21–37.
9. Horst S, Shelby G, Anderson J, et al. Predicting Persistence of Functional Abdominal Pain From Childhood Into Young Adulthood. Clin Gastroenterol Hepatol 2014; [Epub ahead of print].
10. Zar S, Kumar D, Benson MJ. Food hypersensitivity and irritable bowel syndrome. Aliment Pharmacol Ther 2001;15:439–49.
11. Clarke L, McQueen J, Samild A, Swain A. Dietitians Association of Australia review paper. Aust J Nutr Diet 1996;53:89–98.
12. Monsbakken KW, Vandvik PO, Farup PG. Perceived food intolerance in subjects with irritable bowel syndrome – etiology, prevalence and consequences. E J Clin Nutr 2006;60:667–72.
13. Bohn L, Storsrud S, Tornblom H, Bengtsson U, Simren M. Self-reported food-related gastrointestinal symptoms in IBS are common and associated with more severe symptoms and reduced quality of life. Am J Gastroenterol 2013;108:634–41.
14. Russell G, Abu-Arafeh I, Symon DN. Abdominal migraine: evidence for existence and treatment options. Paediatr Drugs 2002;4:1–8.
15. Feingold BF. Behavioral disturbances, learning disabilities, and food additives. Chemtech 1975;5:264–67.
16. Gray P, Mehr S, Katelaris C, et al. Salicylate elimination diets in children: is food restriction supported by the evidence? Med J Aust 2013;198:600–02.
17. Dearlove J, Dearlove B, Pearl K, Primavesi R. Dietary lactose and the child with abdominal pain. Br Med J (Clin Res Ed) 1983;286:1936.
18. Lebenthal E, Rossi TM, Nord KS, Branski D. Recurrent abdominal pain and lactose absorption in children. Pediatrics 1981;67:828–32.
19. Gomara RE, Halata MS, Newman LJ, et al. Fructose intolerance in children presenting with abdominal pain. J Pediatr Gastroenterol Nutr 2008;47:303–08.
20. Escobar MA, Jr, Lustig D, Pflugeisen BM, et al. Fructose intolerance/malabsorption and recurrent abdominal pain in children. J Pediatr Gastroenterol Nutr 2014;58:498–501.
21. Shepherd SJ, Gibson PR. Fructose malabsorption and symptoms of irritable bowel syndrome: guidelines for effective dietary management. J Am Diet Assoc 2006;106:1631–39.
22. Ong DK, Mitchell SB, Barrett JS, et al. Manipulation of dietary short chain carbohydrates alters the pattern of gas production and genesis of symptoms in irritable bowel syndrome. J Gastroenterol Hepatol 2010;25:1366–73.
23. Shepherd SJ, Parker FC, Muir JG, Gibson PR. Dietary triggers of abdominal symptoms in patients with irritable bowel syndrome: randomized placebo-controlled evidence. Clin Gastroenterol Hepatol 2008;6:765–71.
24. Halmos EP, Power VA, Shepherd SJ, Gibson PR, Muir JG. A diet low in FODMAPs reduces symptoms of irritable bowel syndrome. Gastroenterology 2014;146:67–75.e5.
25. Chumpitazi BP, Tsai CM, McMeans AR, Shulman RJ. 823 A Low FODMAPS diet ameliorates symptoms in children with irritable bowel syndrome: a double-blind, randomized crossover trial. Gastroenterology 2014;146:S144.
26. Shepherd S, Gibson P. The Food Intolerance Management Plan. Melbourne: Viking Australia, 2011.

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