

Management of Patients with Disorders of Gut-Brain Interaction and Altered (Reduced) Food Intake Behaviour

Short title: DGBI-induced altered food intake behaviour

Ayesha Shah^{§*#@ 1,2}, Jason P. Connor^{*@3}, Nicholas J. Talley^{%%\$#@4}, Andrew Day⁺⁵, Basil Almehdawy^{*2}, Chamara Basnayake^{#6,7}, Rebecca Burgell^{#8}, Sharon Carey^{~&9,10}, Charles Cock^{#11}, Charlotte Daker⁺¹², Kerith Duncanson^{@\$4}, Vishal Kaushik^{*13}, Simon R. Knowles^{#@14}, Dan Kent^{*15}, Trina Kellar^{*#13}, Natasha Koloski^{*@1,2}, Katherine Lane^{*16}, Neal Martin^{*1,2}, Nikhil Thapar^{*>2,17,18}, Paul Stanley^{*1}, Allison Malcolm^{#19,20}, Kate Elizabeth Murphy¹⁶, Gemma Sharp²¹, Caroline Tuck^{@14}, Michael Jones^{§, 22}, Gerald Holtmann^{*\$@^1,2}

@Member of the guideline steering committee

^Chair of the Steering Committee

%Chair of the GESA Luminal Faculty

>Chair of the GESA Paediatric Faculty

~Australasian Society for Parenteral and Enteral Nutrition

#Member of the GESA Luminal Faculty

*Member of the Statewide Queensland Clinical Gastroenterology Network

&Australian Society of Parenteral and Enteral Nutrition (AuSPEN)

+New Zealand Society of Gastroenterology

§Australian Gastrointestinal Research Alliance and NHMRC CRE in Transforming Gut Health



- ¹Department of Gastroenterology and Hepatology, Princess Alexandra Hospital, Brisbane, QLD, Australia;
- ²Faculty of Health, Medicine and Behavioural Sciences, University of Queensland, Brisbane, QLD, Australia.
- ³Alcohol and Drug Assessment Unit, Princess Alexandra Hospital, Brisbane, QLD, Australia.
- ⁴College of Health, Medicine and Wellbeing, The University of Newcastle, NSW, Australia.
- ⁵Department of Paediatrics, University of Otago Christchurch, Christchurch, New Zealand
- ⁶Department of Gastroenterology, St Vincent's Hospital Melbourne, Melbourne, VIC, Australia.
- ⁷Department of Medicine, University of Melbourne, Melbourne, VIC, Australia.
- ⁸Department of Gastroenterology, Monash University, Alfred Hospital, Melbourne, VIC, Australia.
- ⁹Faculty of Medicine and Health, University of Sydney, Sydney, NSW Australia.
- ¹⁰Department of Nutrition & Dietetics, Royal Prince Alfred Hospital, Sydney, NSW, Australia.
- ¹¹Dept Gastroenterology & Hepatology, Flinders Medical Centre, Southern Adelaide Local Health Network & Flinders University, South Australia.
- ¹²Department of Gastroenterology, North Shore Hospital, Auckland, New Zealand.
- ¹³Department of Gastroenterology and Hepatology, Royal Brisbane and Women's Hospital, Herston, QLD, Australia.
- ¹⁴School of Health Sciences, Swinburne University of Technology, Hawthorn, Australia.
- ¹⁵Consumer Representative, Queensland Clinical Network, Brisbane, Australia
- ¹⁶Queensland Eating Disorder Service, Metro North Mental Health Service, Brisbane, QLD, Australia.
- ¹⁷Gastroenterology, Hepatology and Liver Transplant, Queensland Children's Hospital, Brisbane, QLD, Australia.
- ¹⁸Centre for Childhood Nutrition Research, Queensland University of Technology, Brisbane, QLD, Australia
- ¹⁹Department of Gastroenterology, Royal North Shore Hospital, St Leonards, NSW; Australia
- ²⁰The Faculty of Medicine and Health, University of Sydney, Sydney, NSW, Australia.
- ²¹Department of Neuroscience, Monash University, Melbourne, VIC, Australia
- ²²Department of Psychology, Macquarie University, Sydney, NSW, Australia.

Address for correspondence:

Gerald J. Holtmann, MD, PhD, MBA, FRACP, FRCP, FAHMS
Professor of Medicine
Princess Alexandra Hospital, Brisbane
Department of Gastroenterology and Hepatology & University of Queensland
Ipswich Road, Woolloongabba, Queensland, Australia
Email: g.holtmann@uq.edu.au
Phone +61 424 956 000
Facsimile +61 7 3176 5111

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Summary:

Disorders of Gut-Brain Interaction (DGBI) can result in significant meal-related symptoms, leading to weight loss and nutritional deficiencies. They present clinically as a distinct DGBI patient cohort. This guideline is for gastroenterologists and other healthcare professionals, reflecting expert consensus based upon a review of the available evidence.

Patients, both adults and children with DGBI and altered food intake, frequently face stigma stemming from the perception that their gastrointestinal symptoms are ‘not real’ and primarily due to ‘psychological factors’. This perception can be further exacerbated by presentation with comorbidities such as hypermobility spectrum disorders (HSD), postural orthostatic tachycardia syndrome (POTS), mast cell activation syndrome (MCAS), and median arcuate ligament syndrome (MALS) that complicate subsequent care.

With frequently severe symptoms and multiple comorbidities, these patients often embark on a search for underlying organic causes. In a fragmented healthcare setting where patients consult multiple specialists over time, there is an increased risk that well-intended medical or dietary interventions may cause harm and further exacerbate the situation.

Effective management should consider relevant differential diagnoses, including organic causes or eating disorders, and establish a diagnosis of DGBI predating the altered food intake. Long-term care should focus on empathetic patient support, education and setting realistic expectations, facilitated through an integrated multidisciplinary team approach of gastroenterologists and allied health staff, including mental health specialists. Treatment plans should be tailored to individual symptoms.

Multidisciplinary care should be offered. All pharmacological and non-pharmacological treatments should follow established DGBI treatment principles, tailored to the predominant symptoms, while previous treatment responses are considered. Invasive treatments, such as gastric, post-pyloric tube feeding or parenteral nutrition, should be only considered for short periods of time to address life-threatening, severe nutritional deficiencies after failure of effortful oral intake.

Future research needs to define key epidemiologic characteristics of these conditions, including risk factors for their manifestation, and generate evidence to guide the long-term management.

Scope of the document

Purpose of the clinical guidelines

Disorders of Gut-Brain Interaction (DGBI) include conditions such as irritable bowel syndrome (IBS) and functional dyspepsia (FD), which are characterised by gastrointestinal symptoms and cognitive/emotional factors without significant structural abnormalities of the digestive tract¹. In recent years, there has been a notable increase in the number of patients with DGBI² and meal-related symptoms presenting with weight loss due to altered (reduced) food intake³ and disordered eating behaviours⁴⁻⁶. This includes food avoidance, irregular eating patterns, meal skipping, not eating when hungry, vomiting after meals and a reduced daily caloric intake.

This guideline is intended to support adult and paediatric health care professionals (with a special focus on multiprofessional teams working with gastroenterologists) in providing appropriate and consistent care for these patients with DGBI-induced food intake behaviour. While some of these patients may meet DSM-5 criteria for eating disorders, it is essential to recognise DGBI can also be the primary driver of the altered food intake, necessitating appropriate treatment of the underlying DGBI in these patients. Thus, this guideline does not intend to replace the established clinical guidelines for the management of patients with eating disorders⁷⁻⁹. However, effectively managing patients with DGBI and altered food intake is critical for gastroenterologists and allied health professionals.

Methods:

The members of the consensus team were experts of the Luminal and Paediatric Faculties of the Gastrointestinal Society of Australia (GESA) and the Queensland Gastroenterology Clinical Network selected based on interest and experience related to the topic. In addition, Aotearoa New Zealand clinicians were invited to participate in order to create a consistent, trans-Tasman approach. The consensus team included Gastroenterologists, Dietitians, Psychologists, a Psychiatrist, Nursing staff, representation from the Australian Society of Parenteral and Enteral Nutrition (AusPEN) and a consumer representative. A cross-disciplinary steering committee of eight members was subsequently established. Based on a review of the existing literature, a draft guideline was developed by the members of the Steering Committee with input from the consumer representative and other team members. The draft was further refined with contributions from members of the Australian Gastrointestinal Research Alliance (AGIRA) and the other members of the consensus team. In a subsequent step, 23 statements related to definitions, epidemiology, pathophysiology, clinical presentation and treatment were articulated.

These statements were subsequently graded utilising the GRADE (Grading of Recommendations Assessment, Development and Evaluation) system to assess the evidence and the strength of recommendations (Table 1). The consensus process involved a modified Delphi method¹⁰. The first round of online voting was done in June 2025. The poll was conducted using an electronic online system

(Microsoft Forms), and the Steering Committee analysed the results, determining the level of support for the various statements.

Patient and consumer focus

Patients and families dealing with DGBI and altered food intake need reliable information about the causes, treatment options and long-term outcomes associated with these conditions. While the internet offers a wide range of easily accessible information, it often lacks the nuanced and specialised insights necessary to address the complexities of DGBI and altered food intake. Additionally, these information sources tend to overlook the important distinctions between DGBI and traditional eating disorders and the possibility of an overlap between DGBI and eating disorders. Consequently, individuals seeking guidance may encounter oversimplified or even misleading content that may not adequately reflect the complexities of diagnosis and treatment. Although this document is primarily designed for healthcare providers, it also serves as a foundation for creating a consumer-friendly summary that distils this information into accessible language for patients and their relatives to enable informed decisions without the bias of often interest-driven sources of information. This dual approach reinforces the commitment to promoting informed, patient-centred care in managing DGBI and altered food intake behaviour. For the development of this guideline, the consumer representative of the Queensland Health Clinical Network (DK) attended many of the required team meetings and provided repeated input and feedback on the document.

Context and available international recommendations

This document is intended to be evaluated in conjunction with existing consensus documents from national¹¹⁻¹⁷ and international authorities^{1, 18} regarding DGBI. Furthermore, it is essential to consider relevant guidance documents focused on managing eating disorders^{7, 19, 20}, particularly Avoidant/Restrictive Food Intake Disorder (ARFID).

Unmet clinical need

The unmet clinical needs of patients with established or suspected DGBI and altered food intake behaviours related to gastrointestinal symptoms can be multifaceted and require a differentiated approach. The patient's needs may range from reassurance and basic investigations to exclude treatable structural causes for meal-related symptoms, to comprehensive clinical assessment and holistic treatment approaches. This includes a multi-professional team of gastroenterologists, dietitians, psychologists/psychiatrists and nursing staff. Consideration needs to be given to definitively ruling out other conditions, which may lead to conscious or subconscious food avoidance (see below under diagnostic approaches), and the impact of prescribed/non-prescribed pharmaceuticals and substances. At the same time, patients with altered food intake should be screened using validated age-appropriate tools for psychiatric conditions, including eating disorders, to ensure that these patients receive the appropriate treatment.

Clinical presentation

Patients with DGBI often experience a range of eating- or meal-related symptoms that subsequently significantly impact their eating behaviours and overall well-being²¹. In patients with FD, particularly in those diagnosed with postprandial distress syndrome (PDS), it is common to report symptoms such as post-prandial pain, early satiety and abdominal discomfort²².

FD, which etymologically means "bad digestion," is characterised by gastrointestinal symptoms triggered by a meal. This condition is predominantly observed in two, frequently overlapping subtypes: pure PDS, and the PDS-Epigastric Pain Syndrome (EPS) overlap, which together account for 82% of all FD cases. Australian population-based cohorts²³ reveal that subjects with symptoms consistent with FD frequently experience weight loss when compared to subjects without dyspepsia. 25-30% of subjects with severe FD symptoms reported ≥ 3 kg weight loss in the preceding three months as compared to 10% in subjects with minimal or no FD symptoms. Symptoms such as nausea, vomiting and meal-related complaints, including postprandial fullness, were strongly associated with significant weight loss. Another study revealed that factors contributing to disordered eating patterns resulting in weight loss also include psychological distress and symptom severity²⁴.

Meal-related symptoms may not only manifest in patients with FD but also in patients with IBS. In a study involving adolescents with IBS²⁵, 92% reported experiencing symptoms associated with eating, and among them 95% attributed symptoms to specific foods. Notably, some patients went so far as to avoid these trigger foods entirely, even to the point of not eating when they were hungry. Thus, in a subgroup of patients, severe symptoms prompted changes in their eating habits, modifying and reducing their food intake, and ultimately resulting in weight loss and experiencing other effects such as social isolation and broader effects on quality of life. Data from a recent population-based study from Norway shows that perceived food intolerance is highly prevalent (70%) and has considerable consequences in subjects with IBS²⁶. A recent systematic review⁶ highlights that disordered eating is common in patients with organic gastrointestinal disorders, affecting between 13%–55% of patients. However, higher rates were even observed in patients with DGBI compared to those with organic GI disorders.

To assist in the routine clinical setting with the recognition of patients with '*DGBI and altered food intake*' it is recommended to apply the established Rome Criteria for DGBI^{1, 27} (e.g., criteria for functional dyspepsia with postprandial distress²⁸) in combination with clinically significant weight loss (e.g., $> 5\%$ of body weight during a time period of six to 12 months).

Delineation of DGBI and Altered Food Intake and Eating Disorders.

In the context of patients presenting with altered food intake, it's important to understand that not all presentations of DGBI and altered food intake indicate the presence of a DSM-5-defined eating disorder. *Anorexia Nervosa* is characterised by an intense fear of gaining weight and a distorted body image, leading to severe restriction of food intake and low body weight. Individuals may engage in

excessive exercise or the use of laxatives and other methods (e.g., vomiting) to control weight. *Bulimia Nervosa's* key features are recurring episodes of binge eating, followed by compensatory behaviors to prevent weight gain, such as vomiting, fasting, excessive exercise or misuse of laxatives. These patients may have a normal weight and an unhealthy focus on body shape. Like *Bulimia Nervosa*, patients with *Binge Eating Disorder* also have recurrent episodes of consuming large quantities of food. However, unlike bulimia, individuals do not regularly engage in compensatory behaviors, often leading to obesity and associated health issues. *Pica* involves the consumption of non-nutritive, non-food substances over a period of at least one month. This can include items like dirt, clay, paper or soap. It is often associated with developmental disorders or nutritional deficiencies. There is repeated regurgitation of food, which may be re-chewed, re-swallowed or spat out as occurs in rumination. *Pica* does not occur due to any medical condition or another eating disorder. *ARFID* is characterised by a lack of interest in eating or avoidance of certain foods, leading to significant nutritional deficiency or weight loss. It differs from anorexia as it does not involve body image concerns²⁹. Altered food intake is also a key feature of patients with traditional eating disorders such as anorexia nervosa, bulimia nervosa or *ARFID*. Based upon the DSM-5, *ARFID* is defined as ‘...an eating or feeding disturbance (e.g., apparent lack of interest in eating or food; avoidance based on the sensory characteristics of food; concern about aversive consequences of eating) as manifested by persistent failure to meet appropriate nutritional and/or energy needs..’³⁰ There are also now other specified feeding or eating disorders (OSFED) with symptoms that are similar to one or more eating disorders but do not meet all the criteria for these conditions³¹.

In individuals with DGBI, chronic or recurrent gastrointestinal symptoms related to eating often lead to or occur alongside changes in food intake behaviours. These symptoms may also precede the onset or development of an eating disorder. As a result, distinguishing between DGBI and an eating disorder can be challenging. Therefore, when assessing a patient with DGBI, clinicians should consider an eating disorder as either a differential or co-occurring diagnosis. Thus, it is important to note that DGBI symptoms do not themselves exclude a diagnosis of a primary eating disorder. The two can co-exist and the temporal relationship between gastrointestinal symptoms and abnormal eating behaviour can sometimes be difficult to determine retrospectively.

Indeed, more than 94% of patients with untreated Anorexia Nervosa meet criteria for FD, with the majority (88%) having symptoms consistent with PDS, and >50% also meet criteria for IBS³². Gastrointestinal symptoms in Anorexia Nervosa are also associated with psychological traits such as somatisation, perfectionism and neuroticism³³. Increased prevalence of symptoms consistent with DGBI are observed in other eating disorders as well³⁴. The severity of GI symptoms and number of DGBI diagnosis are influenced by the severity of eating disorder symptomatology, with those reporting severe eating disorder symptoms meeting criteria for more than three DGBI, while those with less severe eating disorder symptoms meeting criteria for one^{34, 35}. Furthermore, malnutrition and restrictive food intake behaviours themselves can cause meal related symptoms and lead to gastrointestinal

physiology changes such as delayed gastric emptying³⁶ and physiological changes which may also be seen in some patients with DGBI. Most patients with eating disorders experience some improvement in their GI symptoms with weight restoration^{37, 38}; however, DGBI diagnosis may change throughout the course of eating disorder treatment and GI symptoms can persist beyond eating disorder recovery for some³⁹. It remains unclear to what extent nutritional rehabilitation, reduction in eating disorders symptoms or improvement in pathophysiological processes in DGBI (regardless of eating disorders status) contribute to improved GI symptoms within this group³³.

Complicating the matter, standardised eating disorder screening questionnaire tools are likely to be invalid in patients with concurrent gastrointestinal conditions, overestimating the prevalence of disordered eating. For example, in a cohort study utilising a standardised screening tool for ARFID involving 289 adults with well-established organic GI conditions including achalasia, celiac disease, eosinophilic esophagitis and inflammatory bowel disease, 53.7% met current diagnostic criteria for ARFID⁴⁰. While a small proportion of this cohort may well have had ARFID, it is likely that gastrointestinal symptoms confound the diagnosis, highlighting the current limitations of such tools⁴⁰.

Differentiating between DGBI and, more specifically, FD with PDS, and eating disorders remains challenging. While overlap of DGBI and eating disorders is frequent, in patients with DGBI, the altered food intake is predated by meal-related gastrointestinal symptoms or the fear of experiencing such symptoms. It is therefore important to consider treating both conditions concurrently where uncertainty exists. Ultimately, which diagnostic label is affixed is less important than the treatment modalities available for the patient. For instance, some sufferers may find it more acceptable to engage with a diagnosis of DGBI and altered food intake rather than a formal eating disorder diagnosis. If an eating disorder is suspected, the patient should be appropriately assessed by a health care provider with experience in managing patients with eating disorders and/or referred to the appropriate provider for this patient cohort. **Table 2** summarises the characteristics of patients who meet the criteria for DGBI and altered food intake behaviour and those with other specific eating disorders.

Comorbidities in patients with DGBI and altered food intake behaviour

Anxiety and depression are highly prevalent psychological comorbidities found in more than 50% of patients with DGBI⁴¹⁻⁴⁴. A systematic review found that anxiety and depression, along with female sex, younger age, gastrointestinal symptom severity, and lower quality of life were associated with disordered eating⁶. Other comorbidities observed in DGBI and altered food intake that have increasingly been seen in young females include debilitating gastrointestinal symptoms such as severe abdominal pain with food intake, gastroesophageal reflux, intractable nausea and vomiting, and constipation⁴⁵.

These symptoms align with the spectrum of symptoms seen in patients with DGBI and are often linked to various systemic rheumatologic, immunologic and cardiovascular conditions. Notable among these are hypermobility spectrum disorders (HSD)⁴⁶, postural orthostatic tachycardia syndrome (POTS)⁴⁷,

mast cell activation syndrome (MCAS)^{48, 49} and median arcuate ligament syndrome (MALS)⁵⁰. Moreover, a growing body of literature highlights the co-occurrence of MCAS, POTS and HSD⁴⁵. Diagnosing these conditions presents significant challenges, largely because the underlying pathophysiology of each remains incompletely understood and the diagnostic criteria are continually evolving. The prevalence of these conditions in the general population varies, but they are generally considered rare. POTS affects approximately 0.1% to 1% of individuals, with higher rates in young women aged 15-50⁵¹. MCAS is estimated to impact 0.5% to 2% of the population, likely underdiagnosed due to its diverse symptoms. Ehlers-Danlos Syndrome (EDS) occurs in about 1 in 5,000 to 1 in 10,000 individuals, while hypermobility EDS may occur in 1 in 10,000⁵². MALS is rarer, affecting approximately 2 per 100,000 people⁵³, with many cases potentially undiagnosed. As highlighted above, these conditions are all associated with significant diagnostic challenges and underreporting, complicating efforts to determine their true prevalence rates. The pathophysiologic role of these comorbidities for the manifestation of otherwise unexplained gastrointestinal symptoms is not yet completely understood. However, it is possible that these conditions are linked to the manifestation of gastrointestinal symptoms via alterations of visceral sensory function and gastrointestinal dysmotility, further augmented by comorbid psychopathology⁴⁵. On the other hand, it is also possible there is an association but no causation. Analysing the association between HDS, MCAS, and POTS, an extensive review of the literature concluded that studies that proposed a relationship between these clinical entities are either biased or based on outdated criteria⁵⁴. The authors concluded that the ‘... reason behind the purported association of these entities stems from an overlapping pool of vague, subjective symptoms, which is inadequate evidence to conclude that any such relationship exists...’. While this conclusion may reflect a type II error, it is critical to ensure that established and scientifically validated criteria need to be used before an association between these conditions and DGBI with altered food is diagnosed⁴⁵.

Diagnostic approaches for patients with DGBI and altered food intake behaviour

Clinical assessment of patients with altered food intake

The initial evaluation of patients with suspected DGBI and altered food intake requires a thorough patient history covering the onset, duration and nature of food intake changes, along with related gastrointestinal or psychological symptoms and any co-morbidities, including past conditions or investigations indicative of DGBI.

Understanding the context of the altered food intake—whether it is due to nausea with or without vomiting, dysphagia, odynophagia, aversions, or nonspecific abdominal pain or discomfort—can provide crucial insights into the underlying condition. The physical examination should identify signs of malnutrition, dehydration or any underlying organic gastrointestinal diseases, including the presence of a palpable mass. Anthropometric measurements, including weight, height, BMI and body

composition are essential for assessing nutritional status. Weight loss or failure to gain weight needs to be objectively documented.

Patients with altered food intake due to meal-related gastrointestinal symptoms should undergo appropriate investigations to rule out organic gastrointestinal causes for their symptoms⁵⁵. If these tests are within normal limits, the likelihood of organic gastrointestinal diseases (e.g. coeliac disease) is significantly reduced. Depending on the diagnosis, further investigations may include endoscopy and cross-sectional imaging. Additionally, it is important to consider other conditions that may present with chronic or recurring severe symptoms associated with meals, such as chronic pancreatitis, gallstone disease or mechanical obstruction. Given the link between food intake and the manifestation of symptoms, it is crucial to consider the diagnosis of food allergies.

There are currently limited opportunities to diagnose food allergies reliably⁵⁶. Skin prick testing may determine if there is a cutaneous reaction to a particular food, while IgE sensitisation testing and oral food challenges offer additional diagnostic options. However, it is important to note that both skin testing and IgE sensitisation have limited sensitivity and specificity. Oral food challenges can be time-consuming and associated with a degree of risk, while most patients with DGBI report atypical food intolerances that are not associated with ‘traditional immune-mediated pathways’ such as IgE⁵⁷. Adverse gastrointestinal responses to food (or intolerance of food) also encompass compounds not considered antigenic such as fermentable carbohydrates and natural food chemicals⁵⁷. New technologies, such as endoscopic confocal laser microendoscopy, are promising approaches for identifying gastrointestinal adverse reactions to food⁵⁸.

A psychological assessment is essential in patients with suspected psychological comorbid conditions like anxiety and depression as they can significantly impact quality of life. Moreover, an exaggerated fear of experiencing pain and discomfort after a meal may adversely affect food intake. Validated instruments to assess gastrointestinal symptoms and screening for depression, anxiety, somatic complaints, gastrointestinal-specific visceral anxiety and pain catastrophising are beneficial^{44, 59-61} to evaluate the patient's condition. This is particularly important since symptom severity is relevant for differentiating eating disorders from other conditions. The DSM-5 criteria require that eating disturbance is not attributable to a concurrent medical condition or better explained by another mental disorder. However, consideration needs to be given that an eating disorder can be accompanied by a DGBI and vice versa.

For patients with a severe impact on quality of life and subsequently increased health care utilisation, a psychological/psychiatric assessment by a psychologist or psychiatrist with specific expertise in the management of patients with DGBI and/or eating disorders is preferable.

Diagnosis of DGBI and altered food intake behaviour

A set of defined criteria is necessary to effectively distinguish patients with DGBI and altered food

intake from other gastrointestinal disorders that may result in disordered eating with or without weight loss. According to the Rome IV criteria, DGBI encompasses various disorders where gastrointestinal symptoms arise from alterations of gastrointestinal function and/or the interaction between the gastrointestinal system and the central nervous system¹. Key features for many DGBI are chronic or relapsing abdominal pain, bloating, early satiety, fullness or altered bowel habits. Currently, altered (reduced) food intake in patients with DGBI can be operationalised as a consequence of a severe DGBI manifestation, often against the background of other comorbidities (e.g., anxiety/depression)^{41,62}. Thus, altered food intake should be clinically documented with the respective DGBI diagnosis. For the diagnosis, patients with chronic or relapsing gastrointestinal symptoms that impair quality of life and result in altered food intake leading to significant unintended weight loss (e.g. >5% of body weight) should have had appropriate diagnostic workup to rule out organic gastrointestinal conditions such as inflammatory bowel disease, microscopic colitis, coeliac disease and other organic gastrointestinal diseases⁶³. While the diagnosis of a DGBI can be made without a comprehensive diagnostic work-up, most patients with chronic or relapsing symptoms will ultimately have undergone one. If with weight loss there are no structural abnormalities (and absence of other alarm gastrointestinal symptoms), the diagnosis of a DGBI is justified.

For patients presenting with altered food intake, it is critical to assess for symptoms consistent with a DGBI. In the routine clinical setting, screening for gastrointestinal and extraintestinal symptoms using validated instruments is advisable. Utilising standardised questionnaires (e.g., the Structured Assessment of Gastrointestinal Symptoms, SAGIS) to evaluate gastrointestinal and extraintestinal symptoms offers several key benefits, including: a) consistent data collection across patients, which reduces variability in symptom interpretation; b) coverage of a wide range of symptoms, providing detailed insights that may be missed in traditional interviews; c) enabling patients to effectively articulate their symptoms, which enhances communication and involvement in care; d) establishing baseline measurements to evaluate treatment effectiveness during follow-up visits; and e) aggregated data that aid research and informs quality improvement measures within clinical practice.

In many cases, it is not clinically possible to determine whether the gastrointestinal symptoms preceded the altered food intake behaviour or emerged during the disordered eating behaviours. Regardless, the association between the gastrointestinal symptoms and the act of eating often reinforces food avoidance.

Diagnostic measures to identify altered gut function

The diagnosis of a DGBI and altered food intake is a clinical diagnosis based upon a typical pattern of symptoms consistent with a DGBI, where symptoms are aggravated by food and followed by a reduction in food intake, combined with the absence of relevant differential diagnoses. Function testing can be relevant for gaining insights into the underlying disease mechanisms but is not required to establish the diagnosis.

Gastric emptying testing

Food ingestion initiates gastric accommodation, which is subsequently followed by the formation of antral contractions. As the food is broken down into smaller particle sizes, mechanisms such as pyloric relaxation and the coordinated activity of the antropyloroduodenal region facilitate the emptying of the stomach.

Gastric emptying testing, which quantifies the emptying of a tracer from the stomach into the duodenum, can be a critical tool in diagnosing and managing conditions like gastroparesis and may provide guidance for the treatment of specific DGBI. Nuclear scintigraphy (four-hours with solid and liquid emptying) is widely considered the gold standard for clinical assessment of gastric emptying. However, breath testing utilising stable isotopes such as ^{13}C octanoic acid is comparable and allows gastric emptying measurements without use of radioactive markers⁶⁴. In addition to delayed gastric emptying, impaired fundic relaxation has been observed in patients with FD⁶⁵. Despite this association, no specific treatments are approved to address impaired fundic relaxation to alleviate symptoms in FD specifically. Gastric emptying is influenced by nutritional status / restrictive eating and hence the diagnostic utility of such tests are unclear in patients with DGBI and altered food intake, eating disorders or malnutrition^{66, 67}. Furthermore, pharmaceuticals (e.g., prescribed opioids, ondansetron, glucagonlike peptide-1 receptor agonists) and substances (e.g. non-prescribed opioids, cannabinoids) may influence gastric emptying⁶⁸⁻⁷⁰. Unless pharmaceuticals / substances affecting gastric emptying are withheld for a time exceeding 3-4 half-lives, gastric emptying studies may not be accurate or interpretable⁷⁰.

Symptoms of gastroparesis include nausea, vomiting, early satiety, postprandial fullness and bloating⁷¹. In a large study⁷² involving >300 patients with FD, delayed gastric emptying was detected in 33.5% of dyspeptic patients. Delayed gastric emptying was particularly frequent in female patients who exhibited characteristics such as low body weight, significant postprandial fullness, nausea, vomiting and a lack of severe epigastric pain. Conversely, these symptoms are not specific for gastroparesis. In a cohort of 425 patients with chronic nausea and vomiting, 106 were found to have normal gastric emptying⁷³. Patients with nausea and vomiting with non-delayed gastric emptying were largely indistinguishable from those diagnosed with gastroparesis. While altered gastric emptying poorly correlated with symptoms, it may serve as a marker for disordered gut function⁷⁴ and validate the concept that gut function is disordered in these patients with DGBI.

Nutrient challenge

The symptom response to a standardised nutrient challenge utilising 400⁵⁶ or 800 mL⁷⁴ is a simple approach to quantify sensory responses to nutrients. This appears to differentiate patients with DGBI from healthy controls and appears to be promising to assess treatment responses in patients with DGBI and meal-related symptoms. While this test can be easily implemented, it is currently not widely used.

Transit studies

Several methods²⁴ are available to assess small intestinal transit in the clinical setting⁷⁵. These include hydrogen or methane breath tests after ingestion of a carbohydrate substrate (e.g., lactulose), scintigraphy techniques and novel approaches, including wireless motility capsules that have been developed and validated to measure small-bowel and colonic transit⁷⁵. While transit studies can provide information about a specific gastrointestinal function, their relevance for the routine assessment and management of patients with DGBI and altered food intake remains to be determined.

Body surface gastric mapping

Body surface gastric mapping is an innovative diagnostic tool used to assess patients with DGBI. This technique involves visualising and mapping gastric activity by recording electrical signals from the stomach's surface. Body surface mapping contributes to management decisions in cases of intestinal failure and suspected neuromuscular disorders and allows for a focus on gut-brain interaction in subjects with normal tests⁷⁶.

Management strategies for patients with DGBI and altered food intake behaviour

Importance of an Integrated Multidisciplinary Team (iMDT) Approach

While many patients with DGBI (both those with and without food-related symptoms) receive appropriate treatment in primary care, patients with severe DGBI manifestations (characterised by severe symptoms, significant impact on quality of life and frequent healthcare utilisation), may not receive the required support in the primary care setting. Data from a cohort study indicated that these patients consequently turn to alternative, non-evidence-based sources of information including medical and dietary information⁷⁷. Patients with severe symptom manifestations require management that involves specific expertise and preferentially input from a multidisciplinary team. Because altered gut-brain interactions and impaired gut function play a crucial role in symptom manifestation and potential medical complications, the specialised knowledge of gastroenterologists and allied health professionals is essential for the care of these patients (and appropriate models of care for patients in rural and remote regions of Australia and New Zealand remain a challenge). Addressing psychological comorbidities (e.g., depression/anxiety) requires the involvement of appropriately trained healthcare specialists such as psychologists and psychiatrists (and if available an eating disorder specialist). Long-term management should be coordinated by a primary care physician and guided by the expert team in a hub-and-spoke model. Effective medication management and medical monitoring are the responsibilities of the gastroenterology team, which collaborates closely with primary care physicians. Insights from managing patients with complex DGBI indicate that many individuals can benefit from psychotherapeutic interventions. This aspect of care should be addressed by clinicians who are credentialed to provide psychotherapy, with trained psychologists often being the most suitable professionals for this role. Nevertheless, other qualified healthcare providers can also contribute to this

crucial aspect of treatment. Furthermore, dietitians play an essential role in assessing the patient's nutritional status, knowledge, motivation, current eating behaviours and dietary intervention strategies. They develop and implement the nutritional component of the treatment plan, providing ongoing support to help patients achieve their goals and achieve sustainable dietary changes⁷⁸.

Dietary interventions, enteral feeding and parenteral nutrition

Dietary interventions are key in managing DGBI and related symptoms. Dietitians conduct detailed dietary assessment including identification of dietary habits that may influence symptoms and use this to implement appropriate dietary interventions. In patients with altered food intake, non-restrictive dietary interventions such as the National Institute of Clinical Excellence diet or Mediterranean diet are more appropriate than restrictive dietary interventions⁷⁹. Restrictive diets are contra-indicated in patients with altered food intake behaviour and malnutrition. While a low Fermentable Oligosaccharides, Disaccharides and Polyols (FODMAP) diet is commonly used to manage symptoms of IBS, its effects on those with DGBI and altered food intake remain unclear. A low-FODMAP diet without a plan for food reintroduction could be harmful over the long term^{80, 81} and should not be continued for more than 4-6 weeks⁸². If restrictive dietary interventions are considered in patients with altered food intake behaviour without malnutrition, patients should be carefully reviewed by an experienced dietitian and monitored and modified based upon the clinical response. There is a delicate balance between symptom control and nutritional inadequacy which can lead to nutritional deficiencies and disordered eating⁸³. This is particularly the case for patients who have self-implemented diets; patients might avoid whole food groups with a subsequently more significant impact on nutritional health⁸⁴. To address this, incorporating specific dietary supplements may be recommended under close medical supervision, ensuring that patients receive essential nutrients while still adhering to their individualised dietary protocols. Ultimately, a well-structured dietary plan can empower patients to manage their conditions better and improve food variety and quality of life. Dietitians play an important role in patients with altered food intake to rebuild food range and diet diversity, using structured approaches such as personalised education, nutrition counselling, gradual food reintroduction, meal planning with a variety of ingredients, and incorporating food exposure techniques.

Enteral nutrition

In malnourished patients with DGBI who cannot achieve adequate oral intake due to gastrointestinal symptoms but have a functioning gastrointestinal tract, it might be tempting to initiate enteral tube feeding. The European Society for Clinical Nutrition and Metabolism (ESPEN) Clinical Guidelines for Home Enteral Nutrition defines an inadequate nutritional state as a patient's inability to eat for a week or energy intake <60% of estimated requirements for 1-2 weeks (usually <10 kcal/kg/d or a lack of 600-800 kcal/d)⁸⁵. However, caution is advised when commencing enteral feeding in patients with DGBI without malnutrition or objective biochemical abnormalities. In these cases, nutritional support via tube feeding or parenteral nutrition can carry significant risks of iatrogenesis, including potential

exacerbation of symptoms, psychological dependence on enteral feeding and complications related to tube-feeding⁷¹. There is also a lack of evidence for improved global function, quality of life or symptoms with enteral feeding in this population⁸⁶⁻⁸⁸. Indeed, a recent study from St Mark's (England, United Kingdom) showed no weight gain in patients with DGBI who remained long-term on enteral tube feeding. In contrast, those who could discontinue tube feeding showed significant improvements⁸⁸. There were similar results from Melbourne in a cohort of DGBI patients on long-term enteral nutrition; 86% had no improvement in gastrointestinal symptoms, and less than half experienced any weight gain⁸⁷.

Thus, enteral (tube) feeding in patients with DGBI and altered food intake is only justifiable under exceptional circumstances in malnourished patients when other measures have been exhausted and appropriate treatment by a multidisciplinary team has failed to address a life-threatening situation. If enteral nutrition is considered in these patients, it is a short- to medium- term (days or up to 4 weeks) measure aimed at stabilising nutritional status, preventing further decline and supporting a transition to adequate oral intake⁸⁹. If enteral feeding does not achieve weight gain and nutritional optimisation, it should be stopped and other treatment strategies in a multi-disciplinary context should be considered. Invasive interventions such as feeding via a percutaneous gastrostomy or jejunostomy should always be preceded by more conservative approaches to avoid escalation to interventions with a greater risk of iatrogenesis⁹⁰ while effects of long-term treatment are widely lacking. Examples for appropriate interventions include dietary counselling and oral dietary adjustments, which may include oral nutrition supplements to maximise *effortful oral feeding*, or, under exceptional circumstances, time-limited naso-enteral tube trials of 4-6 weeks with maximal multidisciplinary input to optimise effective weaning to an oral diet^{85, 86, 89, 91}.

Given the lack of evidence that enteral feeding improves symptoms in DGBI, ongoing enteral feeding for symptom management alone cannot be recommended⁸⁶⁻⁸⁹. Severe complications such as aspiration pneumonia, severe and intractable gastrointestinal symptoms preventing the administration of enteral nutrition or feed tolerance and frequent tube-related complications such as infection or hypergranulation of tissue necessitate a thorough reassessment and may require the discontinuation of enteral feeding strategies⁹². While adult-specific weaning protocols are under-researched, the paediatric literature highlights the importance of a multidisciplinary team approach⁹¹. These principles may be adapted for adults into a structured, multidisciplinary approach to gradual oral diet reintroduction, prioritising sustained oral intake and addressing barriers such as food intolerances, appetite, feeding behaviours, sensory factors and psychological readiness^{89, 91, 93}. Dietitians as members of the multidisciplinary team that also includes gastroenterologists, psychologists and other allied health staff such as exercise physiologists⁴² play a critical role in individualising care plans, monitoring progress, including objective markers of nutrition status, and providing ongoing support throughout this process⁸⁹.

Parenteral nutrition

Parenteral nutrition is a well-established life-saving treatment indicated in patients with intestinal failure (IF)^{94,95} since PN bypasses the gastrointestinal tract, allowing the provision of nutrition in patients who have compromised digestive systems⁹⁶. All currently established definitions of IF, including the 2015 definition published by the European Society for Clinical Nutrition and Metabolism⁹⁵, are based upon the IF definition provided by Fleming and Remington in 1981, who stated that IF is defined by a reduction in the functioning gut mass below the minimal amount necessary for adequate digestion and absorption of food⁹⁷.

The functional classification of intestinal failure applies various criteria, including duration, metabolic state and expected outcome⁹⁵ of the underlying cause of IF⁹⁸. Type I is considered an acute intestinal failure, often short-term and self-limiting. Type II is a sub-acute intestinal failure, usually seen in metabolically unstable patients, and may continue for weeks to months. Type III is a chronic intestinal failure, usually in stable patients who usually require home parenteral nutrition for months to years⁹⁵. While appropriate use of PN can be lifesaving, the use of PN is not only burdensome and costly to patients and payers but can also cause significant complications. They include risks of infections associated with catheter use, metabolic derangements and potential liver disease due to long-term reliance on intravenous nutrient delivery⁹⁹. PN also resulted in a reduction of the thickness of the gastrointestinal muscularis mucosa thickness and marked gut atrophy¹⁰⁰. This was associated with alterations of key gut-systemic signalling regulators. The Liver farnesoid X receptor, liver constitutive androstane receptor, gut FXR, G-coupled bile acid receptor (TGR5), epidermal growth factor, organic anion transporter, Mitogen-activated protein kinases-1, and sodium uptake transporter sodium glucose-linked transporter were all significantly downregulated in animals treated with PN¹⁰⁰. These changes likely explain the adverse outcomes observed in patients receiving long-term PN^{95, 97, 101}.

Therefore, while PN serves a vital role in managing intestinal failure, it is not an appropriate treatment modality for patients who retain normal gastrointestinal function, where oral or enteral feeding should be prioritised to support digestive health and maintain physiological gut function¹⁰².

Indeed, consistent with the above considerations, a position paper from the European Society of Clinical Nutrition and Metabolism, the European Society of Neurogastroenterology and Motility, and the Rome Foundation for Disorders of Gut–Brain Interaction articulated the consensus opinion¹⁰³ that long term (home) PN should not be prescribed for patients without IF, where the oral and/or enteral route can be utilised. The authors clarify that there might be rare occasions when PN commencement is required to treat life-threatening malnutrition in conditions without IF.

Similarly, the Small Bowel and Nutrition Committee and the Neurogastroenterology and Motility Committee of the British Society of Gastroenterology states that significant caution should be exercised to avoid escalating to more invasive forms of nutrition support in patients with functional symptoms,

especially in pain predominant presentations, in the absence of objective features of biochemical disturbance or those who have a high or normal BMI”⁹³.

Thus, PN should only be prescribed in exceptional circumstances for a time-limited period to achieve nutritional safety, while the wider multi-disciplinary team focuses on appropriate biopsychosocial holistic and rehabilitative approaches to manage the patient's primary underlying condition.

Pharmacologic and Psychological interventions

If reassurance and dietary interventions fail, pharmacotherapy, psychotherapy and behavioural interventions, specifically brain-gut behaviour therapies¹⁰⁴ are typically considered for patients with DGBI. In the absence of controlled trials specifically addressing altered food intake behaviour in patients with DGBI, findings from other DGBI studies can inform treatment, though they should be used with caution. The data presented here reflects the best available guidance for managing these patients.

Prokinetics

In the clinical setting, various prokinetics including metoclopramide and domperidone are widely used and have been shown to improve symptoms in patients with FD¹⁰⁵. This aligns with findings from clinical studies⁷⁴, which indicate that altered gastric emptying serves as a marker for DGBI, such as FD (including EPS and PDS). However, pharmacotherapies that selectively accelerate and normalise gastric emptying do not necessarily improve symptoms¹⁰⁶ and a previous systematic review and meta-analysis did not establish the effectiveness of prokinetics in improving quality of life¹⁰⁷

Psychotropic drugs

Psychotropic drugs (or neuromodulators) drugs have shown efficacy in alleviating visceral pain and improving quality of life in patients with IBS and FD, which often present with meal-related symptoms¹⁰⁸. However, in a recent systematic review, only tricyclic antidepressants (TCAs), but not serotonin reuptake inhibitors (SSRIs) were found to be effective^{109, 110}. With regard to the use of TCAs in patients with DGBI, while effective in low doses, effects may only occur after months of treatment and are often associated with side effects that may lead to early treatment discontinuation. This needs to be taken into account when TCAs are prescribed and patients should be educated accordingly¹¹¹.

Special consideration can be given to the psychotropic drug mirtazapine which has been used to treat patients with depression and anxiety disorders. It has antagonistic effects on histamine receptors H1, α_2 -adrenergic receptors, 5-HT_{2C}, and 5-HT₃ receptors, and it has been reported to improve dyspeptic symptoms (inhibits nausea and vomiting), and based upon a small clinical trial has good therapeutic effects in patients with FD and weight loss¹¹². In a randomised, single-blinded study, 8-week treatment with mirtazapine significantly improved the overall severity of dyspeptic symptoms (postprandial fullness, early satiation, nausea and vomiting), gastrointestinal-specific anxiety, quality of life and

increased weight in FD patients¹¹³.

Spasmolytics

Peppermint oil is considered beneficial for patients with DGBI based on studies in patients with FD¹¹⁴ and IBS¹¹⁵. The active ingredient in peppermint oil, menthol, relaxes smooth muscles via calcium channel blockade or direct enteric nervous system effects; in addition, it has effects on visceral sensitivity (via transient receptor potential cation channels)¹¹⁶. Despite its benefits, peppermint oil should be used with caution, especially in individuals with gastroesophageal reflux disease (GERD), as it may exacerbate symptoms. Another antispasmodic used for patients with IBS and meal-related pain is Mebeverine¹¹⁷. A recent systematic review and meta-analysis concluded that Mebeverine is an effective treatment option in IBS, and more specifically reported effects on pain and discomfort¹¹⁸. However, the studies have not been specifically designed for patients with DGBI and altered food intake behaviour.

Psychotherapeutic Interventions

Given that patients with DGBI and altered food intake behaviour can experience thoughts, feelings and behaviours which result in heightened anxiety/stress and low mood related to food intake, incorporating psychotherapeutic strategies is essential. Psychological therapies can assist in managing these health-related comorbidities and can also be used to understand and modify the underlying belief systems, which can potentially amplify symptom severity. Several psychological therapies have been used in DGBI with largely equivalent efficacy (RR 0.71-0.79)¹¹⁹ with Cognitive Behavioural Therapy (CBT) and gut-directed clinical hypnotherapy having the most substantial evidence base for IBS^{119 120}. CBT is frequently part of multidisciplinary treatment approaches for patients with DGBI^{121, 122}.

CBT for DGBI involves enhancing patient skills in managing elevated health-related anxiety and stress, for example, in IBS^{119 120}. CBT typically involves assisting the patient in challenging negative automatic thoughts, behavioural avoidance and anticipated GI-related pain and/or other negative sensations to better manage mental health symptoms. Other CBT-based approaches can target the perceived cause of the disorder through exposure-based techniques that gently and gradually, under medical supervision, expose patients to food stimuli believed to provoke their symptoms^{123, 124}. Pilot data for patients with DGBI and altered food intake behaviour have been published, demonstrating the feasibility of this treatment approach¹²⁵.

Gut-directed Hypnotherapy is based on principles of traditional psychologically-based cognitive and muscle relaxation techniques, but with the addition of hypnotic suggestions from the clinician targeted to individualised gut symptoms, promoting better control (either manage or distract) or reduction of unpleasant symptoms¹²⁶. Efficacy has been tested mainly in IBS, and the quality has been variable across these studies¹²⁰.

Often used in conjunction with CBT, *Mindfulness-based therapies* have been more recently introduced

as a possible psychological treatment for DGBI to promote stress relief and may alter the perception of gastrointestinal symptoms. Mindfulness-based approaches focus on awareness and acceptance of symptoms, whereas CBT typically assesses and challenges negative and dysfunctional thought processes. The effectiveness of Mindfulness-based therapies is similar to CBT in the management of anxiety disorders in general populations⁸⁵, but further research is required in DGBI. Of the studies conducted in IBS cohorts, data suggest that mindfulness-based stress reduction training was associated with improvements in gastrointestinal symptoms¹²⁷⁻¹²⁹. Another evidence-based psychological intervention that utilises mindfulness and found to be efficacious in reducing IBS symptoms and anxiety/depression is Acceptance and Commitment Therapy (ACT)^{130, 131}. ACT is based on strategies that promote psychological flexibility, such as mindfulness and acceptance, to help individuals move toward their values rather than remain stuck in negative cycles of thinking and behaviour.

While the treatment of DGBI patients with altered food intake involves a combination of general measures, reassurance, dietary interventions, pharmacotherapy and psychotherapeutic interventions, there is lack of clinical trials for this specific patient cohort. Dietary interventions, prokinetics, neuromodulators, and tailored psychological support are promising avenues for symptom relief. Future research is warranted to establish definitive treatment protocols for this underserved population.

Specific considerations for children and adolescents

In children with feeding problems, disordered eating behaviours and DGBI are all common¹³²⁻¹³⁴.

The most typical underlying causes of feeding problems relate to other medical conditions, neurodevelopmental and physical disability and behaviour¹³⁵. Feeding problems indeed appear to be more prevalent in younger children than older children and adolescents, where functional rather than organic feeding disorders appear to be commoner^{133, 136}.

Due to the ongoing developmental processes of adolescence, DGBI and eating disorders can be even more difficult to differentiate, in part due to evolving levels of insight from the young person and the impact of parent-child relationships. Clinical experience identifies a number of adolescents with complex DGBI and altered food intake that have a more definite eating disorder presentation after transitioning to adult care. A UK cohort study links childhood recurrent abdominal pain with later disordered eating behaviour¹³⁷.

Moreover, in childhood and adolescence, rumination has been identified as an important mechanism of altered food intake, with the regurgitation often overlapping with dyspepsia rather than being an isolated symptom. In a study of 76 children with rumination identified as the physiology of persistent fore-gut symptoms, 35% had weight loss¹³⁸. Programs targeted at managing rumination in children and adolescence have been shown to facilitate weaning of artificial nutritional support and delay in diagnosis is associated with decreased treatment success¹³⁹. For this reason, early consideration of this entity in children with persistent regurgitation and “vomiting” is important.

Unlike in adulthood where acute malnutrition is of the most importance, chronic malnutrition in childhood and adolescence can have significant or permanent complications relating to puberty, growth, development and bone mineralisation. Although artificial feeding methods remain best avoided as described above, these considerations add complexity to decision making around the mode and duration of nutritional support. Most importantly, they further highlight the need for referral to tertiary multi-disciplinary teams as well as the need for additional research. Furthermore, issues of informed consent, patient autonomy and duty of care are also made more complex by the developmental stage of children and adolescents and the parent-child relationships

Altering diets to improve or resolve symptoms is extremely common across the spectrum of paediatric DGBI¹⁴⁰. Some studies show that up to 93% of DGBI patients identify at least one food and/or food type as worsening gastrointestinal symptoms, without evidence of food allergy^{21, 141 5, 142, 143}. As a result, children with functional abdominal pain disorders or their health practitioners frequently implement additional dietary restrictions or exclusions^{144, 145}. In a retrospective study from Texas, USA of almost 300 children meeting diagnostic criteria for pain-predominant functional gastrointestinal disorders, 43% received medical nutrition therapy, although there was significant variability in the diet guidance provided¹⁴⁶.

ARFID is also recognised to occur in children and adolescents. In a Canadian study, the incidence of ARFID was highest among adolescents 10 to 14 years of age (3.43 per 100000 patients) and girls 15 to 18 years of age (2.24 per 100000 patients)¹⁴⁷. While a high prevalence of ARFID has been reported in children with DGBI, especially FD¹⁴⁸, with some studies suggesting that almost a quarter of such patients present with symptoms consistent with ARFID¹⁴⁹ it is reasonable to assume that altered food intake in paediatric patients with DGBI can be misdiagnosed as eating disorders including ARFID. This raises concern that the use of exclusion diets may put DGBI patients at risk of impaired nutrition, disordered eating behaviours or eating-related quality of life¹⁵⁰.

Therefore, before dietary interventions are recommended, a careful assessment of the child's and family's situation and dietary intake needs to be performed¹⁸. Recommending a more restrictive diet when excessive food restriction is already in place may be inappropriate not only from the perspective of nutritional requirements^{151 149}. Further careful consideration should also be given to the route of administration of feeds. Although the paediatric literature is lacking, it needs to be noted that the use of enteral, or especially parenteral, nutrition in adults was not associated with long-term improvements in symptoms or nutrition but related to complications⁸⁷. In paediatric DGBI, such feeding routes should not be routinely considered.

For managing paediatric patients with DGBI and altered food intake behaviours, a multidisciplinary integrated care model is recommended, ideally consisting of a dietitian, psychologist or psychiatrist, general paediatrician and/or physician, and gastroenterologist. In particular, a general paediatrician should assess patients under 12 years, while an adolescent medicine physician can be involved for ages

13 to 18. Adolescent physicians can address broader psychosocial and mental health factors, including family dynamics, school relationships and peer interactions, and coordinate care with school wellbeing coordinators, community clinicians, National Disability Insurance Scheme (NDIS) case managers and GPs. In this model, each clinician performs individual assessments during an extended clinic session. The team then collaborates to form a comprehensive diagnosis, which is communicated to the family along with a tailored management plan. This unified approach builds patient trust, enhances engagement and reduces team fragmentation.

Managing expectations of consumers

Patients affected by severe DGBI and altered food intake often struggle to maintain work, study and social interactions and suffer high rates of distress and mental illness⁴³. At the same time, patient expectations are increasingly shaped by health information obtained from the internet. Most of this information is not backed by scientific evidence. Still, it has a significant influence with anecdotal stories that resonate deeply with vulnerable individuals who may feel desperate and isolated due to their chronic illnesses. While peer support groups aim to offer understanding and support, they can sometimes inadvertently spread misinformation or promote treatments that may be inappropriate or even harmful for patients. In recent years, many patients display a strong sense of identity and belonging to online peer groups through their illness, termed social media-associated abnormal illness behaviour^{152, 153}. In the absence of structural abnormalities and with the frequent dismissal of these disorders as being caused by psychiatric conditions, patients face stigma that can lead to emotional distress, difficulties in accessing appropriate care and exacerbated symptoms. This can cascade into a search for organic causes that may further complicate care, especially in fragmented healthcare settings where patients may consult multiple healthcare providers over time. At the same time, there is unwillingness to explore the role of psychological and behavioural factors, but rather a focus on structural pathology, invasive testing and treatment. This is associated with increased healthcare utilisation and risk of iatrogenic harm¹⁰³. This situation is not only stressful for patients but also presents a significant challenge for individual healthcare providers. Guided by the principle '*Primum non nocere*' (first do no harm), healthcare providers can become pressured by patients with ill-informed (sometimes social media-informed) preconceived management strategies. In this context, establishing a strong rapport with patients is essential. Healthcare providers should prioritise active listening to fully understand patients' concerns and validate their feelings while also guiding them toward evidence-based information. When myths or misconceptions arise, it's important to address them respectfully and thoughtfully. The focus must be on patient-centred education and communication. Presenting factual, evidence-based results can effectively counter false information without alienating the patient. Involving patients in the decision-making process surrounding their care is vital. This collaborative approach fosters trust and ensures that patients are aware of available clinical guidelines. Additionally, seeking a second independent opinion from another healthcare professional can be beneficial. This step

reinforces the appropriateness of the care provided and protects healthcare providers by ensuring that they adhere to clinical standards, manage patient expectations effectively, and minimise the risk of harm.

All patients can reasonably expect their treating team to provide a thorough assessment, a precise diagnosis and an individualised treatment plan, as well as respectful discussions surrounding treatment options that take into account their preferences and any psychosocial barriers. They can also expect timely reviews, ongoing support and management by the involved healthcare providers. A key role is played by the primary care physician, who needs to be well-informed regarding the management plan to ensure appropriate support in the primary care setting.

Knowledge gaps and future research directions

Despite significant advancements in our understanding of DGBI and their intersection with eating disorders, significant knowledge gaps remain that hinder effective clinical management of patients with DGBI and altered food intake behaviour. Addressing these gaps will enhance our understanding of the relationship between gastrointestinal and psychological disorders, leading to improved patient outcomes. Key areas that require further exploration include:

Clinical assessment: Validation of existing diagnostic criteria and development of standardised assessments to appropriately guide therapy and characterise symptoms and impact on patients' quality of life with DGBI and altered food intake.

Risk factors for the progression: Patients presenting with DGBI and altered food intake likely present only a fraction of patients with meal-related symptoms. Thus, risk factors for the progression of DGBI complicated by severely altered food intake and nutritional deficiencies need to be identified.

Development and validation of screening tools and cut-offs: The development and validation of screening tools specific to this DGBI population are critically needed. These tools should include clinically relevant cut-offs to improve the accuracy of assessments and facilitate early detection of disorders. Effective screening instruments will ensure timely intervention, which is crucial for individuals experiencing complex interactions between gastrointestinal and psychological symptoms.

Define the role of comorbidities in disease progression: It is essential to better understand the role of comorbidities, including psychiatric comorbidities, in manifesting, progressing and responding to therapeutic interventions. Research should aim to identify specific comorbid conditions, their prevalence, and how these relationships change over time. Recognising predictors of these comorbidities can inform targeted interventions and management strategies tailored to each patient's psychological needs.

Adapt and validate therapeutic interventions (medical, dietary, psychological) utilising appropriately powered and designed clinical studies to address the unique needs of these patients.

Define long-term outcomes of patients who utilised enteral or parenteral feeding or continued oral feeding by studying clinical cohorts to inform the future development of prospective randomised clinical trials.

Conclusions

In recent years, there appears to be an increasing number of adult and paediatric patients with severe meal-related symptoms and subsequently altered (reduced) food intake. Some of these patients may experience weight loss and nutritional deficiencies. These patients frequently meet established criteria for DGBI (and in particular FD) while eating disorders (including ARFID) need to be considered as a differential diagnosis (or co-morbidity).

Like many other patient groups with DGBI, these patients may also suffer from psychological comorbidities such as depression and anxiety or systemic disorders such as hypermobility spectrum disorders (HSD), postural orthostatic tachycardia syndrome (POTS), mast cell activation syndrome (MCAS), and median arcuate ligament syndrome (MALS). Currently, there is an absence of high-quality data to support the relationship between these disorders and gastrointestinal symptoms. Thus, these comorbid conditions should be validated against established diagnostic standards, with their relationship to meal-related symptoms noted, considering that the true relevance for the GI symptoms remains to be defined. While these comorbidities may complicate the clinical picture, it is crucial not to overlook the primary issue: the patient's inability to tolerate food intake due to a DGBI.

These patients experiencing severe meal-related symptoms, while the structural abnormalities do not explain the symptoms, face a stigma that can lead to emotional distress, difficulties in accessing appropriate care, and this vicious circle may exacerbate symptoms. This can cascade into a search for organic causes that may further complicate care, especially in fragmented healthcare settings where patients consult multiple specialists over time. A lack of coordinated care increases the risk that well-meaning medical or dietary interventions may cause harm and further exacerbate the situation. Effective management begins by making a positive diagnosis of a DGBI by ruling out organic causes, adhering to Rome Criteria and evaluating for eating disorders where appropriate.

Key components of long-term management include empathetic patient care, education, realistic expectation setting and a collaborative treatment plan, requiring a multidisciplinary team that provides input from gastroenterologists, dietitians and mental health specialists, at least for patients with severe manifestations.

Both pharmacological and non-pharmacological treatment options should be tailored to the predominant symptoms and previous treatment responses. Pharmacotherapy targets may include the gastrointestinal tract, the enteric nervous system or the central nervous system. Non-pharmacological strategies should follow established DGBI treatment principles and may involve cognitive behavioural therapy or similar approaches.

Invasive treatments like gastric or post-pyloric tube feeding or parenteral nutrition should be avoided unless necessary to address short-term, life-threatening, severe nutritional deficiencies. If used to address dietary deficiencies, these treatments should be as brief as possible in a setting that provides the required multidisciplinary support from gastroenterologists, dietitians and mental health specialists. If a patient fails to engage with the treatment options offered, consideration might be given to discharging this patient into the care of the primary care physician with a treatment plan. This decision will be made carefully, considering relevant medical, psychological and medico-legal factors to ensure it is a safe and appropriate choice and this needs to be accompanied by appropriate information to the patient and the primary care physician.

Considering the burden to patients and society from these conditions, more research is required to characterise the disease mechanisms better and develop and assess therapeutic interventions that ultimately can restore an everyday life for affected patients with DGBI.

For patients who frequently present with severe or very severe manifestations, providing a multimodal treatment approach by a multidisciplinary team of gastroenterologists, dietitians, psychologists and as clinically appropriate psychiatrists and other health professions who have preferably experience managing patients with complex DGBI is important. However, it is important to be vigilant about the overlap of DGBI and altered food intake behaviour and eating disorders. If there is a suspicion of an eating disorder, a referral to a specialised eating disorder service should be strongly considered. While clinical trials exploring the safety and efficacy of specific treatments for these patients are scarce, it is reasonable to extrapolate the available evidence for patients with DGBI. All treatments must be closely coordinated with primary care to support optimal long-term management of these patients within the primary care setting. This guideline and associated education will increase awareness about severely reduced oral food intake due to meal-related symptoms in DGBI.

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References

1. Drossman DA, Hasler WL. Rome IV-Functional GI Disorders: Disorders of Gut-Brain Interaction. *Gastroenterology* 2016;150:1257-61.
2. Drossman DA, Hasler WL. Rome IV—functional GI disorders: disorders of gut-brain interaction. *Gastroenterology* 2016;150:1257-1261.
3. Atkins M, Burton Murray H, Staller K. Assessment and management of disorders of gut–brain interaction in patients with eating disorders. *Journal of Eating Disorders* 2023;11:20.
4. Lim HS, Kim SK, Hong SJ. Food Elimination Diet and Nutritional Deficiency in Patients with Inflammatory Bowel Disease. *Clin Nutr Res* 2018;7:48-55.
5. Reed-Knight B, Squires M, Chitkara DK, et al. Adolescents with irritable bowel syndrome report increased eating-associated symptoms, changes in dietary composition, and altered eating behaviors: a pilot comparison study to healthy adolescents. *Neurogastroenterol Motil* 2016;28:1915-1920.
6. Peters JE, Basnayake C, Hebbard GS, et al. Prevalence of disordered eating in adults with gastrointestinal disorders: A systematic review. *Neurogastroenterol Motil* 2022;34:e14278.
7. Crone C, Fochtmann LJ, Attia E, et al. The American Psychiatric Association Practice Guideline for the Treatment of Patients With Eating Disorders. *Am J Psychiatry* 2023;180:167-171.
8. Position of the American Dietetic Association: nutrition intervention in the treatment of anorexia nervosa, bulimia nervosa, and eating disorders not otherwise specified (EDNOS). *J Am Diet Assoc* 2001;101:810-9.
9. Hay P, Chinn D, Forbes D, et al. Royal Australian and New Zealand College of Psychiatrists clinical practice guidelines for the treatment of eating disorders. *Aust N Z J Psychiatry* 2014;48:977-1008.
10. Murphy MK, Black NA, Lamping DL, et al. Consensus development methods, and their use in clinical guideline development. *Health Technol Assess* 1998;2:i-iv, 1-88.
11. Lacy BE, Pimentel M, Brenner DM, et al. ACG Clinical Guideline: Management of Irritable Bowel Syndrome. *Am J Gastroenterol* 2021;116:17-44.
12. Savarino E, Zingone F, Barberio B, et al. Functional bowel disorders with diarrhoea: Clinical guidelines of the United European Gastroenterology and European Society for Neurogastroenterology and Motility. *United European Gastroenterol J* 2022;10:556-584.
13. Vasant DH, Paine PA, Black CJ, et al. British Society of Gastroenterology guidelines on the management of irritable bowel syndrome. *Gut* 2021;70:1214-1240.
14. Barbara G, Cremon C, Bellini M, et al. Italian guidelines for the management of irritable bowel syndrome: Joint Consensus from the Italian Societies of: Gastroenterology and Endoscopy (SIGE), Neurogastroenterology and Motility (SINGEM), Hospital Gastroenterologists and Endoscopists (AIGO), Digestive Endoscopy (SIED), General Medicine (SIMG), Gastroenterology, Hepatology and Pediatric Nutrition (SIGENP) and Pediatrics (SIP). *Dig Liver Dis* 2023;55:187-207.
15. Fukudo S, Okumura T, Inamori M, et al. Evidence-based clinical practice guidelines for irritable bowel syndrome 2020. *J Gastroenterol* 2021;56:193-217.
16. Keller J, Wedel T, Seidl H, et al. [Not Available]. *Z Gastroenterol* 2022;60:192-218.
17. Layer P, Andresen V, Allescher H, et al. [Not Available]. *Z Gastroenterol* 2021;59:1323-1415.
18. Groen J, Gordon M, Chogle A, et al. ESPGHAN/NASPGHAN guidelines for treatment of irritable bowel syndrome and functional abdominal pain-not otherwise specified in children aged 4-18 years. *J Pediatr Gastroenterol Nutr* 2025.
19. Hilbert A, Hoek HW, Schmidt R. Evidence-based clinical guidelines for eating disorders: international comparison. *Curr Opin Psychiatry* 2017;30:423-437.
20. Couturier J, Isserlin L, Norris M, et al. Canadian practice guidelines for the treatment of children and adolescents with eating disorders. *J Eat Disord* 2020;8:4.
21. Chumpitazi BP, Weidler EM, Lu DY, et al. Self-Perceived Food Intolerances Are Common and Associated with Clinical Severity in Childhood Irritable Bowel Syndrome. *J Acad Nutr Diet* 2016;116:1458-1464.

22. Van Den Houte K, Carbone F, Tack J. Postprandial distress syndrome: stratification and management. *Expert Rev Gastroenterol Hepatol* 2019;13:37-46.
23. Jones MP, Talley NJ, Eslick GD, et al. Community subgroups in dyspepsia and their association with weight loss. *Am J Gastroenterol* 2008;103:2051-60.
24. Satherley R, Howard R, Higgs S. Disordered eating practices in gastrointestinal disorders. *Appetite* 2015;84:240-50.
25. van Tilburg MA, Squires MM, Blois-Martin N, et al. Sa2013 Diet and Eating Associated Symptoms in Adolescents With IBS. *Gastroenterology* 2012;5:S-381.
26. Monsbakken KW, Vandvik PO, Farup PG. Perceived food intolerance in subjects with irritable bowel syndrome – etiology, prevalence and consequences. *European Journal of Clinical Nutrition* 2006;60:667-672.
27. Benninga MA, Faure C, Hyman PE, et al. Childhood Functional Gastrointestinal Disorders: Neonate/Toddler. *Gastroenterology* 2016.
28. Stanghellini V, Chan FK, Hasler WL, et al. Gastroduodenal Disorders. *Gastroenterology* 2016;150:1380-92.
29. Fonseca NKO, Curtarelli VD, Bertoletti J, et al. Avoidant restrictive food intake disorder: recent advances in neurobiology and treatment. *J Eat Disord* 2024;12:74.
30. Association AP. *Diagnostic and Statistical Manual of Mental Disorders: DSM-5*, 2013.
31. Riesco N, Agüera Z, Granero R, et al. Other Specified Feeding or Eating Disorders (OSFED): Clinical heterogeneity and cognitive-behavioral therapy outcome. *Eur Psychiatry* 2018;54:109-116.
32. Carpinelli L, Savarese G, Pascale B, et al. Gut-Brain Interaction Disorders and Anorexia Nervosa: Psychopathological Asset, Disgust, and Gastrointestinal Symptoms. *Nutrients* 2023;15.
33. Gibson D, Watters A, Mehler PS. The intersect of gastrointestinal symptoms and malnutrition associated with anorexia nervosa and avoidant/restrictive food intake disorder: Functional or pathophysiologic?-A systematic review. *Int J Eat Disord* 2021;54:1019-1054.
34. Wiklund CA, Rania M, Kuja-Halkola R, et al. Evaluating disorders of gut-brain interaction in eating disorders. *Int J Eat Disord* 2021;54:925-935.
35. Kessler U, Rekkedal G, Rø Ø, et al. Association between gastrointestinal complaints and psychopathology in patients with anorexia nervosa. *Int J Eat Disord* 2020;53:532-536.
36. Corvilain B, Abramowicz M, Féry F, et al. Effect of short-term starvation on gastric emptying in humans: relationship to oral glucose tolerance. *Am J Physiol* 1995;269:G512-7.
37. Benini L, Todesco T, Dalle Grave R, et al. Gastric Emptying in Patients with Restricting and Binge/Purging Subtypes of Anorexia Nervosa. *Official journal of the American College of Gastroenterology | ACG* 2004;99:1448-1454.
38. Waldholtz BD, Andersen AE. Gastrointestinal symptoms in anorexia nervosa. A prospective study. *Gastroenterology* 1990;98:1415-9.
39. Boyd C, Abraham S, Kellow J. Appearance and disappearance of functional gastrointestinal disorders in patients with eating disorders. *Neurogastroenterol Motil* 2010;22:1279-83.
40. Fink M, Simons M, Tomasino K, et al. When Is Patient Behavior Indicative of Avoidant Restrictive Food Intake Disorder (ARFID) Vs Reasonable Response to Digestive Disease? *Clin Gastroenterol Hepatol* 2022;20:1241-1250.
41. Koloski N, Holtmann G, Talley NJ. Is there a causal link between psychological disorders and functional gastrointestinal disorders? *Expert Rev Gastroenterol Hepatol* 2020;14:1047-1059.
42. Bray NA, Koloski NA, Jones MP, et al. Evaluation of a Multidisciplinary Integrated Treatment Approach Versus Standard Model of Care for Functional Gastrointestinal Disorders (FGIDS): A Matched Cohort Study. *Dig Dis Sci* 2022.
43. Knowles SR, Apputhurai P, Palsson OS, et al. The epidemiology and psychological comorbidity of disorders of gut-brain interaction in Australia: Results from the Rome Foundation Global Epidemiology Study. *Neurogastroenterol Motil* 2023;35:e14594.
44. Apputhurai P, Palsson OS, Bangdiwala SI, et al. Confirmatory validation of the patient health questionnaire - 4 (PHQ-4) for gastrointestinal disorders: A large-scale cross-sectional survey. *J Psychosom Res* 2024;180:111654.

45. Quigley EMM, Noble O, Ansari U. The Suggested Relationships Between Common GI Symptoms and Joint Hypermobility, POTS, and MCAS. *Gastroenterol Hepatol (N Y)* 2024;20:479-489.
46. Lam CY, Palsson OS, Whitehead WE, et al. Rome IV Functional Gastrointestinal Disorders and Health Impairment in Subjects With Hypermobility Spectrum Disorders or Hypermobile Ehlers-Danlos Syndrome. *Clin Gastroenterol Hepatol* 2021;19:277-287.e3.
47. Mehr SE, Barbul A, Shibao CA. Gastrointestinal symptoms in postural tachycardia syndrome: a systematic review. *Clin Auton Res* 2018;28:411-421.
48. Weinstock LB, Pace LA, Rezaie A, et al. Mast Cell Activation Syndrome: A Primer for the Gastroenterologist. *Dig Dis Sci* 2021;66:965-982.
49. Hamilton MJ. Mast Cell Activation Syndrome and Gut Dysfunction: Diagnosis and Management. *Curr Gastroenterol Rep* 2024;26:107-114.
50. Lal V, Guazzo L. Median arcuate ligament syndrome: When to consider the diagnosis and management options. *Australian Journal for General Practitioners* 2024;53:28-32.
51. Raj SR. The Postural Tachycardia Syndrome (POTS): pathophysiology, diagnosis & management. *Indian Pacing Electrophysiol J* 2006;6:84-99.
52. Tinkle B, Castori M, Berglund B, et al. Hypermobile Ehlers-Danlos syndrome (a.k.a. Ehlers-Danlos syndrome Type III and Ehlers-Danlos syndrome hypermobility type): Clinical description and natural history. *Am J Med Genet C Semin Med Genet* 2017;175:48-69.
53. Grottemeyer D, Duran M, Iskandar F, et al. Median arcuate ligament syndrome: vascular surgical therapy and follow-up of 18 patients. *Langenbecks Arch Surg* 2009;394:1085-92.
54. Kucharik AH, Chang C. The Relationship Between Hypermobile Ehlers-Danlos Syndrome (hEDS), Postural Orthostatic Tachycardia Syndrome (POTS), and Mast Cell Activation Syndrome (MCAS). *Clin Rev Allergy Immunol* 2020;58:273-297.
55. Black CJ, Ford AC. Rational investigations in irritable bowel syndrome. *Frontline Gastroenterol* 2020;11:140-147.
56. Zingone F, Bertin L, Maniero D, et al. Myths and Facts about Food Intolerance: A Narrative Review. *Nutrients* 2023;15.
57. Fritscher-Ravens A, Pflaum T, Mössinger M, et al. Many Patients With Irritable Bowel Syndrome Have Atypical Food Allergies Not Associated With Immunoglobulin E. *Gastroenterology* 2019;157:109-118.e5.
58. Frieling T, Gjini B, Melchior I, et al. Gastrointestinal adverse reaction to food (GARF) and endoscopic confocal laser endomicroscopy (eCLE). *Z Gastroenterol* 2024;62:1201-1206.
59. Koloski NA, Jones M, Hammer J, et al. The Validity of a New Structured Assessment of Gastrointestinal Symptoms Scale (SAGIS) for Evaluating Symptoms in the Clinical Setting. *Dig Dis Sci* 2017;62:1913-1922.
60. Spiller RC, Humes DJ, Campbell E, et al. The Patient Health Questionnaire 12 Somatic Symptom scale as a predictor of symptom severity and consulting behaviour in patients with irritable bowel syndrome and symptomatic diverticular disease. *Aliment Pharmacol Ther* 2010;32:811-20.
61. Knowles SR, Apputhurai P, Burgell RE, et al. Development and Validation of the Gastrointestinal Unhelpful Thinking Scale (GUTs): A Brief Self-Report Measure for Clinical and Research Settings. *Gastroenterol Nurs* 2022;45:E1-e12.
62. Fairlie T, Shah A, Talley NJ, et al. Overlap of disorders of gut-brain interaction: a systematic review and meta-analysis. *Lancet Gastroenterol Hepatol* 2023;8:646-659.
63. Talley NJ, Silverstein MD, Agraus L, et al. AGA technical review: evaluation of dyspepsia. American Gastroenterological Association. *Gastroenterology* 1998;114:582-95.
64. Keller J, Hammer HF, Afolabi PR, et al. European guideline on indications, performance and clinical impact of (13) C-breath tests in adult and pediatric patients: An EAGEN, ESNM, and ESPGHAN consensus, supported by EPC. *United European Gastroenterol J* 2021;9:598-625.
65. Enck P, Azpiroz F, Boeckxstaens G, et al. Functional dyspepsia. *Nat Rev Dis Primers* 2017;3:17081.
66. Bluemel S, Menne D, Milos G, et al. Relationship of body weight with gastrointestinal motor and sensory function: studies in anorexia nervosa and obesity. *BMC Gastroenterology* 2017;17:4.

67. Heruc GA, Little TJ, Kohn MR, et al. Effects of starvation and short-term refeeding on gastric emptying and postprandial blood glucose regulation in adolescent girls with anorexia nervosa. *Am J Physiol Endocrinol Metab* 2018;315:E565-e573.
68. Camilleri M. Cannabinoids and gastrointestinal motility: Pharmacology, clinical effects, and potential therapeutics in humans. *Neurogastroenterol Motil* 2018;30:e13370.
69. Pannemans J, Carbone F, Tack J. Opioids in Gastroparesis: Bystander or Cause? *Clin Gastroenterol Hepatol* 2020;18:998-999.
70. Parkman HP, Rim DS, Anolik JR, et al. Glucagonlike Peptide-1 Receptor Agonists: The Good, the Bad, and the Ugly-Benefits for Glucose Control and Weight Loss with Side Effects of Delaying Gastric Emptying. *J Nucl Med Technol* 2024;52:3-7.
71. Camilleri M, Parkman HP, Shafi MA, et al. Clinical guideline: management of gastroparesis. *Am J Gastroenterol* 2013;108:18-37; quiz 38.
72. Stanghellini V, Tosetti C, Paternico A, et al. Risk indicators of delayed gastric emptying of solids in patients with functional dyspepsia. *Gastroenterology* 1996;110:1036-42.
73. Pasricha PJ, Colvin R, Yates K, et al. Characteristics of patients with chronic unexplained nausea and vomiting and normal gastric emptying. *Clin Gastroenterol Hepatol* 2011;9:567-76.e1-4.
74. Haag S, Senf W, Tagay S, et al. Is there any association between disturbed gastrointestinal visceromotor and sensory function and impaired quality of life in functional dyspepsia? *Neurogastroenterol Motil* 2010;22:262-e79.
75. Szarka LA, Camilleri M. Methods for the assessment of small-bowel and colonic transit. *Semin Nucl Med* 2012;42:113-23.
76. Varghese C, Xu W, Daker C, et al. Clinical utility of Gastric Alimetry® in the management of intestinal failure patients with possible underlying gut motility disorders. *Clinical Nutrition Open Science* 2023;51:15-25.
77. Jamieson AE, Fletcher PC, Schneider MA. Seeking control through the determination of diet: A qualitative investigation of women with irritable bowel syndrome and inflammatory bowel disease. *Clinical Nurse Specialist* 2007;21:152-160.
78. Tuck CJ, Reed DE, Muir JG, et al. Implementation of the low FODMAP diet in functional gastrointestinal symptoms: A real-world experience. *Neurogastroenterol Motil* 2020;32:e13730.
79. Sultan N, Varney JE, Halmos EP, et al. How to Implement the 3-Phase FODMAP Diet Into Gastroenterological Practice. *Journal of Neurogastroenterology and Motility* 2022;28:343-356.
80. Prospero L, Riezzo G, Linsalata M, et al. Psychological and Gastrointestinal Symptoms of Patients with Irritable Bowel Syndrome Undergoing a Low-FODMAP Diet: The Role of the Intestinal Barrier. *Nutrients* 2021;13.
81. Haller E, Scarlata K. Diet Interventions for Irritable Bowel Syndrome: Separating the Wheat from the Chafe. *Gastroenterol Clin North Am* 2021;50:565-579.
82. Halmos EP, Gibson PR. Controversies and reality of the FODMAP diet for patients with irritable bowel syndrome. *Journal of Gastroenterology and Hepatology* 2019;34:1134-1142.
83. Thomassen RA, Luque V, Assa A, et al. An ESPGHAN Position Paper on the Use of Low-FODMAP Diet in Pediatric Gastroenterology. *J Pediatr Gastroenterol Nutr* 2022;75:356-368.
84. Tuck CJ, Reed DE, Muir JG, et al. Implementation of the low FODMAP diet in functional gastrointestinal symptoms: A real-world experience. *Neurogastroenterology & Motility* 2020;32:e13730.
85. Bischoff SC, Austin P, Boeykens K, et al. ESPEN practical guideline: Home enteral nutrition. *Clin Nutr* 2022;41:468-488.
86. Nightingale JMD, Paine P, McLaughlin J, et al. The management of adult patients with severe chronic small intestinal dysmotility. *Gut* 2020;69:2074-2092.
87. Gallo R, Armstrong E, Griffin H, et al. Impact of enteral tube feeding for individuals with disorders of the gut brain interaction. *Clinical Nutrition ESPEN* 2023;58:604.
88. Martin LD, Wang X, Day C, et al. Enteral tube feeding in functional gastrointestinal disorders: A game of chance? *Clinical Nutrition ESPEN* 2023;57:849.
89. Paine P, McMahon M, Farrer K, et al. Jejunal feeding: when is it the right thing to do? *Frontline Gastroenterol* 2020;11:397-403.

90. Sonnenberg A, Bakis G. Probability of Iatrogenesis in Gastroenterology. *Dig Dis Sci* 2016;61:1775-7.
91. Krom H, de Winter JP, Kindermann A. Development, prevention, and treatment of feeding tube dependency. *Eur J Pediatr* 2017;176:683-688.
92. Cederholm T, Jensen G, Correia M, et al. GLIM criteria for the diagnosis of malnutrition—a consensus report from the global clinical nutrition community. *Journal of cachexia, sarcopenia and muscle* 2019;10:207-217.
93. Lal S, Paine P, Tack J, et al. Avoiding the use of long-term parenteral support in patients without intestinal failure: A position paper from the European Society of Clinical Nutrition & Metabolism, the European Society of Neurogastroenterology and Motility and the Rome Foundation for Disorders of Gut-Brain Interaction. *Neurogastroenterol Motil* 2024;36:e14853.
94. Sanchez-Cerezo J, Nagularaj L, Gledhill J, et al. What do we know about the epidemiology of avoidant/restrictive food intake disorder in children and adolescents? A systematic review of the literature. *Eur Eat Disord Rev* 2023;31:226-246.
95. Pironi L, Arends J, Baxter J, et al. ESPEN endorsed recommendations. Definition and classification of intestinal failure in adults. *Clin Nutr* 2015;34:171-80.
96. Berlana D. Parenteral Nutrition Overview. *Nutrients* 2022;14.
97. Fleming C. Intestinal failure. *Nutrition and the Surgical Patient Clinical Surgery International* 1981;2:219-235.
98. Pironi L, Cuerda C, Jeppesen PB, et al. ESPEN guideline on chronic intestinal failure in adults - Update 2023. *Clin Nutr* 2023;42:1940-2021.
99. Ukleja A, Romano MM. Complications of parenteral nutrition. *Gastroenterol Clin North Am* 2007;36:23-46, v.
100. Guzman M, Manithody C, Krebs J, et al. Impaired Gut-Systemic Signaling Drives Total Parenteral Nutrition-Associated Injury. *Nutrients* 2020;12.
101. Goodall R, Langridge B, Onida S, et al. Median arcuate ligament syndrome. *J Vasc Surg* 2020;71:2170-2176.
102. Ayers P, Adams S, Boullata J, et al. A.S.P.E.N. parenteral nutrition safety consensus recommendations. *JPEN J Parenter Enteral Nutr* 2014;38:296-333.
103. Lal S, Paine P, Tack J, et al. Avoiding the use of long-term parenteral support in patients without intestinal failure: A position paper from the European Society of Clinical Nutrition & Metabolism, the European Society of Neurogastroenterology and Motility and the Rome Foundation for Disorders of Gut-Brain Interaction. *Clinical Nutrition* 2024;43:2279-2282.
104. Keefer L, Ballou SK, Drossman DA, et al. A Rome Working Team Report on Brain-Gut Behavior Therapies for Disorders of Gut-Brain Interaction. *Gastroenterology* 2022;162:300-315.
105. Pittayanon R, Yuan Y, Bollegala NP, et al. Prokinetics for Functional Dyspepsia: A Systematic Review and Meta-Analysis of Randomized Control Trials. *Am J Gastroenterol* 2019;114:233-243.
106. Talley NJ, Verlinden M, Snape W, et al. Failure of a motilin receptor agonist (ABT-229) to relieve the symptoms of functional dyspepsia in patients with and without delayed gastric emptying: a randomized double-blind placebo-controlled trial. *Aliment Pharmacol Ther* 2000;14:1653-61.
107. Pittayanon R, Yuan Y, Bollegala NP, et al. Prokinetics for functional dyspepsia. *Cochrane Database Syst Rev* 2018;10:Cd009431.
108. Ford AC, Luthra P, Tack J, et al. Efficacy of psychotropic drugs in functional dyspepsia: systematic review and meta-analysis. *Gut* 2017;66:411-420.
109. Lu Y, Chen M, Huang Z, et al. Antidepressants in the Treatment of Functional Dyspepsia: A Systematic Review and Meta-Analysis. *PLoS One* 2016;11:e0157798.
110. Xie C, Tang Y, Wang Y, et al. Efficacy and Safety of Antidepressants for the Treatment of Irritable Bowel Syndrome: A Meta-Analysis. *PLoS One* 2015;10:e0127815.
111. Ballou S, Iturrino J, Rangan V, et al. Improving Medication Tolerance: A Pilot Study in Disorders of Gut-brain Interaction Treated With Tricyclic Antidepressants. *J Clin Gastroenterol* 2022;56:452-456.

112. Tack J, Ly HG, Carbone F, et al. Efficacy of Mirtazapine in Patients With Functional Dyspepsia and Weight Loss. *Clin Gastroenterol Hepatol* 2016;14:385-392.e4.
113. Cao L, Li G, Cao J, et al. Randomized clinical trial: the effects of mirtazapine in functional dyspepsia patients. *Therap Adv Gastroenterol* 2025;18:17562848241311129.
114. Rich G, Shah A, Koloski N, et al. A randomized placebo-controlled trial on the effects of Menthacarin, a proprietary peppermint- and caraway-oil-preparation, on symptoms and quality of life in patients with functional dyspepsia. *Neurogastroenterol Motil* 2017;29.
115. Alammari N, Wang L, Saberi B, et al. The impact of peppermint oil on the irritable bowel syndrome: a meta-analysis of the pooled clinical data. *BMC Complement Altern Med* 2019;19:21.
116. Chumpitazi BP, Kearns GL, Shulman RJ. Review article: the physiological effects and safety of peppermint oil and its efficacy in irritable bowel syndrome and other functional disorders. *Aliment Pharmacol Ther* 2018;47:738-752.
117. Connell AM. Physiological and clinical assessment of the effect of the musculotropic agent mebeverine on the human colon. *Br Med J* 1965;2:848-51.
118. Daniluk J, Malecka-Wojcieszko E, Skrzydło-Radomska B, et al. The Efficacy of Mebeverine in the Treatment of Irritable Bowel Syndrome-A Systematic Review. *J Clin Med* 2022;11.
119. Goodoory VC, Khasawneh M, Thakur ER, et al. Effect of Brain-Gut Behavioral Treatments on Abdominal Pain in Irritable Bowel Syndrome: Systematic Review and Network Meta-Analysis. *Gastroenterology* 2024;167:934-943.e5.
120. Black CJ, Thakur ER, Houghton LA, et al. Efficacy of psychological therapies for irritable bowel syndrome: systematic review and network meta-analysis. *Gut* 2020;69:1441-1451.
121. Bray NA, Koloski NA, Jones MP, et al. Evaluation of a Multidisciplinary Integrated Treatment Approach Versus Standard Model of Care for Functional Gastrointestinal Disorders (FGIDS): A Matched Cohort Study. *Dig Dis Sci* 2022;67:5593-5601.
122. Haag S, Senf W, Tagay S, et al. Is there a benefit from intensified medical and psychological interventions in patients with functional dyspepsia not responding to conventional therapy? *Aliment Pharmacol Ther* 2007;25:973-86.
123. Hesser H, Hedman-Lagerlöf E, Andersson E, et al. How does exposure therapy work? A comparison between generic and gastrointestinal anxiety-specific mediators in a dismantling study of exposure therapy for irritable bowel syndrome. *J Consult Clin Psychol* 2018;86:254-267.
124. Biesiekierski JR, Manning LP, Murray HB, et al. Review article: exclude or expose? The paradox of conceptually opposite treatments for irritable bowel syndrome. *Aliment Pharmacol Ther* 2022;56:592-605.
125. Burton Murray H, Weeks I, Becker KR, et al. Development of a brief cognitive-behavioral treatment for avoidant/restrictive food intake disorder in the context of disorders of gut-brain interaction: Initial feasibility, acceptability, and clinical outcomes. *Int J Eat Disord* 2023;56:616-627.
126. Lindfors P, Unge P, Arvidsson P, et al. Effects of gut-directed hypnotherapy on IBS in different clinical settings-results from two randomized, controlled trials. *Am J Gastroenterol* 2012;107:276-85.
127. Naliboff BD, Smith SR, Serpa JG, et al. Mindfulness-based stress reduction improves irritable bowel syndrome (IBS) symptoms via specific aspects of mindfulness. *Neurogastroenterol Motil* 2020;32:e13828.
128. Li J, Cai Z, Li X, et al. Mindfulness-based therapy versus cognitive behavioral therapy for people with anxiety symptoms: a systematic review and meta-analysis of random controlled trials. *Ann Palliat Med* 2021;10:7596-7612.
129. Gaylord SA, Palsson OS, Garland EL, et al. Mindfulness training reduces the severity of irritable bowel syndrome in women: results of a randomized controlled trial. *Am J Gastroenterol* 2011;106:1678-88.
130. Mirsharifa SM, Mirzaian B, Dousti Y. The Efficacy of Acceptance and Commitment Therapy (ACT) Matrix on Depression and Psychological Capital of the Patients with Irritable Bowel Syndrome. *Open Access Maced J Med Sci* 2019;7:421-427.

131. Marchese SH, Naftaly JP, Pandolfino J. Acceptance and commitment therapy for the treatment of irritable bowel syndrome and inflammatory bowel disease: a narrative review. *Transl Gastroenterol Hepatol* 2024;9:43.
132. Manikam R, Perman JA. Pediatric feeding disorders. *J Clin Gastroenterol* 2000;30:34-46.
133. Thapar N, Benninga MA, Crowell MD, et al. Paediatric functional abdominal pain disorders. *Nat Rev Dis Primers* 2020;6:89.
134. Koen Vermeijden N, de Silva L, Manathunga S, et al. Epidemiology of Pediatric Functional Abdominal Pain Disorders: A Meta-Analysis. *Pediatrics* 2025;155.
135. Manikam R, Perman J. Pediatric Feeding Disorders. *Journal of clinical gastroenterology* 2000;30:34-46.
136. Rommel N, De Meyer A-M, Feenstra L, et al. The complexity of feeding problems in 700 infants and young children presenting to a tertiary care institution. *Journal of pediatric gastroenterology and nutrition* 2003;37:75-84.
137. Stein K, Warne N, Heron J, et al. Do children with recurrent abdominal pain grow up to become adolescents who control their weight by fasting? Results from a UK population-based cohort. *Int J Eat Disord* 2021;54:915-924.
138. Puoti MG, Safe M, Thapar N, et al. The role of high-resolution impedance manometry to identify rumination syndrome in children with unexplained foregut symptoms. *J Pediatr Gastroenterol Nutr* 2024;78:1082-1090.
139. Jia MR, Lu PL, Khoo JS, et al. Delay in diagnosis is associated with decreased treatment effectiveness in children with rumination syndrome. *J Pediatr Gastroenterol Nutr* 2024;79:850-854.
140. Nurko S, Benninga MA, Solari T, et al. Pediatric Aspects of Nutrition Interventions for Disorders of Gut-Brain Interaction. *Am J Gastroenterol* 2022;117:995-1009.
141. Carlson MJ, Moore CE, Tsai CM, et al. Child and parent perceived food-induced gastrointestinal symptoms and quality of life in children with functional gastrointestinal disorders. *J Acad Nutr Diet* 2014;114:403-413.
142. Chumpitazi BP, McMeans AR, Vaughan A, et al. Fructans Exacerbate Symptoms in a Subset of Children With Irritable Bowel Syndrome. *Clin Gastroenterol Hepatol* 2018;16:219-225.e1.
143. Chumpitazi BP, Cope JL, Hollister EB, et al. Randomised clinical trial: gut microbiome biomarkers are associated with clinical response to a low FODMAP diet in children with the irritable bowel syndrome. *Aliment Pharmacol Ther* 2015;42:418-27.
144. van Tilburg MA, Felix CT. Diet and functional abdominal pain in children and adolescents. *J Pediatr Gastroenterol Nutr* 2013;57:141-8.
145. Atkins M, Zar-Kessler C, Madva EN, et al. History of trying exclusion diets and association with avoidant/restrictive food intake disorder in neurogastroenterology patients: A retrospective chart review. *Neurogastroenterol Motil* 2023;35:e14513.
146. Alfaro Cruz L, Minard C, Guffey D, et al. Does a Minority of Children With Functional Gastrointestinal Disorders Receive Formal Diet Advice? *JPEN J Parenter Enteral Nutr* 2020;44:1525-1529.
147. Katzman DK, Spettigue W, Agostino H, et al. Incidence and Age- and Sex-Specific Differences in the Clinical Presentation of Children and Adolescents With Avoidant Restrictive Food Intake Disorder. *JAMA Pediatrics* 2021;175:e213861-e213861.
148. Kaul I, Burton-Murray H, Musaad S, et al. Avoidant/restrictive food intake disorder prevalence is high in children with gastroparesis and functional dyspepsia. *Neurogastroenterol Motil* 2024;36:e14777.
149. Murray HB, Rao FU, Baker C, et al. Prevalence and Characteristics of Avoidant/Restrictive Food Intake Disorder in Pediatric Neurogastroenterology Patients. *J Pediatr Gastroenterol Nutr* 2022;74:588-592.
150. Burton Murray H, Staller K. When Food Moves From Friend to Foe: Why Avoidant/Restrictive Food Intake Matters in Irritable Bowel Syndrome. *Clin Gastroenterol Hepatol* 2022;20:1223-1225.
151. Simons M, Taft TH, Doerfler B, et al. Narrative review: Risk of eating disorders and nutritional deficiencies with dietary therapies for irritable bowel syndrome. *Neurogastroenterol Motil* 2022;34:e14188.

152. Salzberg MR, Kim H, Basnayake C, et al. Role of social media in the presentation of disorders of gut-brain interaction: Review and recommendations. *J Gastroenterol Hepatol* 2024;39:2281-2292.
153. Giedinghagen A. The tic in TikTok and (where) all systems go: Mass social media induced illness and Munchausen's by internet as explanatory models for social media associated abnormal illness behavior. *Clin Child Psychol Psychiatry* 2023;28:270-278.

Table 2: Provisional diagnostic criteria for DGBI with altered (reduced) food intake

- a) Gastrointestinal symptoms meeting the current Rome criteria for DGBI (e.g., Functional Dyspepsia or IBS) that predate the onset of altered food intake
- b) Gastrointestinal symptoms that are aggravated by food intake and
- c) result in altered (reduced) food intake with a subsequent (unintended) reduction of the body weight (>5% of body weight over 6 to 12 months).
- d) An eating disorder has been ruled out or does not fully explain the clinical presentation.

Table 2: Clinical features and criteria allowing differentiation of DGBI and altered food intake vs key eating disorders (DSM-5).

	Disorder of Gut-Brain Interaction (DGBI) with altered food intake	DSM-5 Avoidant/Restrictive Food Intake Disorder (ARFID)	DSM-5 Anorexia nervosa (AN)	DSM-5 Bulimia nervosa (BN)	DSM-5 Binge eating disorders (BED)	DSM-5 PICA
Patients meet the criteria for DGBI (Rome IV criteria), predating the onset of altered food intake behavior.	Required	Does not exclude ARFID	Does not exclude AN	Does not exclude BN	Does not exclude BED	Not a typical feature of PICA
The DGBI-related symptoms are severe or very severe, defined by their impact on daily life. Severe symptoms cannot be ignored by the patient and impact daily life.	Required	Does not exclude ARFID	Does not exclude AN	Does not exclude BN	Does not exclude BED	Not a typical feature of PICA
Gastrointestinal symptoms are triggered or aggravated by food intake and are consistent with a DGBI before the onset of the altered food intake. These symptoms are the primary (leading) cause for the restricted food intake.	Required	Does not exclude ARFID	May exclude AN	May exclude Excludes BN	Excludes BED	Not a typical feature of PICA
Eating or feeding disturbance (e.g., apparent lack of interest in eating; avoidance based on the sensory characteristics of food; concern about aversive consequences of eating), failure to meet nutritional and/or energy requirements associated with one or more of the following: weight loss, inability to achieve expected weight gain, dependence on enteral feeding or nutritional support, interference with psychosocial functioning.	May co-occur but may not fully explain the eating or feeding disturbance.	Required	Not a typical feature of AN	Not a typical feature of BN	Not a typical feature of BED	Not a typical feature of PICA
Restriction of energy intake leads to significantly low body weight, an intense fear of gaining weight, and a distorted body image (restrictive vs. binge eating purge behavior).	Not a typical feature but restrictions of caloric intake can occur.	Not a typical feature of ARFID	Required	Not a typical feature of BN	Not a typical feature of BED	Not a typical feature of PICA
Recurrent episodes of binge eating, with consumption of excessive amounts of food with a sense of lack of control, followed by inappropriate compensatory behaviors (such as self-induced vomiting, excessive exercise, or misuse of laxatives).	Not a typical feature	Not a typical feature of ARFID	Not a typical feature of AN	Required	Not a typical feature of BED	Not a typical feature of PICA
Recurrent episodes of binge eating, defined as consuming an excessive amount of food in a discrete period, accompanied by a sense of loss of control overeating.	Not a typical feature	Not a typical feature of ARFID	Not a typical feature of AN	Not a typical feature of BN	Required	Not a typical feature of PICA
Persistent consumption of non-nutritive, non-food substances.	Not a typical feature	Not a typical feature of ARFID	Not a typical feature of AN	Not a typical feature of BN	Not a typical feature of BED	Required
Repeated regurgitation of food, which may be re-chewed, re-swallowed, or spit out. It is considered a learned behaviour that is classified as a DGBI.	Not a typical feature	Not a typical feature of ARFID	Not a typical feature of AN	Not a typical feature of BN	Not a typical feature of BED	Not a typical feature of PICA

Consensus Statements for the Guideline Management of Patients with Disorders of Gut-Brain Interaction and Altered (Reduced) Food Intake Behaviour

Short title: DGBI-induced altered food intake behaviour

This document contains 23 statements in relation to the GESA Guideline ‘Management of Patients with Disorders of Gut-Brain Interaction and Altered (Reduced) Food Intake Behaviour’. These statements should be read in the context of the Guideline. All members of the working group assessed the *Level of Evidence* and, when appropriate, the *Grade of Recommendation*. Below each statement, the *Level of Evidence* and *Grade of Recommendation* are noted based on the criteria below. All statements included in this guideline were required to be supported by more than 80% of the guideline group. Any 'no votes' or 'non-responses' were counted as not supporting the respective statement.

Level of Evidence and Grade of Recommendation

Levels of Evidence

Level I: Randomised Controlled Trials (RCTs) and Systematic Reviews of RCTs

Level II: Prospective Cohort Studies

Level III: Retrospective Cohort Studies and Case-Control Studies

Level IV: Case Series and Poor-Quality Cohort and Case-Control Studies

Level V: Expert Opinion or Bench Research

Grades of Recommendation

Grade A: High-quality, consistent evidence from Level I studies

Interventions receive a Grade A recommendation when supported by consistent, high-quality evidence from RCTs and systematic reviews.

Grade B: Moderate-quality evidence from Level II or III studies

Information/recommendation based on well-conducted cohort or case-control studies.

Grade C: Low-quality evidence from Level IV studies

Recommendations are supported by case series or poor-quality cohort/case-control studies.

Grade D: Evidence from Level V studies or inconsistent findings

Recommendations essentially rely on expert opinions.

Statement 1:

In population-based cohorts, symptoms of dyspepsia (either with nausea and vomiting or meal-related symptoms) can be associated with significant unintentional weight loss (e.g., >5% of body weight within 12 months).

Level of evidence: II; Recommendation: Grade B; Accepted by 92.3%

Statement 2:

Weight loss (>5% of body weight) due to altered food intake behaviour resulting from meal-related symptoms can occur in patients with DGBI, such as functional dyspepsia with postprandial distress syndrome.

Level of evidence: II; Grade of recommendation: B; Accepted by 92.3%

Statement 3:

‘DGBI and altered food intake behaviour’ are characterised by gastrointestinal symptoms that fulfil all the below

- (i) meet the current Rome criteria for DGBI (e.g., functional dyspepsia or irritable bowel syndrome)**
- (ii) are aggravated by food intake and result in altered (reduced) food intake**
- (iii) result in subsequent (unintended) reduction of the body weight (> 5% of body weight over 6 to 12 months)**
- (iv) predate the altered food intake behaviour and weight loss**
- (v) have been deemed to not represent an eating disorder, which has been ruled out or does not explain the clinical presentation.**

Level of evidence: V; Grade of Recommendation: D; Accepted by 88.5%

Statement 4:

Eating disorders need to be assessed and considered in the differential diagnosis of DGBI and altered food intake behaviour.

Level of evidence: II; Grade of recommendation: B; Accepted by 92.3%

Statement 5:

For all malnourished patients with DGBI and altered food intake behaviour, oral nutrition supplements should be comprehensively trialled with dietetic oversight before consideration of enteral or parenteral nutrition.

Level of evidence: V; Grade of recommendation: D; Accepted by 92.3%

Statement 6:

For patients with DGBI and altered food intake behaviour, enteral and/or parenteral nutrition should be avoided.

Level of evidence: V; Grade of recommendation: D; Accepted by 92.3%

Statement 7:

When enteral or parenteral nutrition is considered in patients with DGBI and altered food intake behaviour to address life-threatening nutritional deficiencies, a second opinion from an expert, preferably with experience in the management of patients with complex DGBI or intestinal failure, is recommended to support adherence to clinical standards, manage patient expectations effectively, and minimise the risk of harm.

Level of Evidence: V; Level of Recommendation: D; Accepted by 92.3%

Statement 8:

If enteral or parenteral nutrition is considered in patients with DGBI and altered food intake behaviour to address life-threatening nutritional deficiencies, the patients should be managed at centres with appropriate support from a multidisciplinary team with medical (gastroenterology, psychiatry), allied health (dietetics, psychology) and nursing staff with expertise in both enteral and parenteral nutrition and the management of patients with complex DGBI.

Level of evidence: V; Grade of Recommendation: D; Accepted by 92.3%

Statement 9:

If enteral or parenteral nutrition is required to address life-threatening nutritional deficiencies in DGBI and altered food intake behaviour, oral feeding should be continued and enteral or parenteral nutrition discontinued as soon as oral feeding is sufficient to meet nutritional requirements.

Level of evidence: V; Grade of Recommendation: D; Accepted by 92.3%

Statement 10:

Surgically implanted feeding tubes should be avoided in the management of patients with DGBI and altered food intake behaviour, since enteral feeding should not be routinely used and, if applied, should only be temporary and used for the shortest possible period.

Level of Evidence: V; Grade of Recommendation: D; Accepted by 92.3%

Statement 11:

If enteral nutrition needs to be considered to address malnutrition in patients with DGBI and altered food intake behaviour, it is a short- to medium-term (days or up to 4-weeks) measure aimed at stabilising nutritional status, preventing further decline, and supporting a transition to adequate oral intake as soon as possible.

Level of evidence: V; Grade of recommendation: D; Accepted by 92.3%

Statement 12:

Only if oral feeding and enteral nutrition prove to be unsuccessful and there is severe, life-threatening malnutrition, short-term, peripheral intravenous fluids may be administered in patients with DGBI and altered food intake behaviour.

Level of evidence: V; Grade of recommendation: D; Accepted by 92.3%

Statement 13:

The results of gut function testing need to be interpreted with caution in the context of concomitant medication (anticholinergic drugs, opioids, proton pump inhibitors), nutritional status (e.g., malnourishment or starvation) and the underlying symptoms before another clinical diagnosis (e.g. gastroparesis, small intestinal bacterial overgrowth) is established in patients with DGBI symptoms and altered food intake behaviour.

Level of Evidence: II; Grade of Recommendation: C; Accepted by 84.6%

Statement 14:

Clinical outcomes and patient characteristics in those with suspected DGBI and altered food intake behaviour should be documented where possible using validated instruments (e.g., patient-reported outcome measures (PROM)).

Level of Evidence: II; Grade of Recommendation: B; Accepted by 88.5%

Statement 15:

Anxiety, depression, hypermobility spectrum disorders (HSDs), postural orthostatic tachycardia syndrome (POTS), mast cell activation syndrome (MCAS) and vascular compression syndromes are comorbidities that appear to occur in patients with DGBI and altered food intake behaviour.

Level of Evidence: V; Grade of Recommendation: D; Accepted by 88.5%

Statement 16:

The diagnosis of hypermobility spectrum disorders (HSDs), postural orthostatic tachycardia syndrome (POTS), mast cell activation syndrome (MCAS) or vascular compression syndromes, in patients with DGBI and altered food intake behaviour should always meet contemporary, evidence-based criteria, and these conditions should not be diagnosed based solely on clinical suspicion or self-diagnosis.

Level of evidence: V; Grade of recommendation: D; Accepted by 92.3%

Statement 17:

The aims of treatment for patients with DGBI and altered food intake behaviour are to:

- *Reduce symptoms (e.g., meal-related pain, vomiting, distension, constipation/diarrhoea, bloating/distension)*
- *Reduce overall morbidity and mortality*
- *Facilitate nutritional repletion and achievement of a body weight that supports metabolic and functional recovery*
- *Reduce psychological distress (e.g., anxiety and depression) and improve quality of life*
- *Avoid treatment-related complications*

Level of Evidence: V; Grade of Recommendation: D; Accepted by 92.3%

Statement 18:

In the absence of data from clinical trials specifically targeting patients with DGBI and altered food intake behaviour, treatment and management for these patients should follow the established principles for patients with DGBI. Besides general measures, these treatments include pharmacologic (e.g. neuromodulators) and non-pharmacologic (dietary, psychological and behavioural) interventions, preferably with a multidisciplinary team.

Level of evidence: V; Grade of recommendation: D; Accepted by 92.3%

Statement 19:

The medical treatment of patients with DGBI and altered food intake behaviour should be directed at addressing the main symptom(s), while utilising the minimum number of medications necessary.

Level of evidence: V; Grade of Recommendation: D; Accepted by 92.3%

Statement 20:

Treatment of symptoms with opioids should be avoided in the management of patients with DGBI and altered food intake behaviour.

Level of Evidence: V; Grade of Recommendation: D; Accepted by 92.3%

Statement 21:

Patients with DGBI and altered food intake behaviour should be managed by providers with the required specialised knowledge (e.g., gastroenterologists, psychiatrists, psychologists and dietitians with special expertise on the management of patients with DGBI and/or gastrointestinal disorders).

Level of evidence: V; Grade of recommendation: D; Accepted by 88.5%

Statement 22:

Psychosocial and behavioural issues frequently influence the manifestation of symptoms in DGBI and altered food intake behaviour, highlighting the necessity for timely access to specialist psychosocial support.

Level of evidence: V; Grade of recommendation: D; Accepted by 92.3%

Statement 23:

A multidisciplinary integrated care model is recommended to manage *paediatric patients* with DGBI and altered food intake behaviour effectively. At minimum, this model should include a dietitian, psychologist or psychiatrist, general paediatrician/physician (for patients under 12), and gastroenterologist, with involvement from an adolescent medicine physician for those aged 13 to 18.

Level of evidence: V; Grade of recommendation: D; Accepted by 84.6%