

The Art and Science of Nutrition in Functional Dyspepsia

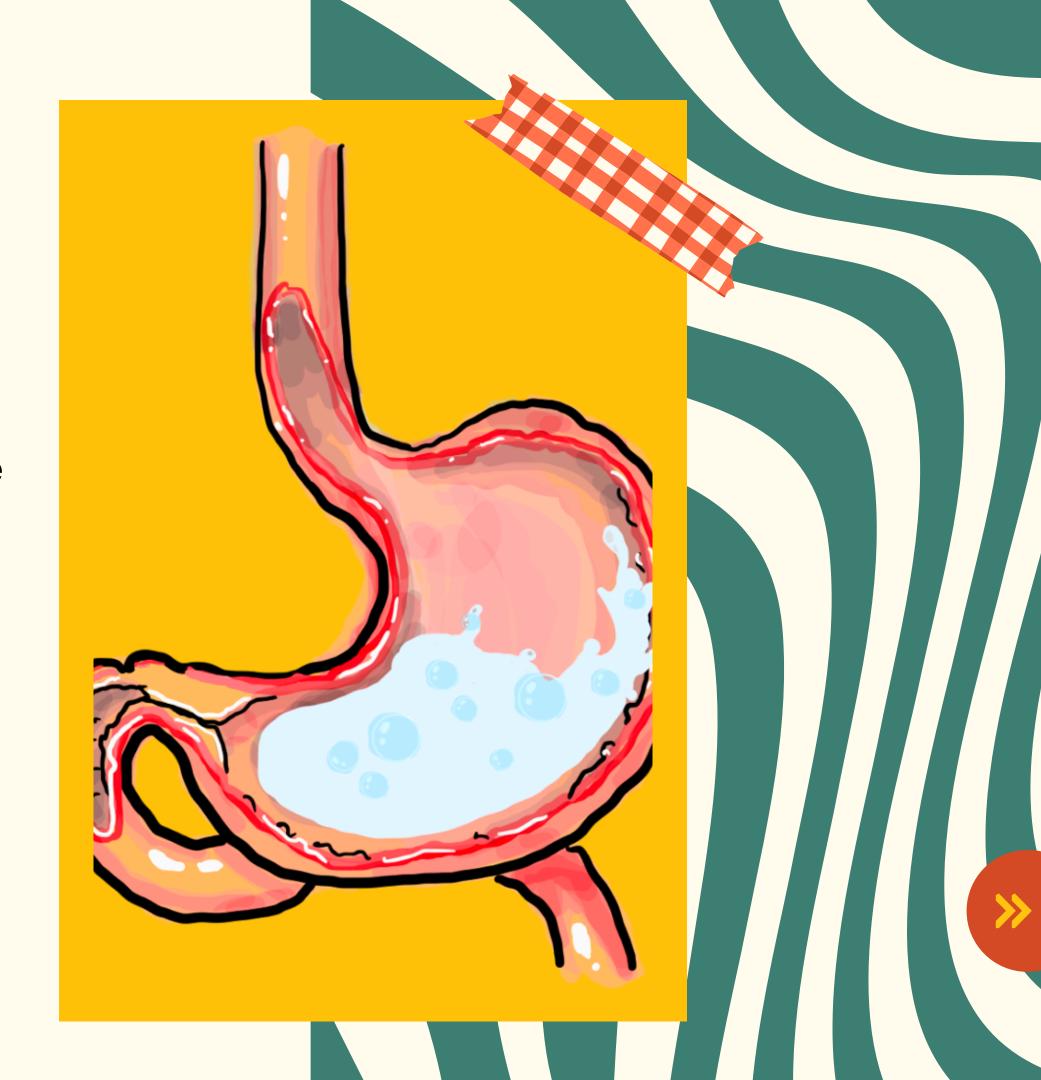


Emily Haller, MS, RDN, DipACLM

Trinity Health Ann Arbor Lifestyle and Culinary Medicine Program Coordinator

Objectives

- Define FD and it's subtypes using Rome
 IV criteria
- Identify dietary patterns + specific
 triggers with evidence for/against them
- Outline a practical, evidence-based nutrition care plan for FD
- Integrate behavioral and lifestyle strategies to complement dietary intervention



Functional Dyspepsia





<u>Bothersome</u> postprandial fullness, early satiety, epigastric pain or epigastric burning in the absence of structural abnormalities



- Postprandial Distress (PDS)= meal related symptoms
- Epigastric Pain Syndrome (EPS) = pain/burning that may or may not be related to meals



Nausea or vomiting can be present Symptoms present > 6 months



eGastroenterology

Functional dyspepsia and gastroparesis: are they distinct disorders, a spectrum of diseases or one disease?

Stella-Maris Egboh, 1,2,3 Kerith Duncanson, 2,3 Michael Potter, 4 Simon Keely, 3,5 Nicholas J Talley 5,2,3

To cite: Egboh S-M. Duncanson K, Potter M, et al. Functional dyspepsia and gastroparesis: are they distinct disorders, a spectrum of diseases or one disease? eGastroenterology 2025:3:e100119. doi:10.1136/ egastro-2024-100119

 Prepublication history for this paper is available online. To view these files, please visit the journal online (https://doi.org/ 10.1136/egastro-2024-100119).

Received 08 August 2024 Accepted 17 December 2024



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Federal Medical Centre.

Yenagoa, Bayelsa, Nigeria ²School of Medicine and Public Health, The University of Newcastle, Callaghan, New South Wales, Australia ³Centre of Research Excellence in Transforming Gut Health. The University of Newcastle, Callaghan, New South Wales, Australia ⁴Department of Gastroenterology, John Hunter Hospital, Newcastle, New South Wales, Australia ⁵School of Biomedical Sciences and Pharmacy. The University of Newcastle, Callaghan, New South Wales, Australia

Correspondence to

Dr Nicholas J Talley; nicholas.tallev@newcastle

ABSTRACT

Functional dyspepsia (FD) and gastroparesis (GP) are clinically managed as distinct upper gastrointestinal conditions but present with symptoms that are often indistinguishable. FD is a common disorder of gut-brain interaction that negatively impacts quality of life, while GP is considered a rare disease exclusively defined by delayed gastric emptying and symptoms. The degree of overlap between these disorders makes them hard to differentiate in clinical practice, thereby impacting treatment decisions. This review is focused on exploring the similarities and differences between FD and GP to guide clinician management and improve treatment outcomes. A comprehensive literature search was performed and the full texts of eligible articles were retrieved for the extraction of information reported in this review. This summary of evidence supports the hypothesis that GP and FD represent two ends of the same disease spectrum in a major subgroup. Improved understanding of the similarities, differences and overlap is likely to help guide the development of objective biomarkers and bettertargeted therapies.

INTRODUCTION

Overlapping symptoms between gastrointestinal (GI) conditions complicate diagnosis, treatment and management. This is particularly challenging for clinicians when the management of conditions differs substantially.

Functional dyspepsia (FD) is one of the most common disorders of gut-brain interaction (DGBI) and is defined by symptoms referable to the gastroduodenal region. These include bothersome epigastric pain or burning (the epigastric pain syndrome subtype), or bothersome postprandial fullness or early satiety (the postprandial distress syndrome (PDS) variant), with no evidence of established structural disease.² In contrast, gastroparesis (GP) is considered a rare heterogeneous neuromuscular disorder characterised by delayed gastric emptying (GE), described as gastric retention of more than 60% at 2 hours, and/or >10% at 4 hours on the GE scintigraphy

without evidence of mechanical obstruction.³ However, there is a considerable clinical overlap between the two disorders⁴⁻⁶ (table 1). Various criteria and validated symptom-based questionnaires have been applied to differentiate GP from FD, but lack of specificity limits their application. The hallmark of GP is delayed GE measured by gastric scintigraphy (GS), but 25%-37% of patients with FD also have some delay in GE, supporting the assertion that these disorders represent a ology.89 The complex interplay of altered central processing, visceral hypersensi-GI peptide release, low-grade mucosal immune activation and alterations in the gut microbiota may play a role in symptom generation in both GP and FD. 89 Although the duodenum has been extensively focused on the stomach in GP (figure 1). and subtle differences between FD and GP that could promote potential targets for future therapies.

A comprehensive search was performed on PubMed, Cochrane Central Register of Controlled Trials (CENTRAL) and Google Scholar from inception of database until February 2024 using the search terms: functional dyspepsia, gastroparesis, pathogenesis of gastroparesis and functional dyspepsia, microbiota, gut dysbiosis, small intestinal bacterial overgrowth (SIBO), bile acids, duodenal eosinophilia, enteric nervous system, visceral hypersensitivity, gastric accommodation, interstitial cells of Cajal (ICC), GI peptides, scintigraphy, electrogastrography (EGG), gastric



spectrum of gastroduodenal pathophysitivity, impaired accommodation, altered studied in FD, researchers have mainly This review aims to evaluate the overlap Neurogastroenterology

Functional dyspepsia or gastroparesis? A guide to assessment, investigation and management

Daniel Keszthelyi

Department of Gastroenterology Hepatology, Maastricht University Medical Center, Maastricht, The Netherlands

Correspondence to Professor Daniel Keszthelyi; daniel.keszthelvi@ maastrichtuniversity.nl

Received 12 January 2025 Accepted 23 March 2025 Published Online First 2 April 2025

ABSTRACT

Patients presenting with upper gastrointestinal symptoms such as postprandial fullness, early satiation, epigastric pain or burning, upper abdominal bloating, nausea and vomiting often receive the diagnoses of functional dyspepsia (FD), a disorder of the gut-brain interaction, or gastroparesis, a gastric motility disorder. The diagnosis of FD is largely symptom-based, using the Rome IV criteria, while for gastroparesis, establishing delayed gastric emptying (GE) in the absence of gastric outlet obstruction is necessary. However, symptoms of FD and gastroparesis can be indistinguishable, as well as certain pathophysiological mechanisms (ie, delayed GE, impaired gastric accommodation, visceral hypersensitivity) and treatments (ie, antiemetics, prokinetics, neuromodulators). Thus, the presentation and diagnosis of both FD and gastroparesis in clinical practice can be challenging for providers. Moreover, numerous studies have demonstrated that the severity of symptoms does not necessarily correlate with the degree of delay in GE. It has also recently been shown that patients often transition between diagnoses based on the volatile nature of GE test results. This calls for a reconceptualisation of these diagnostic entities. The aim of this review is therefore to perform a critical appraisal of similarities and differences between FD and gastroparesis and to provide a practical guide for the practising clinician.

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To cite: Keszthelyi D. Frontline Gastroenterology 2025; **16**:392–400.

Upper gastrointestinal (GI) problems are one of the primary reasons for patients to present at gastroenterology clinics. Yet such symptoms often represent a difficult diagnostic and therapeutic dilemma. Clinical work-up generally results in diagnostic labels of functional dyspepsia (FD) or gastroparesis. Ideally, these separate

KEY POINTS

- ⇒ Symptoms of functional dyspepsia (FD) and gastroparesis are often indistinguishable.
- ⇒ The prevalence of FD is considerably higher compared with gastroparesis (about 500-fold).
- ⇒ Delayed gastric emptying (GE) is not the sole underlying mechanism for symptom generation in gastroparesis.
- ⇒ Delayed GE rate, the defining feature of gastroparesis, can vary largely over time and shows mediocre correlation with symptom severity. Neither does the clinical response to prokinetics relate to GE rate.
- ⇒ Gastroparesis is commonly incorrectly diagnosed due to improperly performed GE tests.
- ⇒ Given the considerable overlap in symptom presentation and underlying pathomechanisms, FD and gastroparesis ought to be considered as being the extreme ends of a spectrum of gastroduodenal neuromuscular disorders.
- ⇒ The term 'gastroparesis' should be reserved for the more severe cases with marked delay in GE and possibly only for non-idiopathic aetiologies.

labels ought to reflect distinct underlying pathophysiological mechanisms and, at the same time, guide specific, effective management strategies. However, the therapeutic outcome with regards to symptom improvement, whichever diagnosis of upper GI problems is made, is often disappointing. In addition, recent studies have highlighted a substantial overlap in the pathophysiology, symptoms and clinical course of gastroparesis and FD. This therefore prompts a conceptual rethinking of the way we approach the management of patients with upper GI

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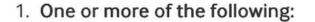
Table 1

Definitions of functional dyspepsia and gastroparesis, in addition to other diagnostic entities to be considered





Other conditions to consider



- a. Bothersome postprandial fullness
- b. Bothersome early satiation
- c. Bothersome epigastric pain
- d. Bothersome epigastric burning AND
- No evidence of structural disease (including at upper endoscopy) that is likely to explain the symptoms

Criteria fulfilled for the last 3 months with symptom onset at least 6 months before diagnosis.

Nausea and vomiting are cardinal symptoms. Frequently coexisting symptoms are early satiation and postprandial fullness.

Diagnosis requires the presence of these symptoms alongside delayed gastric emptying, measured by a 4-hour scintigraphy or gastric emptying breath test of a mixed composition meal in the absence of mechanical obstruction.

- Rumination syndrome
- Cannabinoid hyperemesis syndrome
- Cyclic vomiting syndrome
- Chronic nausea and vomiting syndrome

Comorbid conditions:

- Affective disorders (anxiety, depression)
- Eating disorders

(including avoidant/restrictive food intake disorder)







ACG Clinical Guideline: Gastroparesis

Michael Camilleri, MD, DSc, MRCP (UK), MACG, AGAF1, Braden Kuo, MD, MSc, FACG2, Linda Nguyen, MD3, Vida M. Vaughn, MLIS, MBA4, Jessica Petrey, MSLS4, Katarina Greer, MD, MS5, Rena Yadlapati, MD, I Thomas L. Abell, MD4

Gastroparesis is characterized by symptoms suggesting retention of food in the stomach with gastric emptying in the absence of mechanical obstruction in the gastric outflow. This condition in clinical practice. These guidelines summarize perspectives on the risk factors, diagnosis, gastroparesis in adults (including dietary, pharmacological, device, and interventions directe represent the official practice recommendations of the American College of Gastroenterology these guide lines was assessed using the Grading of Recommendations, Assessment, Developi When the evidence was not appropriate for Grading of Recommendations, Assessment, Devi used expert consensus to develop key concept statements. These guidelines should be consi the only approaches to these conditions.

SUPPLEMENTARY MATERIAL accompanies this paper at http://links.lww.com/AJG/C598.

Am J Gastroenterol 2022;117:1197-1220. https://doi.org/10.14309/ajg.000000000001874; published online June 3, 2022

2022 ACG Guideline

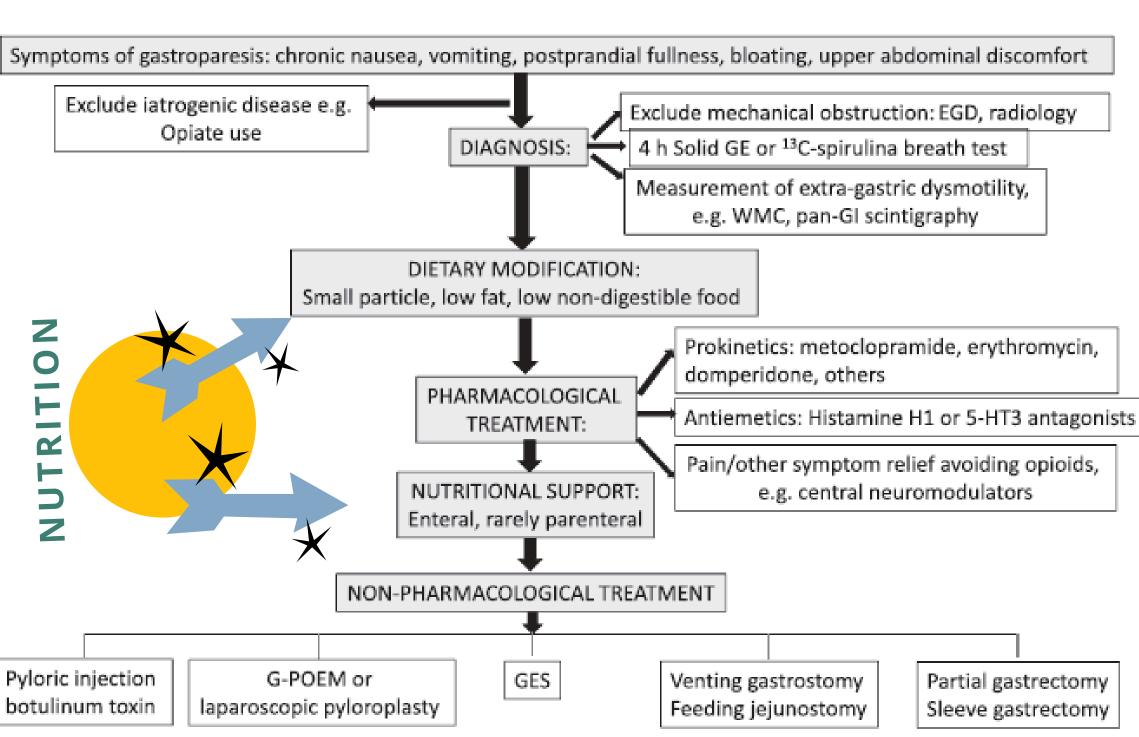


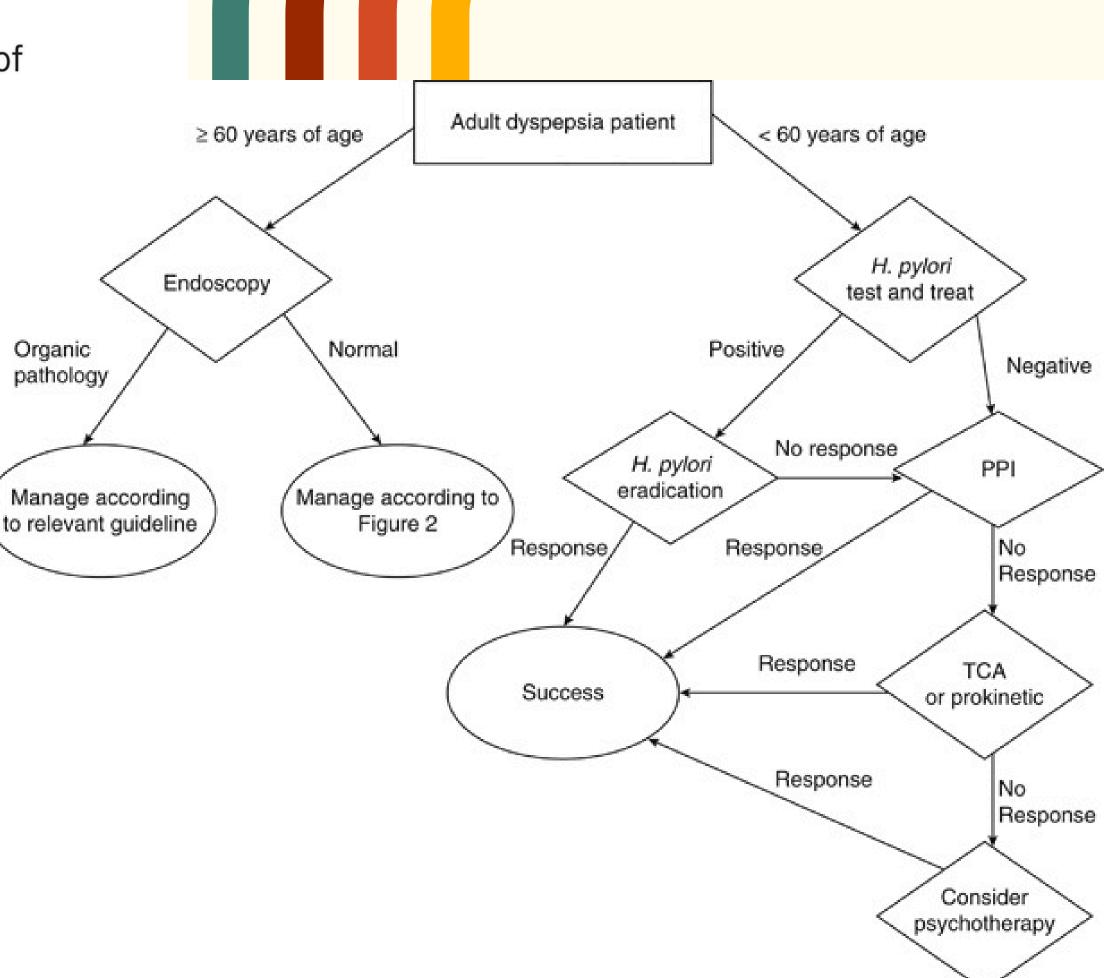
Figure 1. This algorithm updates the algorithm from the 2013 ACG guideline on gastroparesis (1). ACG, American College of Gastroenterology, EGD, esophagogastoduodenoscopy; GE, gastric emptying; GI, gastrointestinal; G-POEM, gastric per-oral endoscopic myotomy; WMC, wireless motility capsule.

ACG and CAG Clinical Guideline: Management of Dyspepsia

Paul M. Moayyedi, MB, ChB, PhD, MPH, FACG1, Brian E. Lacy, MD, PhD, FACG2, Christopher N. Andrews, MD3, Rob Colin W. Howden, MD, FACG5 and Nimish Vakil, MD, FACG6

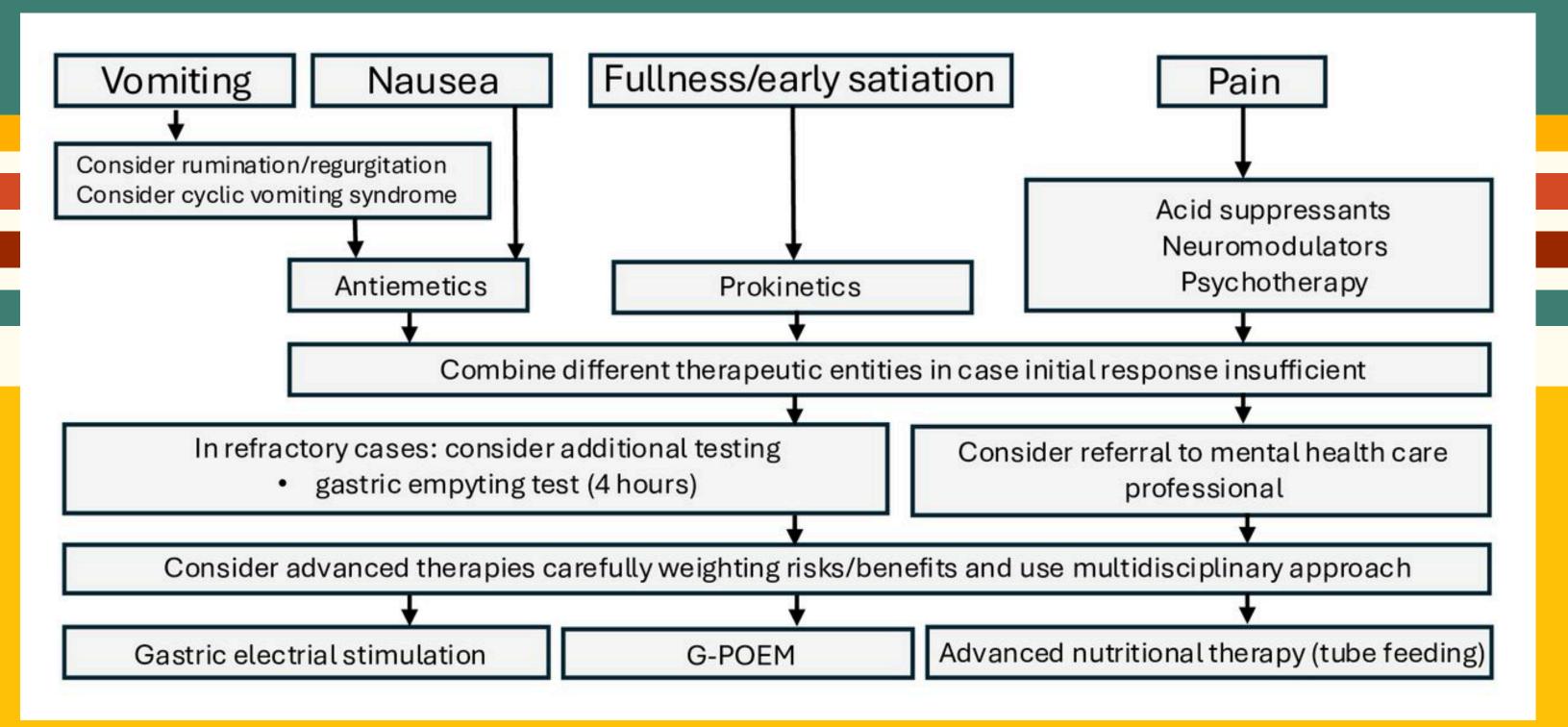
We have updated both the American College of Gastroenterology (ACG) and the Canadian Associat Gastroenterology (CAG) guidelines on dyspepsia in a joint ACG/CAG dyspepsia guideline. We sugge ≥60 years of age presenting with dyspepsia are investigated with upper gastrointestinal endoscopy pathology. This is a conditional recommendation and patients at higher risk of malignancy (such a childhood in a high risk gastric cancer country or having a positive family history) could be offered at a younger age. Alarm features should not automatically precipitate endoscopy in younger patien should be considered on a case-by-case basis. We recommend patients <60 years of age have a new Helicobacter pylori and treatment if positive. Those that are negative or do not respond to this app be given a trial of proton pump inhibitor (PPI) therapy. If these are ineffective tricyclic antidepress prokinetic therapies can be tried. Patients that have an endoscopy where no pathology is found are having functional dyspepsia (FD). H. pylori eradication should be offered in these patients if they We recommend PPI, TCA and prokinetic therapy (in that order) in those that fail therapy or are H. We do not recommend routine upper gastrointestinal (GI) motility testing but it may be useful in s

ACG 2017 and CAG Guideline



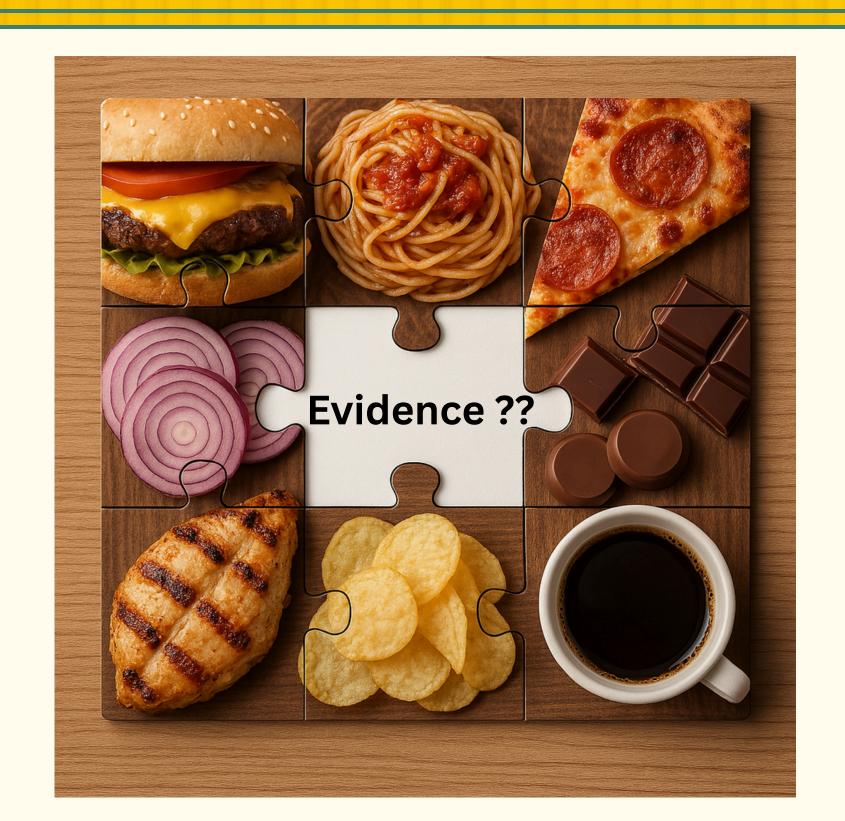


Assessing & Treating Most Bothersome Symptom



Dietary Triggers in FD: Common Concern, Uncommon Consensus

- Many patients identify meals as symptom triggers
- Evidence linking specific foods to FD symptoms is limited
- Dietary advice is often empirical, leading to exclusion diets



Mixed messages

2025: Googling the entries "dyspepsia AND diet" returns 1,110,00 results --- high public interest on this topic.

Exclusion diets may:

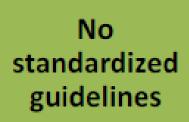
- Lead to over restriction, poor diet quality, nutrient deficiencies
- Reinforce food intolerance perceptions and dyspepsia mechanisms (e.g. hypervigilance)

My patients after Googling 'best diet for dyspepsia' and getting 7 different answers:



Inducers (fatty, spicy foods, soft drinks, wheat products, products containing caffeine, alcohol)

Suppressants (apples, rice, bread, caraway seed, dates, honey, yogurt, quince, and walnuts)



(+) FODMAP diets with variations
(-) ultra-processed foods-Westernized dietary pattern (high fat content, additives)
(+) fruits & vegetables-Mediterranean diet

Functional dyspepsia (FD)

Dietary patterns



Foods

Herbal treatments

behaviors

Eating



At least 3 main meals and 3-5 snacks per day. Late-night snacking, skipping of one or more main meals during the day, irregular meals, dining out, chewing speed and meal duration are of moderate importance

Herbs, ginger, Mastiha, peppermint, caraway- oil have anti-inflammatory effects, contribute to improvement in the function of gut microbiota, immune system, central stimuli and intestinal motility in FD

Goals of Nutrition Therapy



Optimize Nutrition

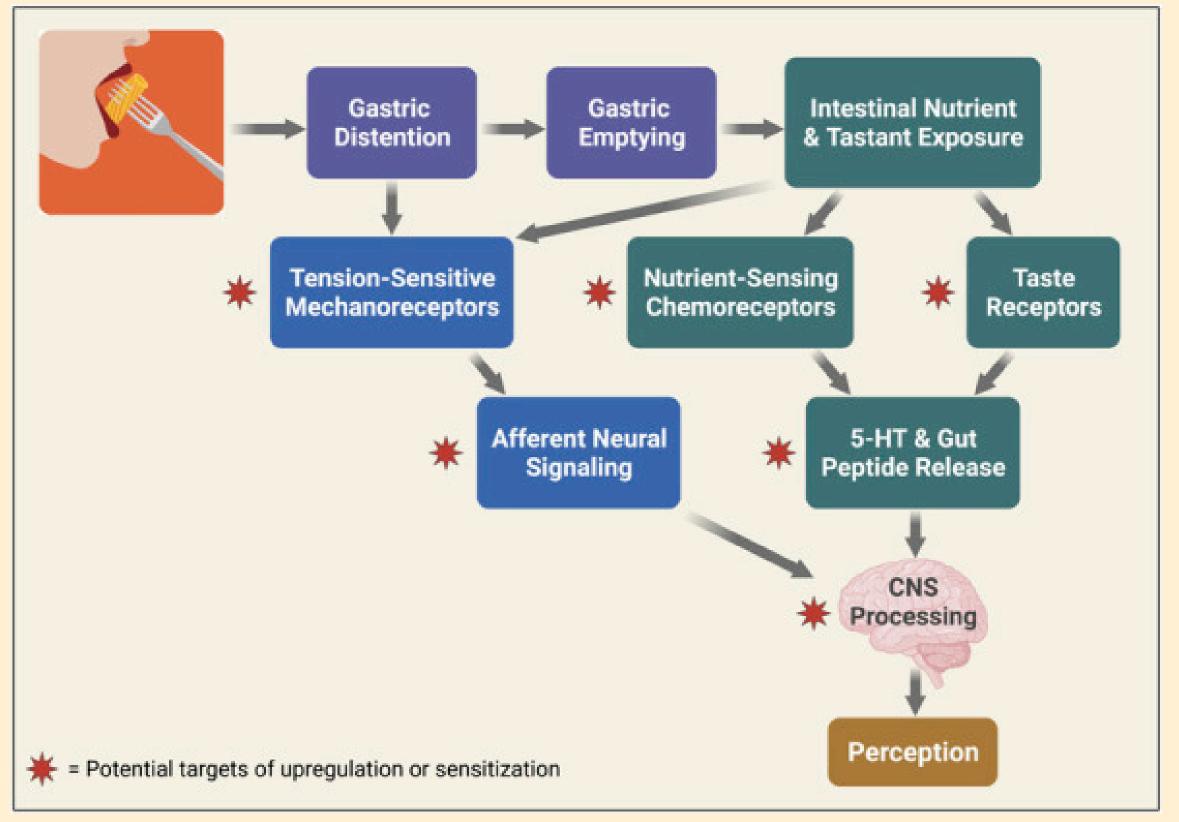


Minimize Symptoms



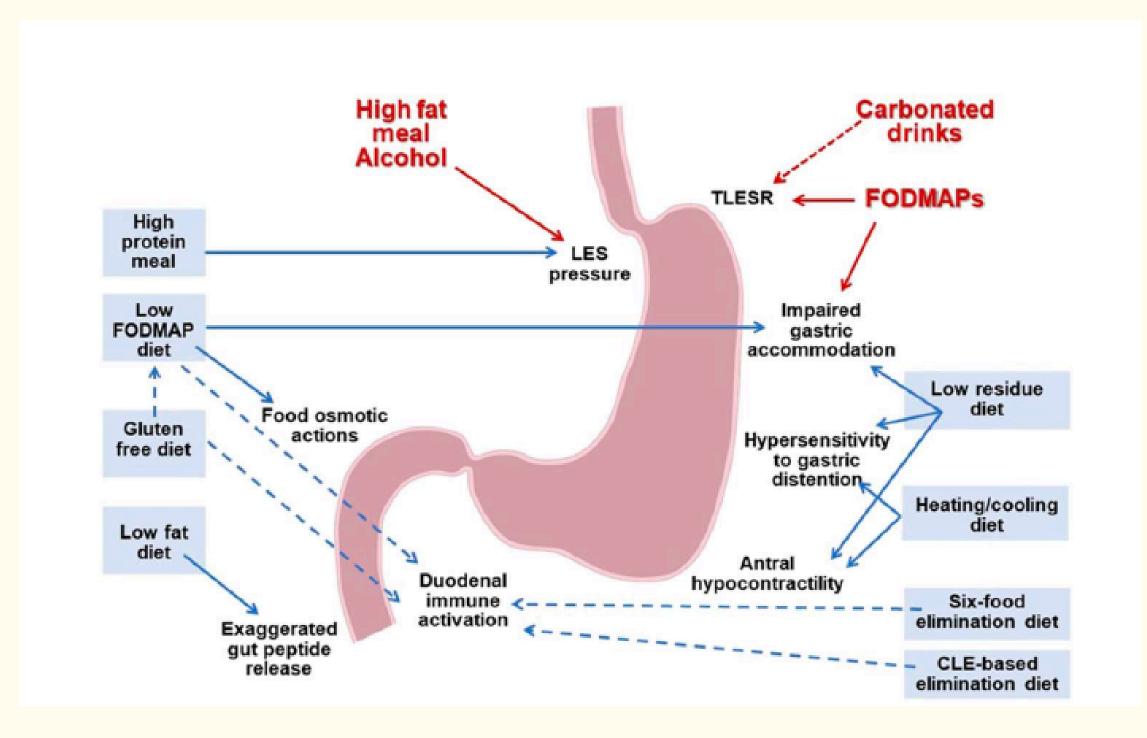
Maintain or Build Positive Relationship with Food

How the Gut Senses Food: Mechanosensitive, Chemosensitive, and Thermosensitive Pathways



Nutrient-specific responses:

- High-fat meals → delayed gastric emptying, increased fundic relaxation.
- Spicy foods → capsaicin and TRPV1 receptor activation, may worsen symptoms in some but benefit others long term.
- Alcohol and Caffeine → ability to increase gastric acid secretion and impair gastric motility.
- Food proteins → duodenal allergic reactions may contribute to FD symptoms, based on findings from 6FED & CLE.





The Low FODMAP Diet

- Emerging evidence suggests a low FODMAP diet may improve FD symptoms
- Proposed mechanisms behind symptom improvement include:
 - ↓ Fermentation and gas production
 - ↓ Duodenal distension
 - ↓ Visceral hypersensitivity
 - ↑ Duodenal transepithelial electrical resistance
 - → Suggesting enhanced mucosal integrity

PHASE 1
Reduce total
FODMAP
intake

Dietitian review

- Reduce FODMAP intake
- 2-8 weeks
- Replace with suitable low FODMAP alternatives from the same food group

If no response occurs, return to usual diet and trial alternate treatment

PHASE 2 Rechallenge to assess tolerance

Dietitian review

Individual rechallenge of each FODMAP subgroup:

- Fructan, e.g. wheat, onion
- •GOS, e.g. legumes/pulses
- Lactose e.g milk
- Excess fructose, e.g. Honey
- ·Polyols, e.g. avocado

Challenge over 2-3 days and monitor symptom response. Order of challenges based on nutritional need and patient preferences.

PHASE 3 Long term maintenance

Dietitian review

Individualized diet based on response to food challenges:

- Tolerated foods reintroduce freely
- Foods causing mild/moderate symptoms – reintroduce when able
- Foods causing severe symptoms – avoid

Continue to challenge poorly tolerated foods in the long-term

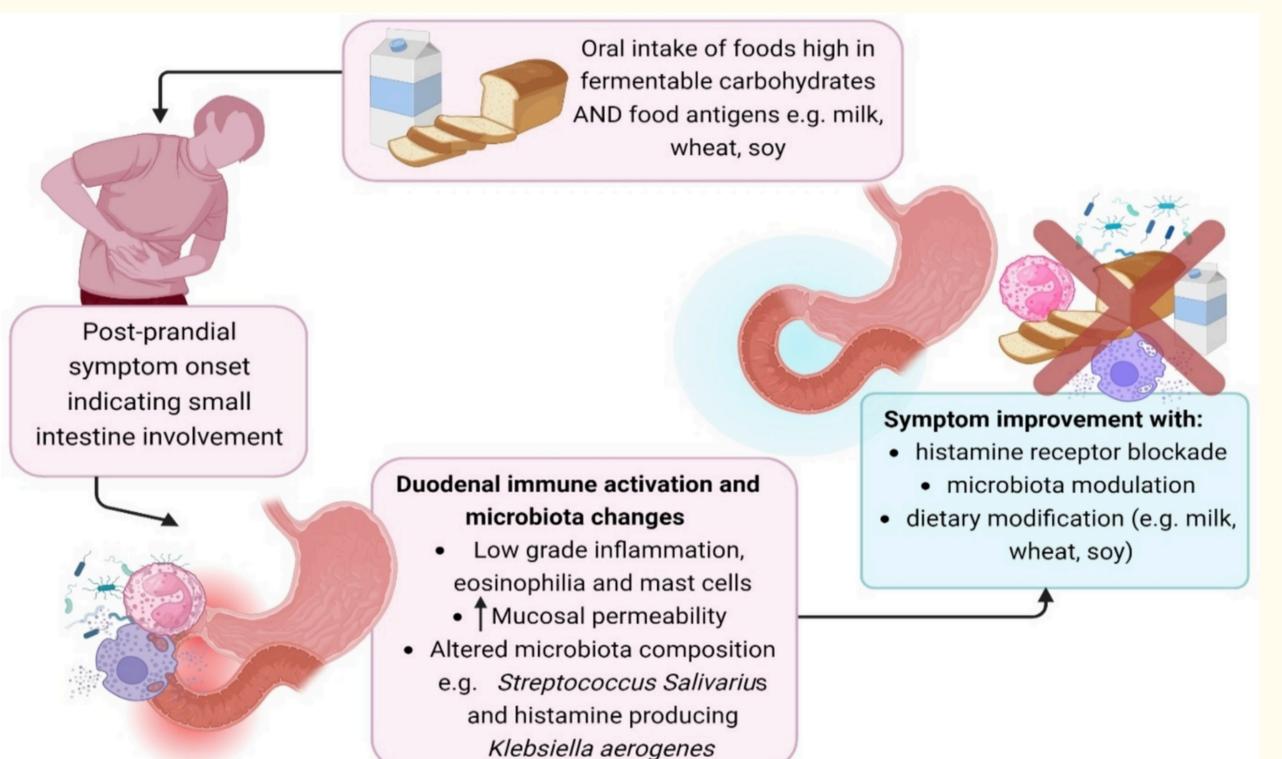
FD symptom recurrence during blinded FODMAP reintroduction phase:

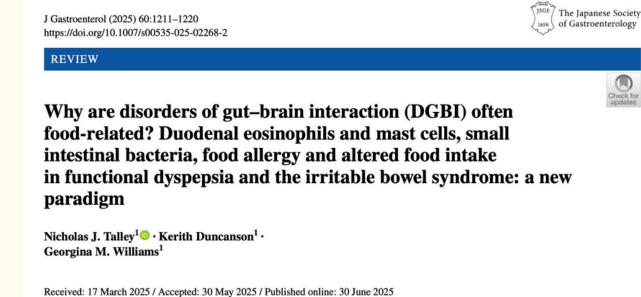
Mannitol & GOS (both 29%), Fructans (21%), Sorbitol & Fructose (14%), Lactose (12%)





Emerging Mechanisms – Food, Immunity & Microbiota in DGBIs



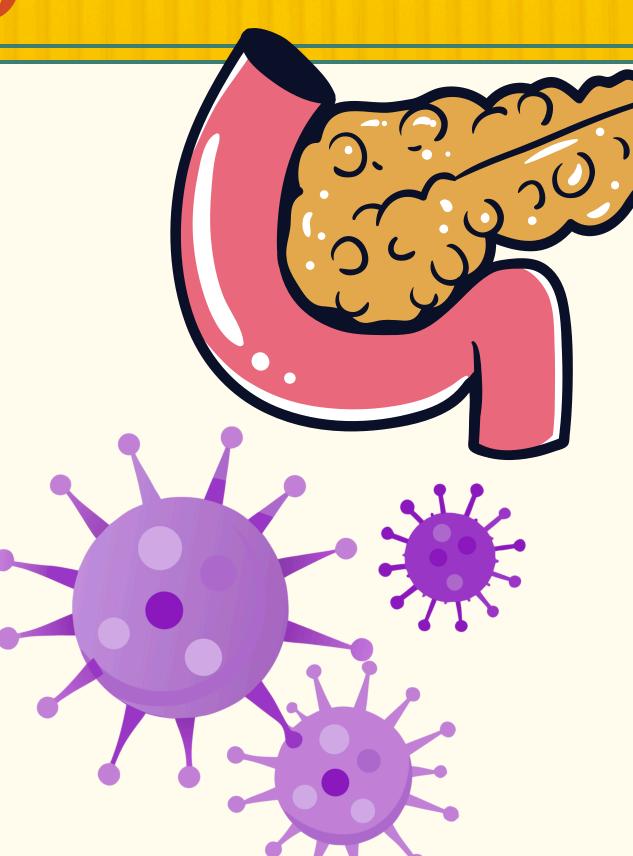


 Possible pathway: Food antigens (milk, wheat, soy) and fermentable carbs may trigger duodenal immune activation and microbiota changes → symptom generation

Duodenal Microbiota and Diet in FD



- FD is associated with altered duodenal microbiota compared to healthy controls.
- Fiber & ↑ refined carbs → ↓ diversity, ↑ symptoms
 (Saffouri et al.).
- Streptococcus is the dominant genus in the duodenum; higher bacterial load → ↑ symptoms, ↓ quality of life.
- Streptococcus salivarius identified in FD; more common in those with wheat sensitivity.
- *Streptococci* may digest wheat proteins → increased antigen exposure and immune activation.



Duodenal Microbiota and Diet in FD

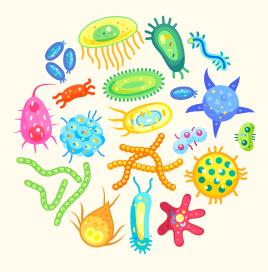


- FD participants showed altered oral, duodenal, and stool microbiota
- Duodenal Changes in FD:
 - Inflammation ↑
 - Tight junction protein expression ↓ (impaired barrier)
- Microbiota Alterations:
 - Duodenal Streptococcus ↑ (p=0.014) → correlated
 with impaired tight junctions and symptom severity
 - Stool Butyricicoccus ↓ (p=0.047) → correlated with symptom severity

Dietary Associations:

- Higher protein, PUFA, and valine intake →
 associated with improved duodenal
 barrier function
- Duodenal Streptococcus abundance → linked with lower carbohydrate intake





Impact of Milk Elimination on FD Symptoms



Design:

- Cross-sectional cohort study, 120 patients with FD (Rome IV)
- 1-month follow-up
- 2 groups:
 - Milk/dairy elimination (no medical treatment)
 - Unrestricted diet
- Symptoms measured by Gastrointestinal Symptom Rating Scale (GSRS)

Results:

- Both groups improved over 1 month
- Milk elimination group had greater reduction in:
 - Total GSRS symptom score (p = 0.01)
 - 7 of 15 symptom subsets (p < 0.05)
- No significant differences in BMI, FD subtype, or endoscopic findings

Removing milk and dairy for 4 weeks led to greater symptom improvement vs. unrestricted diet — but dietary intake beyond milk was not controlled.



Arab Journal of Gastroenterology

Volume 25, Issue 4, November 2024, Pages 390-398



Original Article

The efficacy of removal of animal milk from the diet in functional dyspepsia: A crosssectional study

 $\underline{\text{Atilla Akpınar}^a}$, $\underline{\text{Koray Kochan}^b}$, $\underline{\text{Sercan Kiremitci}^b}$, $\underline{\text{Gulseren Seven}^b}$, $\underline{\text{Ali Tuzun Ince}^b}$, $\underline{\text{Hakan Senturk}^b}$



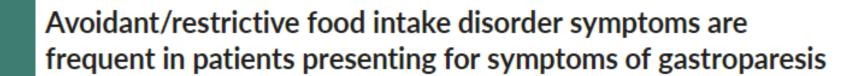
- Looked at the presence of FED symptoms in patients presenting with symptoms of GP
- 55% of patients with symptoms of GP had clinically significant FED symptoms,
 - ARFID (23%-40%).
 - 11.7% had restrictive eating disorders (anorexia nervosa; unspecified FED)
- The more severe the GP symptoms, the increased likelihood of having FED symptoms.
- Screening gastroparesis/dyspepsia pts for avoidant/restrictive eating habits, particularly ARFID, may facilitate behavioral treatment recommendations, which could improve GI symptoms.

Received: 28 April 2020 Revised: 29 May 2020

DOI: 10.1111/nmo.13931

ORIGINAL ARTICLE





Helen Burton Murray^{1,2,3} | Asad Jehangir⁴ | Casey J. Silvernale³ | Braden Kuo^{2,3} | Henry P. Parkman⁴

Accepted: 9 June 2020

¹Department of Psychiatry, Eating Disorders Clinical and Research Program, Massachusetts General Hospital, Boston, Massachusetts, USA

²Harvard Medical School, Boston, Massachusetts, USA

^aNeuroenteric Research Laboratory, Massachusetts General Hospital, Gastroenterology, Boston, Massachusetts, USA

⁴Gastroenterology Division, Temple University Hospital, Philadelphia, Pennsylvania, USA

Correspondence

Helen Burton Murray, Department of Psychiatry, Massachusetts General Hospital, 2 Longfellow Place, Suite 200, Boston, MA 02114, USA.

Email: hbmurray@mgh.harvard.edu

Listen to the podcast for this article.

Abstract

Introduction: Patients with symptoms of gastroparesis/dyspepsia often avoid foods or restrict eating for symptom management. There is growing interest in understanding risk for feeding/eating disorders (FEDs) like avoidant/restrictive food intake disorder (ARFID). Among patients presenting with gastroparesis/dyspepsia symptoms, we aimed to determine: (a) FED symptom frequency, and (b) relation of FED symptoms to gastrointestinal symptom severity and gastric retention abnormalities.

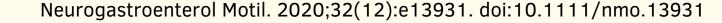
Methods: Adult patients (N = 288; 78% female) referred for gastroparesis/dyspepsia symptoms at two academic medical centers from January 2018—February 2019 completed self-report surveys for gastrointestinal symptom severity and FED symptoms. Gastric retention data were available for 210 patients, using 4-hour EggBeater gastric emptying scintigraphy (GES).

Results: Clinically significant FED symptoms were present in 158 patients (54.9%). Interestingly, 115 patients (39.9%) met conservative self-report cutoff for ARFID symptoms, with 67 (23.3%) patients having documented psychosocial/medical impairment. Of those with survey data for other FEDs (n = 239), only 28 patients (11.7%) had restrictive eating disorders (anorexia nervosa; unspecified FED). Likelihood of having FED symptoms was significantly associated with greater gastroparesis symptom severity (OR = 2.23, P < .001), but not GES. In addition, gastroparesis symptom severity was moderately and significantly associated with greater ARFID symptom severity (b = 0.45, P < .001), but neither GES nor other FED symptoms.

Discussion: In patients presenting with gastroparesis/dyspepsia symptoms, FED symptoms were frequent (55%), particularly ARFID, and were associated with greater gastrointestinal symptom severity, but not gastric retention. Gastroparesis/dyspepsia symptoms may mimic FEDs, particularly ARFID. Clinicians should be cautious about diagnosing ARFID in gastroparesis/dyspepsia patients, and screening for ARFID could assist behavioral treatment referral.

KEYWORDS

avoidant/restrictive food intake disorder, eating disorder, feeding disorder, functional dyspepsia, gastric retention, gastroparesis



The Power of Expectation: Placebo and Nocebo Responses in FD



- High placebo response: 29% in gastroparesis(1), 39% in FD(2)
- Nocebo response: 26% (meta-analysis of 27 RCTs)(3),
 - Common reported AEs on placebo: nasopharyngitis (9%), constipation (6%), headache (5%), diarrhea (3%)
- Expectation, perception, and context strongly influence symptom experience
- Highlights importance of communication and reassurance in care



^{1.} Wise JL et al. Clin Gastroenterol Hepatol. 2023;21::1447-61:.

^{2.} Bosman M et al. Neurogastroenterol Motil 2023;35:e14474.

^{3.} Li RJ et al. J Dig Dis. 2023; 24(8-9):440-451.

Physical Activity: Promoting Health and Digestive Comfort

>>>

- Mild physical activity enhances intestinal gas clearance and reduces symptoms in patients complaining of abdominal bloating. (1)
- Engaging in moderate-intensity aerobic exercise for at least 180 min per week positively impacts IBS symptoms and Physical Capacity.(2)
 - 12-week group walking program, FITT principle
 - IBS-SSS: Total score significantly reduced by 39% compared to baseline and for Abdominal Distension.

PA & the Gut

- Enhances motility
- Improves blood flow
- Anti-inflammatory mechanisms
- Stress reduction
- Positive effects on gut microbiota diversity

^{1.} Villoria A, et al. Am J Gastroenterol. 2006;101(11):2552-2557. doi:10.1111/j.1572-0241.2006.00873.xwalking

^{2.} Bianco A, et al. J Clin Med. 2023;12(21):6786. Published 2023 Oct 26. doi:10.3390/jcm12216786



Effect of Moderate Aerobic Exercises on Symptoms of FD

- Participants: 72 patients with FD
- Groups:

• Control: Conventional treatment only (n=36)

Experimental: Aerobic exercise (30 min/session, 5x/week for 6 weeks) + conventional treatment (n=36)

Assessments:

- Glasgow Dyspepsia Severity Score (GDSS)
- Depression Anxiety Stress Scales-42 (DASS-42)
- Visual Analogue Scale (VAS)
 Measured at baseline and 6 weeks

 Both groups improved, but the experimental group showed significantly greater improvement:

• GDSS, DASS-42, VAS: p < 0.0002

within groups

- **Between-group comparison** at 6 weeks: **p < 0.05** favoring exercise group
- Aerobic exercise as an adjunct to conventional therapy leads to better symptom control and functional wellbeing in FD.





Impact of Low FODMAP Diet & Aerobic Exercise on FD

- Type: Prospective randomized controlled trial
- Duration: 24 weeks
- Participants: 70 patients with FD
- Groups:
 - Intervention: Low FODMAP + Aerobic Exercise + Conventional Therapy
 - Control: Conventional Therapy only
- Outcomes Measured:
 - SAGIS: GI symptom severity
 - SF-NDI: Health-related quality of life



Impact of Low FODMAP Diet & Aerobic Exercise on FD

Results:

- Symptom Reduction (SAGIS):
 - Mean difference: -3.571 ± 1.044
 - P = 0.001
- Quality of Life Improvement (SF-NDI):
 - Mean difference: -12.086 ± 1.394
 - P < 0.001

Combined lifestyle intervention significantly:

- Reduced epigastric symptoms
- Improved quality of life
- Outperformed conventional therapy alone





Correction of Dyssynergic Defecation Reduces FD Symptoms



RCT (n = 50) FD (PDS subtype) + functional constipation + dyssynergic defecation

Intervention (4 weeks):

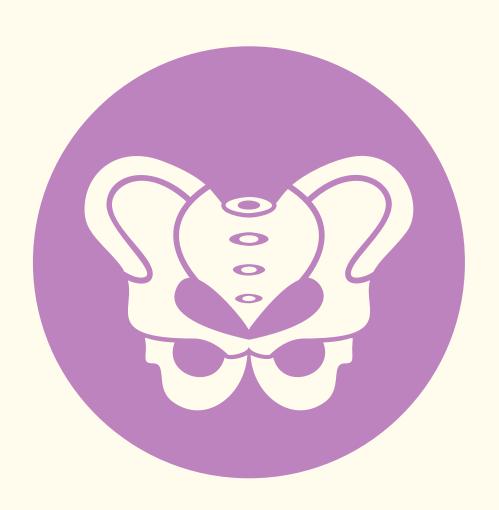
- Biofeedback therapy (2-3 sessions + daily pelvic floor exercises)
- vs. Fiber supplementation (3.5 g plantago ovata/day)

Results:

- Biofeedback corrected dyssynergic defecation in 19/25 participants
- → Postprandial fullness by 22% (p < .001)
- **↓** Anal evacuations by 21% (p = .009)
- Fiber supplementation: no improvement in fullness or evacuations

Conclusion:

- Treating pelvic floor dysfunction, not just the upper GI tract, can significantly reduce dyspeptic symptoms.
- Highlights: lower GI function can influence upper GI symptom expression.



Sleep and Quality of Life in FD



- 81% of patients with FD had altered sleep quality
- 61% met criteria for insomnia
- Poor sleep associated with:
 - → Quality of life (GIQLI: 75 vs. 92)
 - ↑ Symptom severity
 - ↑ Anxiety and depression
- Predictors of poor sleep: older age, higher depression scores, greater FD symptom severity



ORIGINAL RESEARCI published: 08 February 202

Check for updates

Sleep Quality and Insomnia Are Associated With Quality of Life in Functional Dyspepsia

Fabien Wuestenberghs^{1,2,3}, Chloé Melchior^{2,4,5,6}, Charlotte Desprez^{1,2}, Anne-Marie Leroi^{1,2,5}, Marie Netchitailo¹ and Guillaume Gourcerol^{1,2,5*}



¹ Department of Physiology, Rouen University Hospital, UNIROUEN, Normandy University, Rouen, France, ² INSERM Unit 1073, UNIROUEN, Normandy University, Rouen, France, ³ Department of Gastroenterology and Hepatology, CHU UCL Namur, Université catholique de Louvain, Yvoir, Belgium, ⁴ Department of Gastroenterology, Rouen University Hospital, UNIROUEN, Normandy University, Rouen, France, ⁵ INSERM CIC-CRB 1404, Rouen University Hospital, UNIROUEN, Normandy University, Rouen, France, ⁶ Department of Molecular and Clinical Medicine, Institute of Medicine, Sahlgrenska Academy, University of Gothenburg, Gothenburg, Sweden

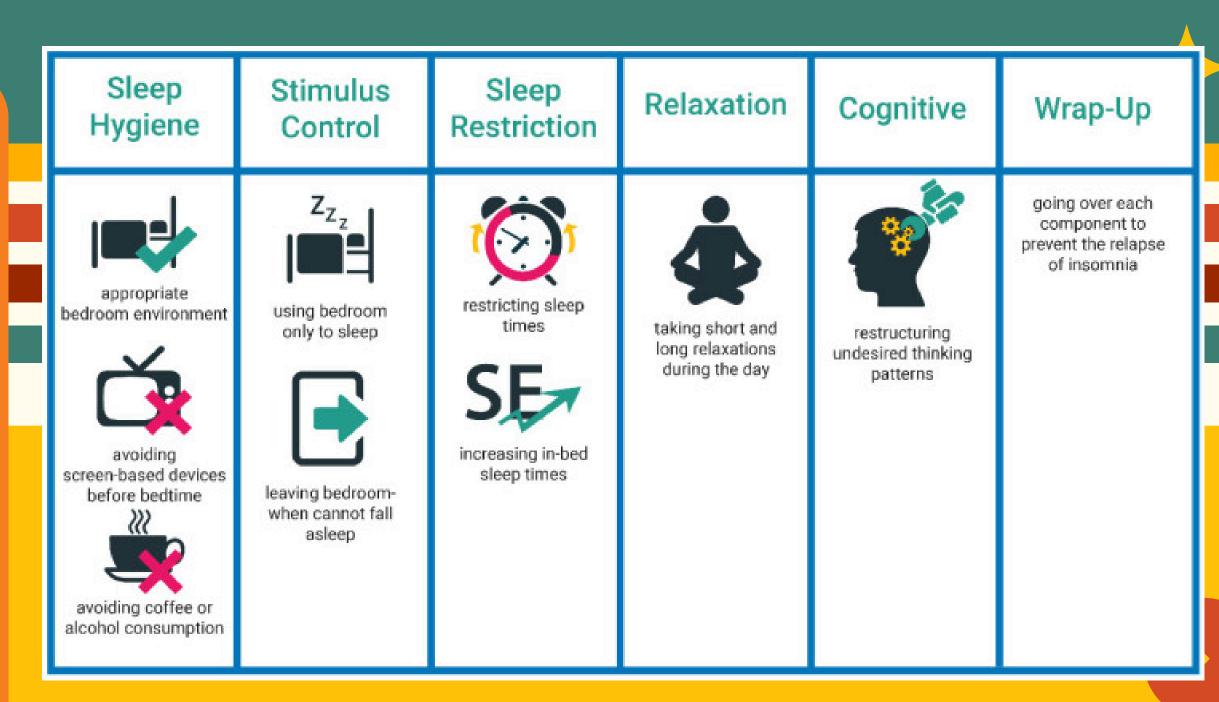
Cognitive Behavioral Therapy for Insomnia (CBT-I)

Gold standard behavioral treatment

Delivered by: therapist - 1:1, in groups, phone, or workshops or by an app.

Psychology Today: "Find a Sleep or Insomnia Therapist"

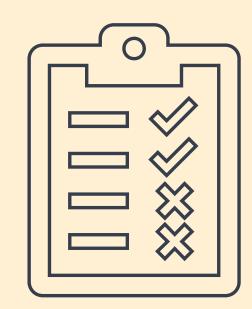
Apps: Sleepio, Shuti, CBT-I coach, Sleeprate, Nightowl



Nutrition Assessment

Key Considerations

- GI symptoms: Type, severity, timing, and frequency
- Food exclusions: Any current restrictions or avoided foods
- Perceived triggers or helpful patterns: Foods or eating habits that improve or worsen symptoms
- **Previous interventions**: Diets already trialed (e.g., low FODMAP, gluten-free, elimination diets)
- Cooking habits: Frequency of dining out, cooking skills, and confidence
- Diet quality & eating patterns:
 - General fiber intake (low, moderate, high)
 - Food security
 - Meal size and frequency
 - Intake of common triggers: high-fat, alcohol, spicy foods, coffee, carbonation, etc.
- Supplements: Type, dose, frequency, reason for use





Herbal Treatments



Supplement	Mechanism of Action	Evidence & Clinical Utility	Notes
Ginger	Anti-inflammatory, prokinetic, antiemetic	Enhances gastric emptying and motility; reduces nausea, vomiting, and inflammation. Polyphenols support GI function.	Typical dose: 1,000–1,500 mg/day. Caution with anticoagulants.
Peppermint & Caraway Oil	Spasmolytic, prokinetic effect, anti-inflammatory, gastroprotective	More effective than placebo in reducing epigastric pain and improving dyspeptic symptoms	Use enteric-coated formulas.
STW-5	Enhances antral motility and gastric accommodation	Nine-herb formula shown to reduce overall FD symptoms including early satiety and bloating.	Contains alcohol; caution in pregnancy and liver disease.





Putting it all Together: Nutrition

Inducers vs Suppressants:

Potential for diet expansion?

• Dietary Pattern:

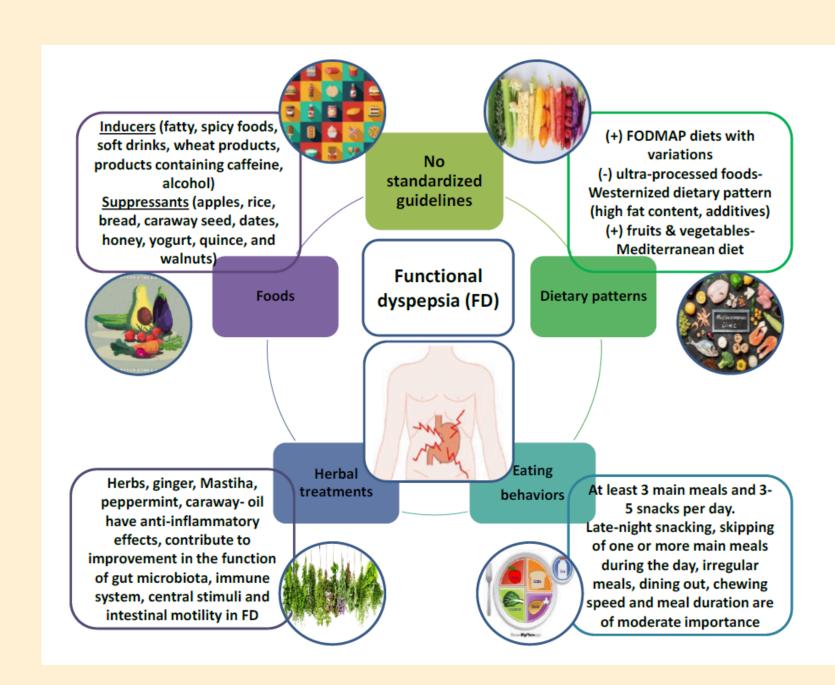
- Assess FODMAP intake
 - Wheat: gluten vs fructan;
 - Dairy vs lactose intolerance?
- Constipated? Kiwifruit: supports regularly + sleep

• Eating Behaviors:

- Assess for & encourage consistent meal routines
- o Emphasize small, frequent meals and mindful eating
- Late-snack snacking? Can impact sleep.

Herbal Treatments:

Consider ginger or peppermint-carraway oil



Putting it all Together: Lifestyle

- Encourage physical activity
- Review sleep hygiene and stress modulation (CBT-I, mindfulness)
- Collaborate with HCP & psych for integrated care



