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The impact of diet on functional dyspepsia: a critical review of current evidence

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Abstract

This review examines the relationship between diet and functional dyspepsia (FD), a prevalent disorder of gut–brain interaction affecting 8% of the global population and characterised by postprandial fullness, early satiety and epigastric pain or burning. Despite 40–70% of FD patients reporting symptom onset within minutes of eating, standardised dietary recommendations remain limited. The pathophysiological mechanisms underlying food-related symptoms in FD involve complex interactions between altered gastric accommodation and emptying, visceral hypersensitivity, duodenal immune activation and small intestinal microbial dysbiosis. Current evidence most strongly supports dietary lipids as potent triggers of dyspeptic symptoms, likely mediated through cholecystokinin pathways and heightened visceral sensitivity. Additionally, emerging research indicates potential benefits of fermentable carbohydrate restriction, with the low FODMAP (fermentable oligosaccharides, disaccharides, monosaccharides and polyols) diet showing promise particularly for patients with postprandial distress syndrome. Other dietary factors such as alcohol, coffee, food chemicals, bioactive compounds and meal patterns may also influence FD symptoms though current evidence remains insufficient to inform clinical practice. While existing evidence provides a foundation for understanding diet–symptom relationships in FD, significant gaps remain in translating mechanistic insights into personalised dietary recommendations. Future research should focus on developing evidence-based dietary strategies tailored to FD subtypes, ensuring nutritional adequacy while addressing the complex interplay between nutrient sensing, duodenal immune activation and gut microbiota in symptom generation.

Introduction

Functional dyspepsia (FD) is a disorder of gut–brain interaction (DGBI), characterised by a complex of upper gastrointestinal symptoms such as epigastric pain or burning, postprandial fullness and early satiation[1,2]. As one of the most prevalent DGBI, FD affects approximately 8% of the global population[3,4], with higher incidence observed in developing countries[2,3]. The economic impact of FD is extensive, with annual costs reaching up to $18.4 billion USD in the United States[4–6]. This financial burden stems from frequent healthcare utilisation, extensive diagnostic testing, and indirectly from reduced work productivity and absenteeism[4,6,7].

FD is classified according to the Rome IV criteria, which defines FD as the presence of one or more bothersome dyspeptic symptoms (postprandial fullness, early satiety, epigastric pain and/or epigastric burning) persisting for at least 3 months with symptom onset at least 6 months prior to diagnosis, with exclusion of structural disease at upper endoscopy[1,2]. The Rome IV criteria further distinguish FD into two subtypes. Postprandial distress syndrome (PDS) is the most common subtype (66.6%), characterised by meal-related symptoms of early satiation and/or postprandial fullness and, Epigastric Pain Syndrome (EPS) (15.3%), associated with epigastric pain and/or burning. There is recognised overlap (18.1%) between these two subgroups[1,2,8]. FD typically follows a chronic, relapsing–remitting course with only about 10% of affected individuals achieving long-term symptom resolution[9].

Women consistently show higher FD prevalence than men[3,10,11], possibly due to biological differences in gastrointestinal function, visceral hypersensitivity, central nervous system processing and sex hormone-related influences[3,12]. Risk factors include gastrointestinal infections (including *H. pylori* or traveller's diarrhoea)[10,13], non-steroidal anti-inflammatory drug (NSAID) use, smoking[10] and visceral adiposity[14,15]. The relationship between *H. pylori* and FD is complex; *H. pylori* is considered an organic cause of dyspepsia and a separate entity from FD, though FD can be considered if symptoms persist after 6 months following eradication[16].

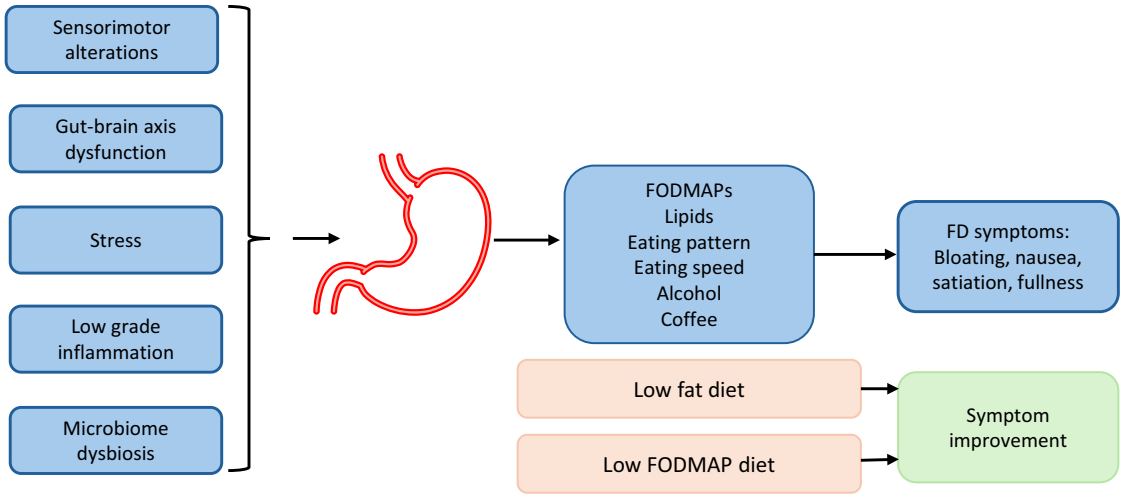

Figure 1. Pathophysiological mechanisms linking diet and symptoms in functional dyspepsia. Dietary components interact with altered gastrointestinal physiology in FD, including visceral hypersensitivity, impaired gastric accommodation, duodenal inflammation and microbial dysbiosis. These interactions trigger symptoms via chemosensory pathways, mechanical distension, immune activation, and altered gut–brain signalling. Targeted dietary strategies may improve symptoms by modulating these underlying processes.

FD frequently coexists with other DGBI, particularly irritable bowel syndrome (IBS)[17], with up to 37% of FD patients having concomitant IBS – an 8-fold increase compared to the general population[18]. Additionally, FD often overlaps with gastro-esophageal reflux disease (GERD) and functional heartburn, with up to 50% of FD patients reporting regular reflux symptoms[2,19,20]. This symptom overlap complicates the diagnosis and suggests shared pathophysiological mechanisms.

Between 40–70% of FD patients report symptom onset within 15–45 minutes of eating, including symptoms such as belching, nausea, bloating, burning, epigastric pressure and early satiation[17,21–23]. This temporal relationship strongly indicates a direct connection between food intake and symptom generation, as illustrated in Figure 1. Carbone *et al.* analysed temporal patterns of postprandial symptoms in FD patients completed over six hours following standardised meals, confirming distinct symptom patterns in EPS and PDS subgroups[23]. PDS and PDS/EPS groups exhibited similar symptom patterns, with severity peaking rapidly between 30 and 90 minutes after meal ingestion. In contrast, EPS symptoms did not consistently correlate with meal intake. The study additionally found that PDS symptoms (fullness, early satiation) originate from the stomach while EPS symptoms (epigastric pain or burning) suggests duodenal or jejunal origin[23]. This temporal pattern provides important clues about the anatomical origins of different FD symptoms.

Current treatment options for FD remain limited in efficacy. Pharmacological therapy, including proton pump inhibitors, *H. pylori* eradication therapy, prokinetics and neuromodulators yield modest benefits and often fail to provide sustained symptom relief[24–26]. Despite evidence that 40–70% of FD patients experience symptoms within minutes of eating, dietary management remains neglected in clinical guidelines[17,21,22]. Unlike GERD or IBS, no standardised dietary recommendations exist for FD, leaving both patients and clinicians without evidence-based guidance[27,28]. This gap is particularly concerning given the central role of food in symptom provocation and the significant impact on patients' quality of life[17,21,22].

This review aims to: (1) summarise the pathophysiological mechanisms underlying food-symptom relationships in FD; (2) critically evaluate evidence linking specific nutrients and food components to symptom generation; (3) assess the impact of eating behaviours on symptom manifestation; and (4) provide clinically relevant dietary management strategies for nutrition professionals. By synthesising current evidence, we establish a foundation for developing targeted dietary interventions for this challenging condition.

Overview of FD pathophysiology

Gastric sensorimotor dysfunction

The gastrointestinal tract utilises sophisticated networks of receptors and signalling pathways to detect and respond to nutrients via three primary sensory modalities: chemosensitivity (nutrient composition), thermosensitivity (temperature) and mechanosensitivity (pressure and distension)[29]. Gastric sensing of food is primarily mediated by mechanosensitive pathways that signal to the brain via vagal afferent nerves, with this process modulated by the gastric accommodation reflex[30–32].

In the small intestine, specialised chemosensors detect changes in pH, osmolarity and nutrient composition[29], triggering the release of gut peptide hormones such as cholecystokinin (CCK), glucagon-like peptide-1 (GLP-1) and peptide YY (PYY)[29,33]. These hormones influence gastric emptying, appetite regulation and energy intake through both local and systemic effects[29].

FD is associated with alterations in these sensory processes. Visceral hypersensitivity – an exaggerated response to normal physiological stimuli – is a key pathophysiological feature of FD, particularly sensitivity to chemical and mechanical stimuli which has been associated with weight loss, belching and epigastric pain[17,29,34–38]. Impaired gastric accommodation correlates with reduced drinking capacity, early satiety, postprandial fullness and weight loss[39–42], while delayed gastric emptying more commonly presents with nausea, vomiting, postprandial fullness and early satiety[43,44]. However, the correlation between delayed gastric emptying and FD symptoms remains inconsistent[45]. These disturbances in gastric sensorimotor function serve as key targets for treatment strategies showing beneficial symptomatic effects when addressed[41,46–48].

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Duodenal inflammation and microbiota dysbiosis

Low-grade mucosal inflammation, characterised by increased eosinophils and mast cells has emerged as a significant pathophysiological mechanism in FD[49]. The activation of these inflammatory cells leads to the release of pro-inflammatory cytokines, resulting in tissue damage, disrupted epithelial barriers and altered enteric nerve function[50]. This process increases epithelial permeability, potentially allowing greater infiltration of luminal antigens and amplifying immune responses[2]. Recent evidence points to small intestinal microbial dysbiosis as a potential contributor to FD pathophysiology[51–53]. Studies have identified significant alterations in duodenal mucosal microbial composition in FD patients, including increased abundance of Streptococcus spp. and decreased anaerobic genera such as Prevotella[52,53]. Importantly, duodenal bacterial load has been negatively correlated with quality of life and positively associated with symptom severity in FD patients[51]. Symptoms including pain, nausea and epigastric discomfort, as well as impaired gastric emptying, have been correlated with elevated levels of small bowel-homing T cells and pro-inflammatory cytokine, suggesting that motor dysfunction may be secondary to duodenal inflammation and microbial alterations[54,55].

Ford *et al.* propose that physiological or psychological insults can lead to loss of tolerance to previously tolerated food antigens, enabling interaction between microbiota, food antigens and the immune system, resulting in localised inflammatory responses[56]. Supporting this, recent studies have identified small intestinal dysbiosis associated with both the development and occurrence of FD[51,57–60].

Gut–brain axis dysregulation

Dysfunction of the gut–brain axis plays an important role in FD pathophysiology. Psychological factors, particularly stress, exacerbate symptoms in many FD patients, with up to 50% identifying stress as a clear symptom trigger[61]. Studies demonstrate that stress, through corticotrophin-releasing hormone release, can significantly impact gastrointestinal function by increasing epithelial permeability and altering immune function[62,63]. Notably, anxiety has been associated with duodenal eosinophilia in FD, highlighting the interplay between psychological stress and immune activation[64].

Neuroimaging studies have identified structural and functional alterations in brain regions involved in the processing of visceral input[65,66]. These findings support bidirectional gut–brain alterations in FD, where overactive visceral signalling from the gut may heighten symptom perception, while central sensitisation may amplify symptom severity.

These interacting pathophysiological mechanisms – sensorimotor dysfunction, duodenal inflammation, microbial dysbiosis and altered gut–brain signaling – create multiple potential targets for dietary intervention in FD management.

Nutrient-symptom relationships

Lipids: primary triggers of symptoms in FD

FD patients frequently report sensitivity to high-fat foods, and a systematic review examining the relationship between food and FD found dietary fats to be consistently associated with PDS symptoms[67]. Barbera *et al.* validated the nutrient-specific effects of lipids in FD in a cohort of 18 dyspeptic patients and 9 healthy subjects. Their study demonstrated that isocaloric duodenal glucose infusion did not produce the same symptomatic response of epigastric fullness and discomfort as lipid infusion during gastric distension in FD patients. Healthy controls experienced no symptoms in response to either duodenal glucose or lipid infusions[68]. The same group later demonstrated that duodenal lipid infusion increased gastric sensitivity to distension in FD patients, provoking symptoms of nausea and epigastric bloating absent in healthy controls[69]. Both studies reported no differences in gastric motor response between healthy controls and FD patients during distension, suggesting that FD symptoms may stem from visceral hypersensitivity[69]. Interestingly, while duodenal lipid infusion heightened gastric sensitivity to distention in FD patients, it had the opposite effect in healthy controls and induced gastric relaxation – potentially an adaptive response to accommodate greater volumes of nutrient-dense food[68,69].

Feinle *et al.* found duodenal lipid infusion induced dyspeptic symptoms of fullness, epigastric discomfort and nausea in response to gastric distension[34]. The study additionally demonstrated that symptoms experienced by FD patients during lipid infusion and gastric distension were significantly alleviated by CCK-A receptor antagonist dexloxiglumide, thus confirming the likely involvement of CCK-A receptors in mediating lipid-induced dyspeptic symptoms[34]. Bharucha *et al.* confirmed this association between plasma CCK levels and symptom severity (nausea, fulness and bloating) during lipid infusion[70]. The increase in CCK observed in both healthy controls and FD patients suggests that hypersensitivity to normal CCK levels, rather than excessive CCK secretion, underpins the pathophysiological involvement of CCK in FD symptom generation[34,70]. Notably, both studies demonstrated exacerbations in FD symptoms during lipid infusion even in the absence of gastric distensions, thus implicating chemosensitivity as a potential mechanism in the pathophysiological response to lipids[34,70]. A single study has evaluated the relationship between increasing doses of duodenal lipid infusion, plasma CCK levels and symptom severity in FD. Using 10% and 20% Intralipid infusions, the study demonstrated a clear dose-dependent relationship where higher lipid doses were associated with increased plasma CCK concentrations and intensified symptoms of fullness, epigastric discomfort and nausea[34]. The release of CCK contributes to symptom generation in FD through multiple mechanisms: (1) direct activation of vagal afferents by carrying sensory information to the brain, (2) increased sensitivity of gastric mechanoreceptors to distension, (3) increased chemosensitivity to presence of duodenal lipid (4) reduced gastric motility and (5) delayed gastric emptying[21,34,70,71]. These effects are exaggerated in FD patients compared to healthy controls, suggesting hypersensitivity to normal physiological CCK responses rather than excessive CCK production.

While duodenal infusion studies provide valuable mechanistic insights into the role of lipids in FD, they do not replicate normal eating conditions. These studies bypass normal digestive processes, are invasive, and often involve unnaturally high lipid concentrations. Yet, these mechanistic findings are supported by diet studies comparing high versus low-fat meals. A high-fat meal, simulated by adding 30 g of margarine to soup, elicited greater symptomatic response (epigastric pain and nausea) in FD patients compared to a low-fat soup without added fat[72]. Furthermore, a high-fat yoghurt (24 g fat, 330 kcal) significantly increased symptoms of fullness, bloating and nausea compared with low-fat yoghurt (1 g fat, 143 kcal) in FD patients[73]. Both studies may be confounded by differences in caloric content. However, Pilichiewicz *et al.* addressed this limitation by utilising equicaloric

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Table 1. Summary of infusion and dietary challenge studies in adults with functional dyspepsia

Author (year), country	Population	N	Study design	Intervention	Comparator	Outcome measures	Key findings
Dietary-fat studies							
Houghton (1993), United Kingdom	Non-ulcer dyspepsia, HC	FD = 31, HC = 17	Crossover	Low-fat (3 g fat, 1.2 g CHO, 4.2 g protein) *v.* high-fat soup (30 g margarine added)	Within/ between group	Gastric emptying, postprandial symptoms (Likert scale)	Greater symptom response to high-fat meals in FD; normal gastric emptying
Feinle-Bisset (2003), Switzerland	FD	FD = 15	Crossover	High-fat (330 kcal, 18.5 g CHO, 8 g protein) *v.* low-fat yoghurt (143 kcal, 1.3 fat, 21.6 g CHO, 9.8 g protein)	Within group (meal-type)	Gastric barostat, FD symptoms (VAS), plasma CCK	Cognitive factors and high fat increased symptoms and CCK
Pilichiewicz (2008), Australia	FD (Rome III), HC	FD = 8, HC = 8	Crossover	High-CHO (500 kcal, 6 g fat, 96 g CHO, 14.4 g protein), high-fat (500 kcal, 31.6 g fat, 41.2 g CHO, 12.8 g protein) and low-nutrient meals (180 kcal, 2.4 g fat, 26.4 g CHO, 12.8 g protein)	Within/ between group	Antral area, FD symptoms (VAS), plasma CCK, PYY, ghrelin, blood glucose	High-fat meal induced more symptoms (nausea and pain) and CCK in FD
Lipid infusion studies							
Barbera (1995), United Kingdom	FD, HC	FD = 18, HC = 9	Non-RCT	Duodenal lipid (10% Intralipid) *v.* glucose (26.7% glucose) infusion	Between group (FD *v.* HC)	Gastric pressure, FD symptoms	Intraduodenal lipid, not glucose, increased gastric mechanosensitivity in FD
Barbera (1995), United Kingdom	FD, HC	FD = 10, HC = 10	Crossover	Duodenal lipid (10% Intralipid *v.* saline (0.9% saline) infusion	Between group (FD *v.* HC)	Gastric pressure, FD symptoms	FD more sensitive to lipid and gastric distension; lipid sensitised FD to distension
Feinle-Bisset (2001), Switzerland	FD, HC	FD = 12, HC = 6	Non-RCT	Duodenal lipid (10% *v.* 20% lipid)/saline infusion, or CCK-A blocker dexloxiglumide	Within/ between group	Gastric barostat, FD symptoms (VAS), plasma CCK	CCK-A blocker relieved lipid-induced symptoms; dose–response shown
Bharucha (2014), United States	FD (Rome III), HC	FD = 30, HC = 35	Crossover	Duodenal dextrose *v.* lipid infusion	Between group (FD *v.* HC)	Gastric emptying, small bowel transit time, FD symptoms (Likert scale), QoL, CCK, GLP1, peptide YY	Lipid/dextrose increased symptoms and hormones in FD *v.* HC
FODMAP studies							
Potter (2010), Australia	FD (Rome III)	FD = 11	RCT	Low FODMAP and gluten-free diet; gluten, fructan, or placebo rechallenges	Within group	FODMAP intake, FD symptoms (VAS, NDI)	Modest, non-significant improvement; specific trigger not found
Staudacher (2021), Australia	FD	FD = 59	Cohort	Low FODMAP *v.* SDA	Between group (low FODMAP *v.* SDA)	Dietary adherence, symptom scores (SAGIS)	Greater symptom reduction with low FODMAP; similar adherence
Goyal (2022), India	FD (Rome IV)	FD = 184	RCT	Low FODMAP *v.* SDA; FODMAP reintroduction	Between group (low FODMAP *v.* SDA)	FD symptoms (SF-NDI)	Both groups improved; low FODMAP better for bloating/PDS

CCK, cholecystokinin; CHO, carbohydrates; FD, functional dyspepsia; FODMAP, fermentable oligosaccharides, disaccharides, monosaccharides and polyols; HC, healthy controls; NDI, Nepean Dyspepsia Index; PYY, peptide YY; QoL, quality of life; RCT, randomised controlled trial; SAGIS, Structured Assessment of Gastrointestinal Symptoms; SDA, standard dietary advice; SF-NDI, Short Form Nepean Dyspepsia Index.

high-fat and high-carbohydrate meals (400 g yoghurt). Their findings demonstrated significantly greater FD symptoms, particularly nausea and pain, with the high-fat meal (32 g fat, 500 kcal) compared to the isocaloric high-carbohydrate meal (6 g fat, 500 kcal), confirming the involvement of dietary fats in symptom generation[21]. Table 1 provides a summary of the key infusion and dietary challenge studies investigating nutrient-symptom relationships in FD.

Limited research has examined the specific effects of different fat types on FD symptoms. Existing duodenal infusion studies have consistently utilised Intralipid – a soyabean oil formulation naturally high in polyunsaturated fats – while dietary intervention studies have typically increased fat content using cream, rich in saturated fats. Given all studies reported a positive association between fat consumption and dyspeptic symptoms such as nausea, pain, epigastric fulness, bloating and discomfort, various fat types

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may contribute to symptom generation in FD. Although current dietary intervention trials have some limitations, such as the use of semi-solid or liquid-based meals that may not reflect typical real-world eating patterns, they nonetheless provide valuable insights. Overall, the current evidence from both nutrient infusion and dietary intervention trials consistently demonstrates that dietary lipids are potent triggers of FD symptoms, likely through CCK-mediated pathways and heightened visceral sensitivity.

Fermentable carbohydrates

FODMAP (fermentable oligosaccharides, disaccharides, monosaccharides and polyols) are short-chain carbohydrates poorly absorbed in the small intestine. Upon reaching the colon, they are fermented by gut bacteria, leading to gas production and luminal distention. FODMAP are found in various foods including fruits, vegetables, grains, dairy products, legumes and sweeteners. The low FODMAP diet has substantial research demonstrating efficacy in reducing IBS symptoms and is widely utilised as first-line dietary therapy[74]. Although the impact of FODMAPs on gastric motility in FD has not been directly studied, evidence from IBS and healthy controls indicates that FODMAPs may affect upper GI function. One study found that acute intragastric fructan infusion increased postprandial intragastric pressure in both IBS patients and healthy individuals[75]. Symptoms developed rapidly – within 30 minutes – suggesting involvement of the proximal small intestine in FODMAP-induced symptom generation[75]. Given the frequent clinical overlap between IBS and FD and their shared pathophysiological features, the low FODMAP diet may also alleviate symptoms in at least a subgroup of FD patients. A systematic review identified that foods frequently associated as FD symptom triggers were also high in FODMAPs (e.g. wheat/grain products, some soft drinks, fruit/fruit juice, milk)[67]. Supporting this, a recent cross-sectional study by Cooke *et al.* found 55% of dyspepsia patients identified FODMAPs as symptom triggers[76].

In an early study, Wilder-Smith *et al.* screened 1372 patients with various DGBI, including 606 with FD, for both lactose and fructose malabsorption and intolerance[77]. Of those testing positive, 312 participants undertook a 4-week dietary intervention, beginning with saccharide and polyol restriction, followed by systematic reintroduction of defined doses of fructose, fructan, inulin and lactose to assess individual tolerance. Although fructose and lactose intolerance tests lack sensitivity[78], over 80% achieved substantial symptom relief following dietary restriction of saccharides and polyols[77]. Subsequent studies focusing specifically on FD have yielded encouraging results. Potter *et al.* conducted a pilot double-blind, randomised, placebo-controlled trial investigating effects of a four-week gluten-free and low FODMAP diet in nine FD patients. The intervention showed modest, non-significant symptom improvement. Four participants met the response threshold for rechallenge with muesli bars containing fructan, gluten or placebo, but no specific food trigger could be reliably identified[79]. The study's robust design was limited by its small sample size and combined dietary approach, preventing definitive conclusions about specific dietary components in FD. Staudacher *et al.* conducted an observational study comparing low FODMAP diet to standard dietary advice (SDA) in FD patients[80]. The SDA consisted of recommendations to reduce caffeine, alcohol, fat, fibre, or healthy eating advice based on the Australian Dietary Guidelines. Participants on the low FODMAP diet experienced greater reduction in epigastric scores for symptoms of postprandial pain, bloating and belching than those on SDA. Notably, 81% of participants had co-existing IBS, potentially confounding results. In a preliminary report, 25 FD patients followed a 6-week low FODMAP diet and subsequently underwent blinded reintroduction of FODMAP powders to determine individual triggers. Over half experienced improvements in fullness, satiation and upper abdominal bloating while on the low FODMAP diet, with mannitol and galacto-oligosaccharides most identified as symptom triggers[81,82]. The study additionally showed that the low FODMAP diet was associated with improved duodenal mucosal integrity. Goyal *et al.* further explored this in a prospective, single-blind trial comparing low FODMAP and SDA in 105 FD patients over 4 weeks, followed by guided FODMAP reintroduction for the low FODMAP group[83]. No significant difference in overall symptom improvement was observed between groups, with both achieving symptom relief and improved quality of life. However, patients with PDS subtype or bloating responded significantly better to the low FODMAP diet.

Several mechanisms may explain how FODMAPs elicit symptoms in FD. First, FODMAPs exert osmotic effects in the small intestine drawing water into the lumen as demonstrated by MRI studies in healthy controls and IBS patients[84–86]. This luminal distension may activate small intestinal mechanoreceptors in viscerally hypersensitive FD patients, generating symptoms well before colonic fermentation occurs. Second, rapid fermentation of FODMAPs produce gas and SCFA[87] that directly stimulate chemosensitive pathways in the proximal gut. Third, FODMAPs may influence gut microbiota composition and mucosal immune function, as suggested by preliminary data showing the low FODMAP diet improves duodenal mucosal integrity in FD patients[81]. Finally, colonic fermentation of FODMAPs may trigger gastro-colonic reflexes that alter proximal gastrointestinal motility and sensitivity[87]. Current evidence suggests that FODMAP restriction may be particularly beneficial for patients with PDS subtype FD, especially those experiencing bloating or with concurrent IBS symptoms. The identification of specific FODMAP triggers through systematic reintroduction may allow for personalised dietary modifications rather than long-term global FODMAP restriction. Beyond macronutrients, other dietary factors and bioactive components may also contribute to FD symptom generation.

Additional dietary triggers and modifiers

The gastrointestinal tract is exposed to various naturally occurring or synthetic bioactive substances. Food chemicals such as salicylates, amines, glutamates and lectins, found in many plant and animal-based foods, have also been suggested to influence gastrointestinal function[88]. While a diet low in food chemicals has been shown to benefit patients with IBS, their potential to induce symptoms has not been specifically studied in the FD population, nor has its therapeutic potential[89]. In addition to naturally occurring substances – compounds introduced during food processing to improve appearance, texture, shelf life, or nutritional value – represent another potential trigger. A large-scale web-based cohort study identified that increased proportion of ultra-processed foods was associated with higher risk of FD with concomitant IBS, though not FD in isolation[90]. This may suggest a potential association between food additives and FD occurrence, particularly in overlapping syndromes.

Certain commonly consumed beverages, such as coffee and alcohol, have also been investigated for their role in FD symptoms. Coffee consumption has been linked to increased gastric acid

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secretion and associated with induction of FD symptoms across several studies[67,91–94]. However, evidence remains inconclusive; one study specifically investigating coffee intake effects in FD patients found no significant association between consumption and symptom exacerbation[95]. The inconsistency in findings suggests individual variation in response to caffeine that warrants personalised assessment. Alcohol increases gastric acid secretion and influences gastric emptying rates[96], potentially contributing to FD symptoms. Some studies report a positive association between alcohol consumption and both development and worsening of FD symptoms. However, findings have been inconsistent, and no definitive relationship has been established[95,97,98]. As with coffee, the effects of alcohol may vary significantly between individuals, highlighting the importance of personalised dietary assessment.

Several bioactive plant compounds directly affect sensory receptors in the gastrointestinal tract[99]. Capsaicin from red pepper may modulate symptoms through TRPV1 receptor stimulation, showing potential to reduce pain, fullness and nausea in FD patients[100]. Menthol from Mentha species acts on TRPM8 channels, potentially reducing abdominal pain and improving quality of life[101]. Ginger compounds, particularly 6-gingerol and 6-shogaol, may alleviate FD symptoms through thermosensitive and mechanosensitive pathways[102]. However, evidence for these compounds comes mainly from small trials or supplement studies rather than typical dietary interventions.

Meal patterns and eating behaviours in FD

Meal patterns and eating behaviours potentially influence symptom generation in FD and may represent modifiable targets for intervention. However, evidence regarding whether FD patients actively alter their dietary habits remains inconsistent and sometimes contradictory. Early research suggested FD patients tend to consume smaller, more frequent meals or snacks instead of traditional three meals per day[103]. However, the study did not quantify or define what constituted a 'meal' or 'snack', limiting interpretability. Subsequent studies similarly found FD patients were more likely to skip meals compared to healthy controls yet also failed to define what constituted 'meals' and 'snacks'[93,104,105]. This lack of standardisation complicates interpretation of findings on meal patterns in FD. Only one study clearly distinguished between 'meals', 'light meals' and 'snacks', reporting that while FD patients consumed fewer meals, their intake of snacks and light meals was comparable to healthy controls[106]. Other studies found no significant differences in overall meal frequency between FD patients and healthy individuals[92,107]. Beyond meal frequency, the pace of eating has also been explored. Self-reported eating speed has been frequently noted in FD patients[92,93,103,108], yet quantitative studies measuring actual eating speed found no difference between FD patients and healthy individuals[92,108]. This discrepancy suggests potential perception bias in how FD patients view their eating behaviours. Recent data indicates widespread self-directed dietary management among FD patients. In a cross-sectional study by Cooke *et al.*, 88% of participants had tried special diets for symptom management, with low FODMAP being the most common (69%). This self-directed dietary modification resulted in significantly lower intake of fibre, calcium and FODMAPs compared to healthy controls, raising concerns about nutritional adequacy when dietary advice lacks professional oversight[76]. Such self-directed dietary changes may have consequences for overall nutritional status, as discussed below.

Nutritional consequences of dietary modifications

The established connection between food intake and symptom generation in FD creates risk for nutritional compromise through food avoidance. Several studies have identified high prevalence of unintentional weight loss in FD patients from tertiary centres, though these findings may not represent the general FD population[35,40]. Conversely, other studies have identified high prevalence of overweight and obesity in FD patients[92,93,103]. Dietary composition also appears altered in FD. Carvahlo *et al.* found FD patients had reduced fat intake but similar caloric intake compared to healthy controls due to increased proportion of carbohydrates[92]. In contrast, Pilicheiwicz *et al.* reported significantly lower intake of both fat and total calories in FD patients compared to healthy controls, potentially contributing to weight loss[106]. Despite these insights, the prevalence and risk of specific nutrient deficiencies in FD remain poorly understood. Given the potential impact of restrictive dietary interventions, careful nutritional monitoring is essential – especially when implementing elimination diets. These findings highlight the need for professional dietary guidance to manage symptoms effectively without compromising nutritional status. Further research is warranted to better define and address nutritional risk in this population.

Dietary recommendations and future directions

Research priorities

Current evidence provides a foundation for understanding diet–FD relationships, but significant knowledge gaps remain. Although dietetic management of FD commonly involves trial of a low-fat diet, evidence is limited regarding optimal restriction levels, differential effects of fat types and symptom-specific responses. Future studies should determine whether certain fatty acids are more symptom-provoking than others. For FODMAPs, research should focus on identifying specific FODMAP subgroups that trigger symptoms, assessing variations in FODMAP sensitivity across FD subtypes, and determining symptom patterns most associated with FODMAP intolerance. Additionally, further investigation is needed to determine whether dietary interventions address underlying pathophysiological mechanisms, such as low-grade inflammation and alterations in the gut microbiome, or primarily alleviate symptoms without addressing root causes. Methodological improvements in FD dietary research are essential. Development of validated FD-specific dietary assessment tools would enhance research quality and enable more reliable cross-study comparisons. Longer-term studies are needed to assess sustained efficacy, adherence challenges and nutritional adequacy of dietary modifications.

Clinical practice recommendations

Stepwise approach

Dietitians should adopt an individualised, stepwise approach beginning with comprehensive assessment to identify potential trigger foods. First-line interventions should focus on establishing regular meal patterns, moderating portion size, reducing caffeine and alcohol intake if identified as triggers, and adhering to healthy eating guidelines. These general modifications may be sufficient for many patients and minimise unnecessary dietary restrictions.

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Targeted dietary interventions

Current evidence suggests that a low FODMAP diet may benefit FD patients with PDS subtype, symptoms of bloating and/or co-occurring IBS, while low-fat diet may benefit across subtypes. A modified low FODMAP approach may be an adequate starting point to avoid overly restrictive diets in patients who may already be limiting several foods.

Risk management

Restrictive dietary interventions should be avoided in high-risk groups including individuals with complex nutritional needs, those at risk of eating disorders, and elderly or malnourished patients. Regular monitoring of nutritional status, weight and symptom response is essential, with dietary strategies adjusted accordingly.

Patient education

Dietitians should educate patients about the gut–brain connection, address food fears, provide practical strategies for managing meals in various settings and set realistic expectations about symptom improvement. Coordinated care between gastroenterologists, primary care physicians and allied-health professionals optimises outcomes for this challenging condition.

Conclusions

This review has examined the complex relationship between diet and FD, highlighting key mechanisms and potential dietary targets. Nutrient sensing, particularly lipid-mediated pathways involving CCK, appear central to symptom generation. Additional contributors such as low-grade duodenal inflammation, altered microbiota and impaired gastric accommodation present potential targets for dietary modulation. Fat consistently emerges as a major symptom trigger across controlled studies, supporting its restriction as an important component of dietary management. FODMAPs may be relevant for patients with PDS subtype, bloating or concomitant IBS, while meal patterns and eating behaviours warrant further investigation. Current evidence supports individualised dietary approaches tailored to FD subtype, symptom pattern and temporal relationship to meals. While data on long-term efficacy and optimal implementation are limited, the pathophysiological insights presented here provide a foundation for evidence-based nutritional care. Combining dietary strategies with pharmacological treatment may offer the most benefit. As understanding of FD pathophysiology deepens, dietary interventions will likely become increasingly targeted and effective. Integrating mechanistic insights – spanning nutrient sensing, immune activation and gut–brain signaling – with clinical observations offers promise for developing personalised nutrition strategies that meaningfully improve outcomes for those living with this challenging condition.

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