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Review Article

Dietary and nutraceutical interventions for functional dyspepsia: A narrative review

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ABSTRACT

Functional dyspepsia (FD) is a prevalent disorder of gut–brain interaction. Commonly, meals may exacerbate FD symptoms like postprandial fullness, early satiation, epigastric pain, and nausea. We narratively synthesize meta-analyses, randomized trials, and observational evidence to outline mechanism-based dietary and nutraceutical options. Evidence favors small, regular, lower-fat meals and adjusting texture/osmolarity to minimize gastric distension. In selected phenotypes—particularly postprandial distress with bloating—a brief, dietitian-supervised low-FODMAP trial with staged reintroduction can define personal thresholds. Among nutraceuticals, peppermint–caraway, ginger, STW-5, curcumin, and selected probiotics show benefit, with melatonin and barrier-forming agents promising in subsets, while safety remains product-specific.

1. Background and definitions

Functional dyspepsia (FD) is one of the most common gastrointestinal disorders encountered in general practice. It is defined by the presence of recurrent or persistent symptoms originating from the gastro-duodenal region, in the absence of any organic disease that could explain them [1]. According to the Rome IV criteria, and consistently supported by the most recent guidelines, FD can be classified into two distinct subtypes: epigastric pain syndrome (EPS) and postprandial distress syndrome (PDS). This subdivision is based primarily on two factors: the predominant symptom pattern and its association with meal intake [2,3]. For a diagnosis, symptoms must be present for at least one day per week in EPS and three days per week in PDS with a symptom duration exceeding six months and persistent for at least three months [3]. Patients with PDS typically report postprandial fullness and early satiation, often leading to reduced appetite, and triggered by food consumption. Conversely, individuals with EPS experience epigastric pain or burning, which is not necessarily meal related. In some cases, EPS symptoms may be relieved by eating, while in others they may also occur during fasting. Both subtypes may present with an acute or chronic course and can be accompanied by additional complaints such as nausea, bloating, or belching. Importantly, these categories are not mutually exclusive, and many patients exhibit features of both, resulting in an EPS–PDS overlap [1].

Analysis of data from the Rome Foundation Global Epidemiology Study estimates the worldwide prevalence of functional dyspepsia at 7.2 % [4]. Prevalence is significantly higher in women than in men and tends to decrease with age. Among the subtypes, PDS is the most common, accounting for 66.6 % of cases, compared with 15.3 % for EPS and 18.1 % for overlapping PDS–EPS [4].

Regarding comorbidities, overlapping Rome IV IBS was identified in 26.1 % of individuals meeting dyspeptic symptom criteria. Functional heartburn and chronic nausea or vomiting were present in 9 % and 7 % of these patients, respectively [5,6]. These overlap-

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ping disorders were more common in the EPS subgroup than in PDS, with the highest prevalence observed in patients presenting with both EPS and PDS: 60.7 % for IBS, 26.7 % for functional heartburn, and 17.6 % for nausea or vomiting [4].

Several studies have shown that a substantial proportion, around two-thirds, of patients with FD experience anxiety, depression, or psychological distress [7]. FD symptoms are linked to substantial healthcare utilization, with one-third of affected individuals reporting physician visits and frequent use of acid-suppressive, analgesic, and antinausea medications. This pattern was most pronounced among patients with overlapping PDS/EPS, who also showed the highest use of antidepressants, anxiolytics, and sleep aids [4].

Food is frequently reported by dyspeptic patients as a common trigger for symptoms. Recent Italian guidelines on FD recommend against the use of restrictive exclusion diets in the management of FD. However, general healthy lifestyle measures represent reasonable advice for FD patients [1].

Therapeutic management is challenging due to the partially known and heterogeneous pathophysiology. At present, the main therapy should be directed toward controlling the predominant symptoms, with recent guidelines recommending a short course of proton pump inhibitors (PPIs), regardless of the dyspepsia subtype, while in patients presenting with the PDS phenotype, prokinetic agents are considered appropriate as second line treatment. For EPS forms refractory to standard therapy, the use of tricyclic antidepressants may be considered [1]. Alongside established pharmacological strategies, there has been growing interest in nutraceuticals in recent years, which are progressively gaining relevance as complementary therapeutic options. The evidence supporting the efficacy of complementary and alternative medicine in the management of functional dyspepsia remains limited. Nevertheless, it is estimated that nearly half of patients with functional dyspepsia resort to complementary and alternative medicine approaches for symptom relief. This trend is not unexpected, considering the suboptimal efficacy of currently available pharmacological treatments [8].

We conducted a structured narrative review with a focus on clinically actionable evidence for dietary and nutraceutical interventions in FD. Searches were performed in MEDLINE/PubMed, Embase, Cochrane CENTRAL, Web of Science, and Scopus from January 2000 to October 2025, complemented by hand-searching reference lists. Search terms combined controlled vocabulary and keywords for “functional dyspepsia,” “postprandial distress syndrome,” “epigastric pain syndrome,” “diet,” “dietary pattern,” “low-FODMAP,” “Mediterranean,” “nutraceutical,” and specific agents (e.g., peppermint oil, ginger, curcumin, STW-5, probiotics, melatonin). We preferentially prioritized in the evidence synthesis meta-analyses, randomized controlled trials, and high-quality observational cohort studies, assessed by an adapted version from the Oxford Centre for Evidence-Based Medicine Levels of Evidence [9], and contemporary guidelines; mechanistic and physiological studies were included when they provided clinical interpretation.

2. Pathophysiology relevant to diet and nutraceuticals

FD is a disorder of gut–brain interaction with multifactorial pathophysiology (Fig. 1). Key contributors include gastric sensorimotor dysfunction (delayed emptying and impaired accommodation), visceral hypersensitivity, low-grade mucosal inflammation (especially duodenal), and gut–brain axis/microbiota alterations [10–13]. These mechanisms are not independent but dynamically interact within a bidirectional brain–gut network, influencing perception, motility, and immune signaling. Delayed emptying occurs in 25–35 % [14], while up to 40 % show impaired gastric accommodation [15], both linking mechanistically to postprandial fullness, early satiety, and nausea. Beyond these motor abnormalities, gastric hypersensitivity amplifies postprandial discomfort, even when emptying and accommodation appear normal, suggesting the central modulation plays a key role. Visceral hypersensitivity—an exaggerated response to physiological stimuli—emerges in roughly one-third of FD; duodenal nutrients (notably lipids) or acid perfusion provokes dyspeptic symptoms more readily than in controls [12,16,17]. High-fat meals can delay gastric emptying and augment cholecystokinin release, reinforcing fundic relaxation and hypersensitivity [12,18]. Capsaicin activates Transient Receptor Potential Vanilloid 1 and can acutely trigger symptoms, although longer exposure may induce desensitization [19,20]. Collectively, these findings reflect how normal postprandial physiological responses may become symptom-generating in sensitized individuals.

Duodenal immune activation and barrier dysfunction are increasingly recognized, especially in postprandial distress syndrome: eosinophils/mast cells and increased permeability correlate with symptom burden [15,21]. Mediators (histamine, proteases) may sensitize afferents and impair accommodation, connecting duodenal pathology to gastric symptoms. These data, supported by *Vanheel et al.* work, redefine FD as a disorder with subtle yet measurable mucosal pathology rather than a purely functional condition [21]. Post-infectious FD after gastroenteritis and cytokine changes (e.g., TNF-α linked to anxiety) emphasize immune–brain crosstalk [15,22,23]. Early data also suggest dietary antigen hypersensitivity and benefit from targeted exclusion diets (e.g., low-wheat/low-FODMAP) in selected patients [24,25]. This supports the emerging view of FD as a low-grade inflammatory and immune-mediated disorder triggered or perpetuated by environmental and dietary factors [26].

A dysregulated gut–brain axis amplifies symptom generation: anxiety/stress are common, with altered central processing of gastric signals on neuroimaging [7,27]. Emotional arousal and autonomic imbalance modulate visceral perception, explaining the clinical observation that stress exacerbates postprandial distress [28]. Microbiota involvement is supported by dysbiosis/SIBO signals, duodenal microbial shifts, and modest responses to probiotics/rifaximin in some studies [13,29–31]. Helicobacter pylori infection remains uniquely disease-modifying in a subset; eradication can resolve symptoms, although in a minority of patients, underscoring how mucosal inflammation can mimic FD [15,32].

These mechanisms map directly to diet/nutraceutical strategies. For motor/accommodation issues: small, frequent, low-fat meals can reduce distension and accelerate transit [33]; avoidance of very fatty/spicy/acidic foods, caffeine, alcohol, and carbonation is reasonable given mechanistic plausibility [34]. Individualized dietary counselling helps to identify patterns of symptom-triggering foods, an approach increasingly emphasized in modern disorders of gut-brain interaction management. Personalized trigger identification is key; short, structured trials of low-FODMAP or wheat-light diets can be considered in bloating/food-sensitive phenotypes [35,36]. For hypersensitivity/motility, peppermint oil (smooth-muscle calcium-channel effects) and ginger (prokinetic/antiemetic) have supportive signals; STW-5 targets motility, secretion, and sensitivity [37–39]. Poliprotect—a mucoadhesive protective complex—may relieve functional upper-GI symptoms including FD/gastroesophageal reflux disease (GERD) by barrier effects [40–44]. Duodenal eosinophilic activity provides a rationale for anti-inflammatory nutraceuticals (e.g., curcumin; DGL licorice) as adjuncts [45,46]. Probiotics may modulate barrier and signaling (strain-specific effects; evidence emerging) [47]. Melatonin—given gut/central receptors—can reduce visceral sensitivity and improve sleep, benefiting symptoms in some patients [48].

Overall, FD represents a spectrum disorder where dietary, microbial, immune, and neurogastroenterological factors converge; thus, integrated, mechanism-driven nutritional and nutraceutical

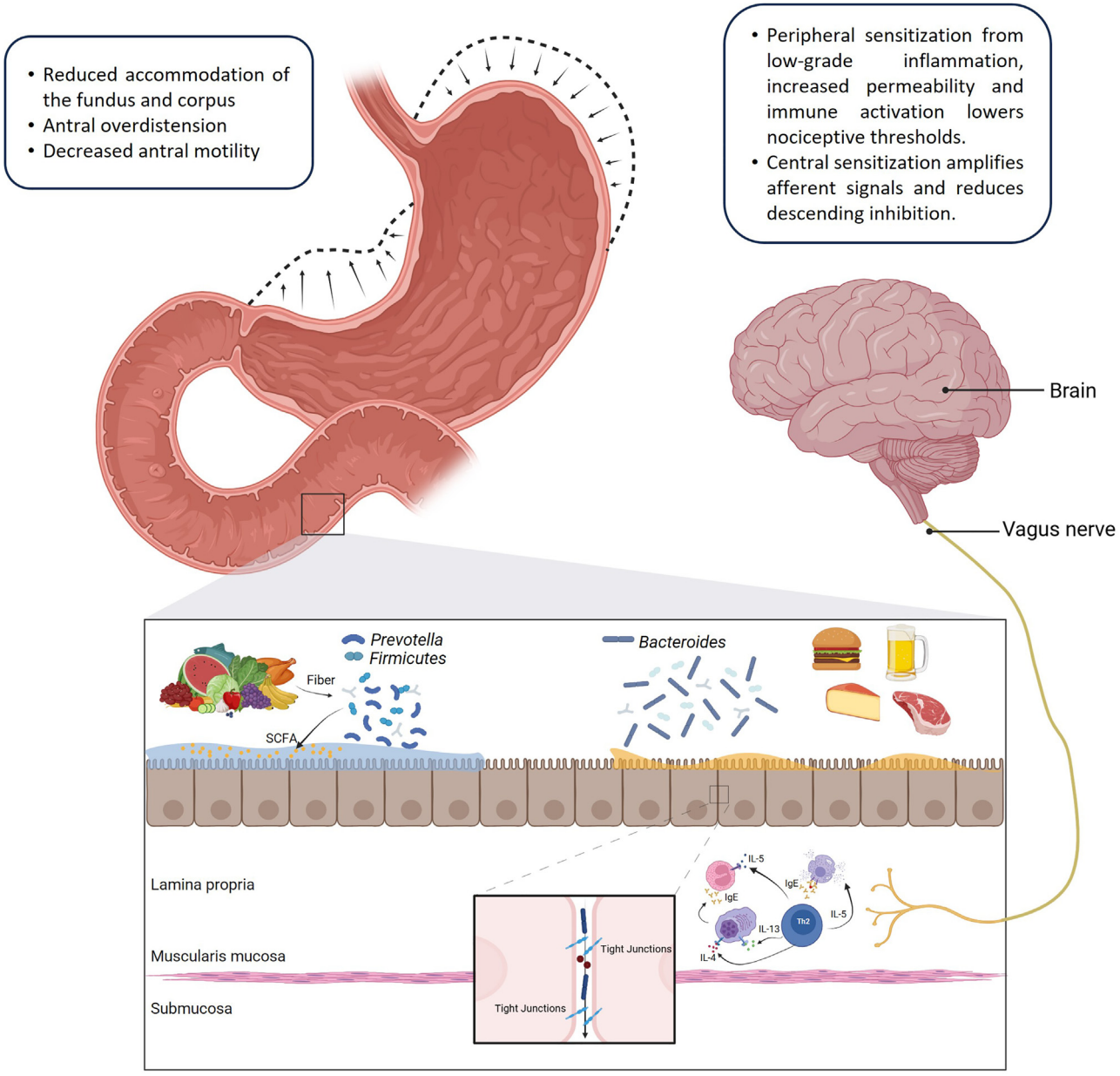

Fig. 1. Diet, duodenal barrier dysfunction and gut–brain axis in functional dyspepsia. Schematic overview of how different dietary patterns shape the duodenal environment, the mucosal barrier, and immune activation, with downstream effects on visceral sensitivity and symptom perception. The upper part of the figure shows the stomach and proximal duodenum as key sites of motor and sensory dysfunction, from which afferent signals travel to the central nervous system via vagal and spinal pathways. The lower panel zooms in on the duodenal mucosa. A diet rich in unprocessed foods and fibre is linked to a more favourable microbiota, an intact mucus layer, and preserved tight junctions. In contrast, a diet high in fat, sugar, and ultra-processed foods favours dysbiosis, bile- and nutrient-driven epithelial insults, disruption of the mucus layer, and increased epithelial permeability. When the barrier is compromised, luminal antigens and bacteria can penetrate the mucosa, leading to mild chronic inflammation and activation of immune cells and sensory nerve endings. These peripheral changes contribute to central sensitization in brain regions involved in pain modulation, thereby maintaining postprandial symptoms and visceral hypersensitivity. Figure created with BioRender.com.

approaches are increasingly seen as a rational complement to pharmacologic therapy [10,49,50].

3. Dietary intervention and patterns

A cornerstone of FD management is tailoring diet to minimize symptom triggers while maintaining nutritional adequacy [51,52]. Most patients benefit from small, regular meals. Indeed, smaller portions limit gastric distension, thereby reducing early satiety and postprandial discomfort [53]. Observational data associate consuming at least three meals per day (with optional small snacks) with fewer FD symptoms, whereas meal skipping—especially breakfast or lunch—correlates with higher odds of both postprandial distress and epigastric pain [54–56]. In a recent cross-sectional study in young Japanese adults, skipping breakfast and/or lunch was independently associated with higher FD prevalence, whereas skipping dinner or snacking was not; moreover, FD prevalence decreased with greater meal frequency, with an inverse, dose–response association [55]. Complementing this, a cross-sectional study found that extending the dinner-to-breakfast interval >10 h was associated with higher symptom burden, with a greater likelihood of indigestion, heartburn, and regurgitation, underscoring the risks of skipping meals or maintaining long inter-meal gaps [56]. Thus, counseling should emphasize portion control, steady meal timing, and avoidance of large late-evening meals, with individualized adjustments based on tolerance and symptom tracking.

Beyond size and timing, mindful eating is advisable. Many FD patients describe themselves as “fast eaters” or report eating under stress; rapid ingestion increases aerophagia and may blunt satiety signaling, exacerbating fullness and bloating [52,57,58]. In a cohort study conducted on 4763 adults, emerged that both an irregular meal pattern and a moderate-to-fast eating rate were independently associated with higher odds of chronic uninvestigated dyspepsia [59]. Moreover, the role of stress along the gut–brain axis is underscored by a 10-year follow-up, where baseline anxiety predicted new-onset functional dyspepsia with a 7.6-fold increased risk [60]. Considering this evidence, practical measures—slowing pace, thorough chewing, minimizing distractions, and fostering a low-stress mealtime environment—are conceptually aligned with FD pathophysiology, even though randomized trials specifically testing “mindful eating” in FD remain limited.

Patients should also be guided to identify and avoid their personal trigger foods. Evidence reported that up to 60–70 % of FD patients report that specific foods precipitate symptoms, often within minutes of ingestion. Eliminating such trigger foods from the diet has been shown to alleviate symptoms in many cases [61,62].

3.1. Meal composition and common triggers

Within meal composition, dietary fat emerges as the macronutrient most consistently linked to symptom exacerbation [63]. Duodenal exposure to lipid increases the release of cholecystokinin and related mediators, slowing gastric emptying and heightening sensations of fullness, nausea, and pain [64]. Patients commonly report that meals rich in fat leave them disproportionately uncomfortable; over time, they often notice that shifting toward naturally lighter preparations, with an emphasis on leaner protein choices and cooking methods that do not add substantial fat, lowers the symptomatic burden of any single meal without compromising pleasure or nutrition [12].

Portion volume and energy density exert parallel influences. Large meals create mechanical distension and prolong gastric processing [57,65]. Moreover, highly concentrated sweet drinks or very salty broths can elicit duodenal osmo-receptor responses, slowing gastric emptying and precipitating lingering fullness [66,67]; patients who notice this pattern often feel better when such beverages are diluted and taken slowly. For solids, textures that are tender or finely divided, alongside thorough chewing, reduce the work of gastric trituration and can make meals more “digestible” [68–70].

Many FD patients identify spicy foods and acidic foods as symptom triggers [34]. Capsaicin can acutely stimulate transient receptor potential vanilloid subtype 1 (TRPV1) afferents and provoke burning or pain, and population data have linked high spice consumption with greater FD prevalence in some settings [71,72]. A prospective study in FD showed that spicy-food intake was associated with symptom generation and that responses varied with TRPV1 genotypes; notably, capsaicin-rich foods were linked to sensations of postprandial fullness [73]. On the contrary, small trials suggest that chronic low-dose exposure may induce desensitization and eventually improve symptoms, but short-term tolerability limits widespread application [20].

Similarly, acidic foods such as citrus may irritate sensitive mucosa in some patients [74]; in a recent cross-sectional study, patients with overlapping upper-GI symptoms more often reported that citrus triggered their complaints than patients without such overlap [75]; similarly, in a observational study of patients with reflux, tomatoes and tomato products were frequently reported as triggers; however, these were self-reports, not associations confirmed by provocation testing [76]. In this context, coffee is both acidic and a source of caffeine [77]; while some people experience clear worsening even with small amounts, others tolerate a modest cup when taken with food. An alternative approach is temporary replacement with a non-caffeinated coffee substitute: in a small prospective study of adults with FD, substituting regular or decaf coffee with a grain-based, caffeine-free beverage for 4 weeks was associated with a significant reduction in dyspeptic symptom scores [78]. Nevertheless, a time-limited coffee reduction remains a practical first step to gauge individual sensitivity, after which careful reintroduction in small quantities can help patients identify their personal threshold [63,77,79]. Beyond coffee, alcohol — particularly in higher-strength forms or when consumed without food—can irritate the gastric mucosa and alter motility. This aligns with a recent population-based study, where alcohol consumption was significantly associated with functional dyspepsia, and—on multivariable analysis—emerged as an independent risk factor for FD within the broader spectrum of functional gastrointestinal diseases [78]. In parallel, carbonated beverages expand intragastric volume and promote belching, which in susceptible individuals amplifies fullness and pressure [80]. Analogously, behaviors that increase swallowed air—such as frequent gum chewing—can compound belching and bloating [81,82].

3.2. Dietary patterns

Shifting the focus from single foods or nutrients to dietary patterns, is more helpful for FD than prescriptive lists of “allowed” and “forbidden” items. A Mediterranean-style, minimally processed pattern offers a pragmatic dietary pattern that can be tailored to symptom thresholds while preserving diet quality. Observational data consistently suggest that higher adherence to Mediterranean Diet is associated with a lower probability of dyspeptic symptoms, probably through a reduced burden of saturated fats and ultra-processed foods and a higher intake of fibers, polyphenols, and mono- and omega-3 fatty acids, which may attenuate low-grade duodenal inflammation, reinforce epithelial barrier function, and modulate the gut microbiota [30,83]. Dietary fiber influences FD in dose- and type-dependent ways and is best framed within a Mediterranean-style pattern rather than as an isolated target. Tabibian et al. reported that higher fruit and vegetable intake—and, more broadly, greater adherence to Mediterranean-type eating that is naturally richer in fiber—associates with lower odds of dyspeptic symptoms such as early satiation and postprandial fullness [84]. At the same time, excess fiber or specific fiber types can acutely amplify fullness, bloating, or discomfort in patients with delayed emptying. Viscous soluble fibers (e.g., oats, psyllium, pectins) slow gastric emptying—useful metabolically but potentially problematic in FD if taken as large boluses—and the texture/structure of meals further modifies tolerance. Using magnetic resonance imaging, Marciani et al. showed that a whole-meal bread meal formed a thick, uniform mass in the stomach that prevented the normal sieving of liquids and small particles; as a result, it emptied more slowly than a rice meal, which separated more readily and emptied faster [85]. Likewise, an open label, crossover, randomized, controlled trial in healthy adults demonstrated that processing apples to puree or juice accelerated gastric emptying and reduced fullness/satiety compared with whole apples, underscoring the clinical relevance of particle size and viscosity to upper-GI comfort [86].

In light of interindividual heterogeneity, particularly in phenotypes characterized by fullness/bloating or IBS overlap, a low-FODMAP (fermentable oligo-, di- and monosaccharides and polyols) diet can be considered, notwithstanding recent guidelines indicating insufficient evidence to recommend specialized diets for FD [51]. FODMAPs are short-chain carbohydrates and sugar alcohols—principally excess fructose, lactose, fructans, galacto-oligosaccharides, and polyols (e.g., sorbitol, mannitol)—whose poor absorption and fermentability provide the mechanistic rationale

for dietary manipulation [36,87,88]. Owing to poor intestinal absorption and osmotic activity, they increase small-intestinal water content, then, once delivered to the colon, they undergo rapid microbial fermentation with gas production, promoting luminal distension [89,90]. The combined water influx and gas generation cause a rapid increase in intraluminal volume. In patients with heightened visceral sensitivity of the foregut, this distension is perceived as early satiety, postprandial fullness, pain, or nausea. By reducing the luminal FODMAP load, the low-FODMAP diet attenuates these water shifts and fermentative volume changes, thereby dampening the distension stimulus and improving symptoms [25,89].

Evidence supporting the low-FODMAP diet in FD is notable: in a real-world cohort study, patients that received low-FODMAP advice presented greater reductions in both epigastric and global gastrointestinal symptom scores than standard dietetic advice [91]; In a prospective, single-blind randomized trial, patients were allocated to 4 weeks of a low-FODMAP diet versus traditional dietary advice. Both groups improved on global dyspepsia measures over time; however, a subgroup analysis demonstrated a greater improvement in patients with the postprandial distress phenotype and bloating assigned to the low-FODMAP arm [25]. Nevertheless, restrictive approaches should be considered only after screening for disordered eating and, ideally, delivered with dietitian supervision; accordingly, the low-FODMAP diet should be delivered as a short, dietitian-supervised elimination (≈4–6 weeks) with appropriate substitutions to prevent micronutrient shortfalls (notably calcium, B-vitamins, and fiber), followed by systematic reintroduction of one FODMAP class at a time to define individual thresholds.

In summary, dietary management of FD should prioritize physiology-based, low-risk adjustments surrounded within a balanced, nutritionally complete pattern of eating. Regular small meals, unhurried, mindful ingestion, and modest fat reduction can mitigate impaired accommodation and postprandial hypersensitivity, while attention to osmolar load and food texture often lessens fullness and nausea. Because responses to trigger food are heterogeneous, brief, structured reduction trials with symptom monitoring are preferable to extensive exclusions. Fiber intake should be modulated rather than drastically reduced, favouring cooked sources and avoiding large raw/fibrous boluses in symptomatic people. Where foundational measures are insufficient, a short, dietitian-supervised low-FODMAP trial may clarify fermentable carbohydrate sensitivities, followed by a gradual reintroduction. A Mediterranean-style, minimally processed pattern provides a nutrient-dense, anti-inflammatory dietary pattern that can be flexibly adapted to individual tolerances. Overall, the aim is not rigid restriction but personalization, supported where possible by dietetic guidance, particularly in patients with severe or refractory FD [51], so that symptom control coexists with a nutritionally complete diet.

4. Nutraceuticals

Nutraceuticals are increasingly adopted in patients with FD seeking symptom relief beyond—or in combination with—conventional pharmacotherapy [92–94]. Their rationale stems from the condition's heterogeneous pathophysiology [13,53,95].

Evidence quality varies widely across agents, with some compounds (i.e., enteric-coated peppermint with caraway, multibotanical STW5, artichoke extract, selected probiotics, melatonin, and curcumin) supported by randomized controlled trials, whereas others (i.e., zinc-carnosine, deglycyrrhizinated licorice) are backed by smaller trials or mechanistic and extrapolative data (Table 1) [96–99]. Given product variability and strain-or extract-specific effects, clinicians should preferentially use formulations tested in trials, align the choice with the patient's predominant phenotype, and reassess after 4–8 weeks [51]. Safety profiles are generally favorable, but attention to interactions, rare idiosyncratic events, and special populations is valuable [94].

4.1. Peppermint oil

Peppermint oil, whose principal active constituent is L-menthol, exerts antispasmodic effects by blocking L-type calcium channels in gastrointestinal smooth muscle and by modulating sensory afferents including transient receptor potential channels [39]. A systematic review with meta-analysis of five RCTs, including around 580 patients found significant pain reduction and greater global improvement vs placebo with good tolerability [100]. Earlier RCTs showed non-inferiority to cisapride and superiority to placebo in reducing pain/fullness [101,102]. Common adverse effects are mild (e.g., peppermint eructation, occasional heartburn) [39]. Peppermint oil is sometimes associated with caraway oil which provides complementary antispasmodic and flatus-relieving actions. Used together, they plausibly address key FD mechanisms: impaired gastric accommodation, postprandial distention, and visceral hypersensitivity [100]. A common mixture is the association of 90 mg peppermint oil with 50 mg caraway oil, with a posology of 1 capsule twice daily for 4 weeks [103].

4.2. Ginger

Ginger's principal constituents—gingerols and their dehydration products, shogaols—exert prokinetic actions (via cholinergic and 5-HT_4 pathways) and antiemetic effects (through 5-HT_3 antagonism), thereby potentially improving PDS-type symptoms such as postprandial fullness and early satiation, as well as nausea [104,105]. Standard study doses range from 1 to 2 g/day of powdered root equivalents or standardized extracts 200–500 mg twice daily. Two randomized, double-blind trials found ginger superior to placebo for global symptom relief over 4 weeks, with one study showing particularly greater improvement in abdominal distension [106,107]. More recent randomized work—including a 12-week trial of a steamed-ginger extract (480 mg/day)—supports a signal for symptom reduction, though results vary across preparations [108]. Adverse events are usually mild gastrointestinal effects (bloating, heartburn) [109]. Overall, the evidence is promising but appears formulation-dependent, and larger confirmatory RCTs are warranted.

4.3. STW5: multi-botanical formulations

STW5 is a fixed combination of nine herbal extracts (i.e., *Iberis amara,* peppermint, caraway, chamomile, licorice, lemon balm, angelica, celandine, milk-thistle) with multimodal actions—fundic relaxation/accommodation, antispasmodic effects, and sensory modulation. The usual dose is 20 drops (∼1 mL) three times daily before meals. Evidence includes an older meta-analysis of double-blind RCTs showing benefit over placebo [110] and more recent randomized data for the updated formulation (STW5-II) [111,112]. A 2024 systematic review reports significant improvements in FD symptom scores at 4 and 8 weeks without excess serious adverse events versus placebo [98]. In several markets, the currently marketed formulation has been updated to a six-herb version (STW5-II) that omits angelica, greater celandine, and milk-thistle—introduced amid pharmacovigilance discussions and label warnings about rare idiosyncratic hepatotoxicity (particularly linked to celandine)—although clinical trials have not shown a hepatotoxic safety signal [113]. Safety appears favorable in trials; however, rare idiosyncratic hepatotoxicity has been reported post-marketing [114]. However, STW5 should be avoided in active liver disease and

Table 1
Adjunctive nutraceuticals and botanicals for functional dyspepsia: typical dosing, target phenotypes, and strength of evidence.

Agent/formulation	Typical dose & duration	Target phenotype / symptoms	Key evidence summary	Evidence
Peppermint oil ± Caraway	90 mg PO + 50 mg CO, 1 cap BID × 4 wk	PDS (fullness, pain), hypersensitivity	Multiple RCTs & meta-analyses show improvement vs placebo/cisapride; good tolerability.	+++
STW5 / STW5-II	20 drops (~1 mL) TID before meals × 4–8 wk	Global FD, gas tolerance, accommodation	Older RCT meta-analysis + newer RCTs; patient-data meta-analysis supports benefit.	+++
Ginger	200–500 mg BID (≈1–2 g root equiv) × 4–12 wk	PDS (fullness, early satiety), nausea	Several RCTs (variable preparations) show symptom reduction; prokinetic/antiemetic mechanisms.	++
Curcumin	500 mg QID (2 g/day) × 8 wk	EPS/PDS pain/fullness; low-grade inflammation	Large multicenter RCT: non-inferior to omeprazole at 4–8 w; smaller trials supportive.	++
Artichoke leaf extract	Std. dry extract 320 mg: 2 tabs TID (~1.9 g/day) × 6 wk	PDS after fatty meals	Double-blind RCT ($n \approx 247$) improved dyspepsia scores; combo with ginger also positive.	++
Deglycyrrhizinated licorice (DGL)	75 mg BID × 4 wk (std. extract e.g., 3.5 % glabridin)	Global FD symptoms	One RCT showed reduction in total dyspepsia scores vs placebo; good tolerability.	+/++
Probiotics	Examples: *B. coagulans* MY01 + *B. subtilis* MY02; *B. animalis lactis* BL-99; ~8 wk	PDS (early satiety, bloating), barrier/microbiota phenotype	Meta-analysis: modest benefit; RCTs show strain-dependent efficacy (adequate relief ↑).	++
Polyanionic polysaccharide complex	1 dose up to 5 × /day × 2 wk, then on-demand	EPS/heartburn-like epigastric pain/burning	Randomized non-inferiority vs omeprazole in endoscopy-negative heartburn/EPS; barrier mechanism.	+++
Zinc-carnosine	75 mg BID (≈17 mg elemental Zn/dose)	Barrier/"mucosal injury" phenotype	FD-specific RCTs limited; supportive data from gastritis/ulcer and mechanistic studies.	+
Melatonin	5 mg at bedtime × 8–12 wk	EPS pain; sleep disturbance/visceral sensitivity	Small RCTs suggest higher pain resolution vs placebo; neurosensory mechanism plausible.	+

Abbreviations — FD: functional dyspepsia; PDS: postprandial distress syndrome; EPS: epigastric pain syndrome; RCT: randomized controlled trial; O: peppermint oil; CO: caraway oil; PO: peppermint oil; BID/TID/QID: twice/three/four times daily; wk: weeks. Evidence grading (adapted from the Oxford Centre for Evidence-Based Medicine Levels of Evidence [9]): +++: consistent benefit supported by meta-analyses and/or multiple RCTs; ++: evidence from a single RCT with generally positive results; +: limited evidence (small trials) and/or mainly supportive mechanistic/physiological data.

advise discontinuation with prompt LFT assessment if jaundice or enzyme elevations occur [115].

4.4. Curcumin

Curcumin exhibits anti-inflammatory, antioxidant, and mast-cell–modulating effects relevant to low-grade duodeno-gastric inflammation and visceral hypersensitivity in FD. Typical RCT dosing has been 500 mg four times daily (2 g/day) versus omeprazole 20 mg/day and in a multicenter, double-blind RCT which included more than 200 patients with FD, curcumin was non-inferior to omeprazole at 4 and 8 weeks, with no added benefit from combination therapy [116]. Smaller trials generally support symptomatic improvement, while noting variability across products [117]. Adverse events are uncommon (mainly mild GI upset) and caution should be used in biliary diseases and with anticoagulants [116,117].

4.5. Artichoke leaf extract

Artichoke leaf extract contains choleretic constituents (e.g., cynarin) and may modestly enhance gastric motility—mechanisms aligned with PDS symptoms after fatty meals. In clinical practice, a common regimen is a standardized dry extract 320 mg, two tablets three times daily (~1.9 g/day) for 6 weeks. In a large double-blind RCT ($n = 247$), artichoke leaf extract significantly improved dyspeptic symptom scores and the Nepean Dyspepsia Index versus placebo [99]. A separate RCT found a ginger–artichoke combination superior to placebo, consistent with complementary prokinetic and choleretic effects [118]. Adverse events were low and comparable to placebo; avoid in biliary obstruction and use caution in patients with Asteraceae allergy [99,118].

4.6. Deglycyrrhizinated licorice

Deglycyrrhizinated licorice (DGL) is a licorice root extract with glycyrrhizin removed to avoid mineralocorticoid effects such as hypertension and hypokalemia. It preserves flavonoids and other constituents believed to enhance gastric mucus, improve mucosal blood flow, and modestly stabilize mast cells, yielding anti-inflammatory actions [119,120]. A randomized, placebo-controlled trial in 2012 evaluated a standardized extract (3.5 % glabridin) in 50 patients with functional dyspepsia for 30 days: DGL 75 mg twice daily significantly reduced total dyspepsia scores versus placebo, with improvements evident by day 15. Global assessments and Dyspepsia Index quality-of-life scores also improved, and no significant adverse effects or lab changes occurred, indicating good tolerability [121]. DGL appears safe, but patients should confirm products are deglycyrrhizinated and seek medical supervision when taking warfarin or digoxin [121].

4.7. Probiotics

The rationale for using probiotics in FD relies on several mechanisms, including epithelial barrier support, immune modulation dampening Th17 signaling, and effects on motility and visceral sensitivity [122]. A 2020 meta-analysis of prebiotics/probiotics showed a modest but significant benefit on global symptoms versus placebo, with marked strain-level heterogeneity [123]. One notable placebo-controlled trial by Wauters et al. (2022) tested a spore-forming probiotic (a combination of *Bacillus coagulans* MY01 and *Bacillus subtilis* MY02) in patients with postprandial distress syndrome. After 8 weeks, the probiotic group had significantly higher adequate relief rates than placebo, and symptom scores, like early satiety and bloating, improved [124]. Another recent trial from East Asia evaluated *Bifidobacterium animalis lactis* (strain BL-99) in patients with FD in the context of a 8-week study with four arms: placebo, rabeprazole, low-dose and high-dose probiotic. The high-dose probiotic achieved a 90 % clinical response rate vs ~58 % on placebo. Moreover, 78 % of patients on high-dose BL-99 became completely symptom-free, compared to 36 % of placebo [125]. To note, it is likely that not all probiotics seem to have targeted effects in FD, whereas others may be ineffective.

4.8. Polyanionic polysaccharide complex (Poliprotect)

Poliprotect combines a polysaccharide fraction (from Aloe vera, Malva sylvestris, and Althea officinalis), bicarbonate/carbonate minerals, and a flavonoid fraction (from Glycyrrhiza glabra and Matricaria recutita) to form a bioadhesive, pH-buffering layer that shields the foregut epithelium [42,126].

Preclinical work shows protection against ethanol/indomethacin injury, ∼2-hour mucoadhesion, and antioxidant activity [42,126]. In a multicenter, randomized, double-blind, double-dummy non-inferiority trial, 275 endoscopy-negative adults with heartburn and/or epigastric pain/burning received either omeprazole 20 mg daily or Poliprotect five times daily for two weeks, then on-demand for two more, with VAS change to day 13 as the primary endpoint and a prespecified non-inferiority margin of −11 points. Poliprotect met non-inferiority in both ITT and per-protocol analyses, and during on-demand use patients consumed about 2–3 tablets per day while requiring fewer rescue antacid sachets than the PPI arm, despite similar symptom trajectories. Microbiota profiling showed stability with Poliprotect, whereas omeprazole increased several oral-cavity taxa (e.g., Streptococcus and Veillonella). Safety was favorable in both groups (178 adverse events, none serious; four IP-related events in the PPI arm) [43]. In addition, Poliprotect is recommened also for GERD treatment [40,127].

Important caveats include the absence of pH-impedance phenotyping and the exclusion of patients with severe baseline symptoms. Overall, Poliprotect appears a reasonable option for endoscopy-negative heartburn/EPS when PPI minimization is desired, pending phenotype-guided trials [43,128].

4.9. Others with emerging evidence

4.9.1. Zinc-carnosine

For patients in whom barrier dysfunction is suspected, zinc–carnosine represents a pragmatic and generally well-tolerated adjunct aimed at supporting mucosal healing. By adhering to the gastric and duodenal lining, it provides localized coverage, promotes epithelial repair, and appears to attenuate low-grade inflammation [129,130]. In clinical practice, a commonly used regimen is 75 mg twice daily (≈ 17 mg elemental zinc per dose). Although FD-specific randomized trials remain limited, converging evidence from studies in peptic ulcer, gastritis, and other mucosal-injury contexts—together with a strong mechanistic rationale—supports consideration of zinc–carnosine in “barrier-phenotype” presentations. The safety profile is favorable, with only occasional nausea or soft stools reported; nonetheless, prolonged high total zinc intake should be avoided, and clinicians should be mindful of potential zinc–copper imbalance with extended use [131]. Overall, while current support is partly extrapolative, the biologic plausibility and clinical experience justify cautious, time-limited trials pending FD-focused RCTs.

4.9.2. Melatonin

Beyond its circadian effects, melatonin appears to exert antinociceptive, antioxidant, and possibly accommodation-enhancing actions in the upper GI tract; by improving sleep, it may also favorably modulate symptom perception [132]. The regimen most studied is 5 mg at bedtime for 8–12 weeks. In a randomized trial of H. pylori–negative adults meeting Rome II criteria for ‘ulcer-like’ functional dyspepsia, complete pain resolution at 12 weeks occurred in 56.6 % of participants receiving melatonin versus 3.3 % with placebo, and most of the remaining melatonin-treated patients reported partial improvement—findings that support a predominant neurosensory/analgesic mechanism relevant to EPS [132,133]. Tolerability is excellent with the main adverse effect is morning sleepiness, which can often be mitigated by modest dose or timing adjustments.

5. Gaps and future directions

Despite growing interest in dietary and nutraceutical therapies for FD, significant knowledge gaps persist, pointing to future research directions. Notably, patients often implicate food triggers, yet current guidelines remain cautious, and a 2025 consensus recommends against restrictive diets in FD due to insufficient evidence [1]. This highlights the need for rigorous studies to validate dietary interventions.

An important challenge is the heterogeneity of FD with one-size-fits-all approaches likely miss subgroups of patients who would respond to specific interventions. Better phenotyping of responders is needed: FD’s varied pathophysiology suggests that identifying further patient subtypes could enable “precision” diet or nutraceutical therapy [63].

Another promising field is the gut microbiome. Dysbiosis and microbial shifts have been linked to FD, and microbiome-modulating strategies (probiotics, fermented foods, prebiotics/fibers) are being explored. Small studies of probiotics and even non-absorbable antibiotics like rifaximin report modest symptom improvements, reinforcing a microbiome role [13]. For instance, a randomized trial of rifaximin showed significantly higher global symptom relief vs placebo (78 % vs 52 % at 8 weeks), though such findings need confirmation and clarification of which patients benefit most [31].

A conspicuous gap is the lack of head-to-head comparative trials among different diets and nutraceuticals. Most available studies test a single diet or supplement against placebo, so it remains unclear whether a low-FODMAP diet is superior to a Mediterranean diet, or how various nutraceuticals up against each other in efficacy [134]. Comparative effectiveness studies are needed to prioritize the best approaches. Finally, concerns about long-term safety and product standardization must be addressed.

Many nutraceutical products lack consistent quality control, leading to variability in active components. Reported adverse events – such as rare hepatotoxicity with certain herbal blends – underscore the importance of safety monitoring. Future trials should incorporate longer follow-up to assess safety and use standardized, well-characterized nutraceutical formulations.

In summary, advancing the field will require large-scale, well-phenotyped trials that integrate these considerations. Emerging research initiatives are beginning to fill these gaps, but more evidence is needed to inform clinical guidelines and optimize personalized dietary/nutraceutical therapies for FD.

6. Conclusions and take-home points

The pathophysiology of FD is multifactorial, involving interactions among dietary, microbial, and mucosal immune mechanisms, together with bidirectional signaling between the gut and the brain. Mechanism-based nutritional and nutraceutical interventions are increasingly recognized as valuable adjuncts to standard pharmacologic treatment.

Dietary management should aim to mitigate symptom triggers while ensuring nutritional adequacy. Beyond meal size and timing, mindful eating and individualized identification of trigger foods are essential. Emphasizing overall dietary patterns—particularly a Mediterranean-style, minimally processed diet—is more effective than restrictive food lists, allowing personalization according to symptom thresholds while maintaining diet quality. The effects of dietary fiber on FD are dose- and type-dependent and are best considered within a balanced dietary context. Before implementing restrictive dietary strategies—including a low-FODMAP approach in

selected cases given the frequent overlap between disorders of the gut-brain interaction and disordered eating— screening for these behaviors is advisable, and dietitian-led counselling can help ensure safe, individualized, and nutritionally adequate interventions.

Nutraceuticals and botanicals can provide additional symptom relief with generally favourable safety profiles, although potential interactions, rare adverse events, and population-specific factors require attention.

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