



Narrative Review

Rapidly fermentable prebiotic substrates: Benefits, FODMAP effects and host tolerance

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SUMMARY

Rapidly fermentable prebiotic substrates occupy a contradictory position in human nutrition. In microbiome and functional-food research, low molecular weight non-digestible carbohydrates such as inulin, fructo-oligosaccharides, and galacto-oligosaccharides are added to foods and supplements to promote bifidogenic, metabolic and gastrointestinal benefits. In parallel, clinical nutrition often restricts the same substrates as FODMAPs to reduce symptoms in irritable bowel syndrome and related disorders of gut–brain interaction, while food-science research seeks to reduce raffinose-family oligosaccharides because of their flatulence-producing and antinutritional effects.

This review examines these contradictory framings and asks whether the same rapidly fermentable substrates should be understood as beneficial prebiotics, symptom-provoking FODMAPs, or tolerance-limiting food components. It argues that the physiological effects of these substrates are not fixed properties of the compounds alone but also arise from interactions between intrinsic fermentability and host context. Rapidly fermentable substrates may support beneficial microbial and metabolic responses in tolerant individuals, but may contribute to gas production, osmotic effects, luminal distension, symptom generation or even immune activation in susceptible contexts.

Future research should move beyond defining prebiotic efficacy primarily by enrichment of selected bacterial taxa. Instead, each substrate should be tested through dose–response studies that establish both beneficial and tolerance thresholds across healthy individuals and clinically relevant phenotypes. Future studies should also examine prebiotics within a host-ecology framework, testing whether benefit depends less on maximising microbial stimulation and more on maintaining a controlled, spatially contained and clinically tolerable microbiome.

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1. Introduction

The prebiotic concept has become a major framework for linking dietary substrates to gut microbiota and host health. Since its original formulation, it has shifted attention from dietary fibre as a largely bulking material towards the capacity of non-digestible substrates to modify microbial composition and activity in ways that may benefit the host [1,2]. Recognised prebiotic substrates, particularly inulin-type fructans, fructo-oligosaccharides and galacto-oligosaccharides, have been widely studied for their bifidogenic and lactogenic effects and for their

potential influence on short-chain fatty acid (SCFA) production, bowel function, immune regulation and metabolic health [3–5]. This evidence base explains their growing use in functional foods, supplements and clinical nutrition products [1,6]. Simultaneously, these prebiotic substrates occupy a more complicated position in human nutrition than the term “prebiotic” alone suggests. In clinical nutrition, the same substrates are frequently restricted as FODMAPs to reduce gastrointestinal symptoms in irritable bowel syndrome and related disorders of gut–brain interaction [7–9]. In food-science and legume-processing research, raffinose-family oligosaccharides are commonly reduced or removed due to their flatulence-producing and antinutritional effects [10–14].

Taken together, these parallel framings raise an important conceptual question regarding the nutritional interpretation of rapidly fermentable substrates. The same, or closely related,

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compounds may be promoted as beneficial prebiotics, restricted as symptom-provoking FODMAPs, or reduced as tolerance-limiting food components (Fig. 1). Therefore, this review examines these contrasting interpretations by considering substrate structure, fermentation behaviour, dose and host context, and by comparing rapidly fermentable low molecular weight prebiotic substrates with structurally complex high molecular weight dietary fibres in relation to fermentation kinetics, physical function and host tolerance.

2. Search strategy and evidence selection

This review was designed as a structured narrative review and conceptual synthesis rather than a systematic review. Literature searches were conducted using PubMed/MEDLINE, Web of Science and Google Scholar. Searches were organised around four evidence streams as follows: 1) prebiotic addition and recognised benefits, 2) FODMAP restriction and clinical tolerance, 3) food-processing removal of raffinose-family oligosaccharides as flatulence-producing or antinutritional factors, and 4) human milk oligosaccharides as a biologically distinct comparator. Search terms included combinations of prebiotics, inulin, fructo-oligosaccharides, galacto-oligosaccharides, raffinose-family oligosaccharides, FODMAP, irritable bowel syndrome, disorders of gut–brain interaction, flatulence factors, antinutritional factors,

fermentation kinetics, gas production, short-chain fatty acids, dietary fibre, human milk oligosaccharides and host–microbiome interactions. References were selected according to their relevance to the four evidence streams described above. No formal risk-of-bias assessment, PRISMA flow diagram or meta-analysis was undertaken.

3. Prebiotic addition: recognised benefits of rapidly fermentable substrates

The addition of rapidly fermentable substrates to foods, supplements and clinical nutrition products has expanded over the last decades due to their prebiotic properties [1,5]. Examples include inulin-type fructans, fructo-oligosaccharides, galacto-oligosaccharides and related substrates which are non-digestible by host enzymes and can be utilised by selected gut microorganisms. The most consistent reported effect is the enrichment of bifidobacteria and, in some contexts, lactobacilli, which has provided an important empirical basis for their classification as recognised prebiotics [15,16]. This microbial responsiveness is partly explained by the intrinsic properties of many low molecular weight prebiotic substrates. Their solubility, resistance to host digestion and microbial accessibility allow them to reach the colon and be fermented rapidly. These properties can support bifidogenic and metabolic responses, but they also mean that prebiotic

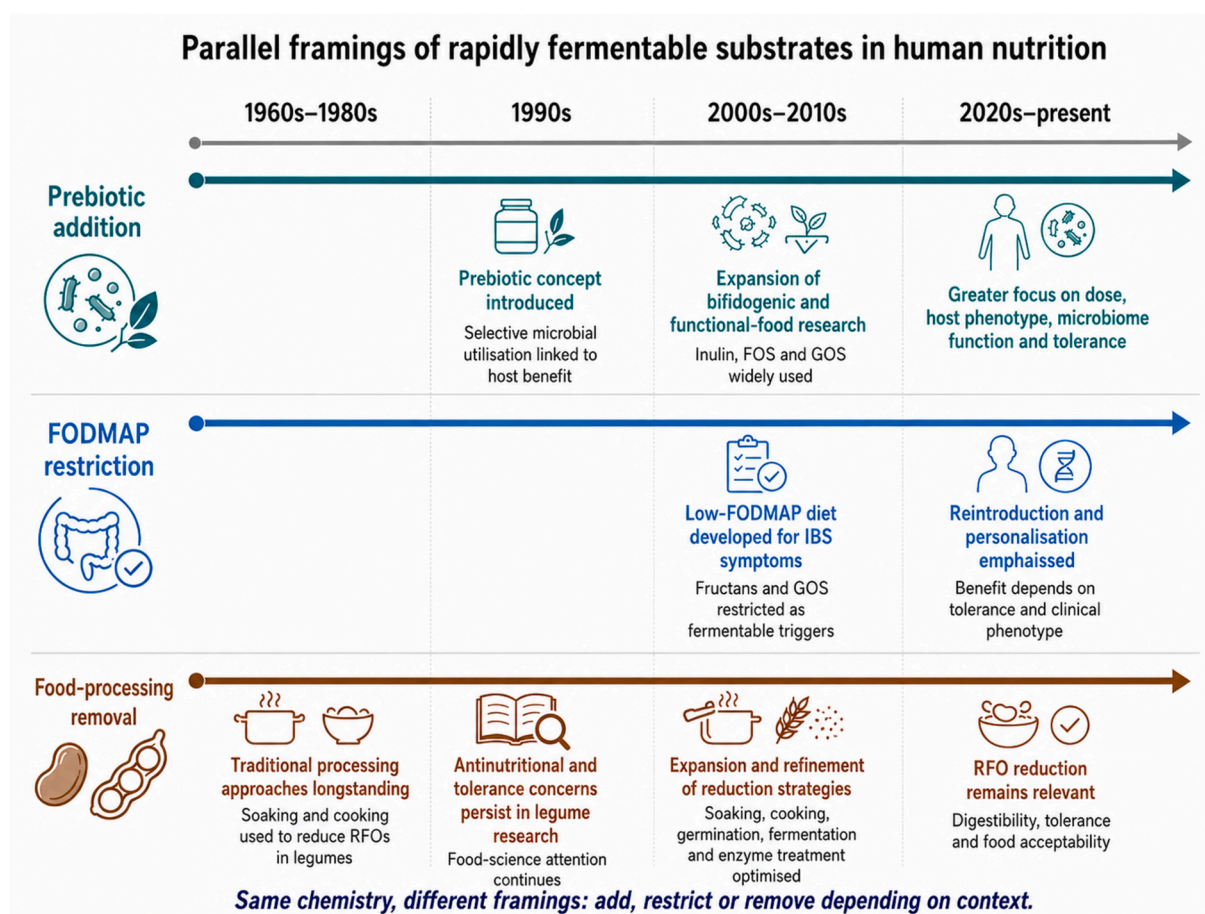


Fig. 1. Parallel framings of rapidly fermentable substrates in human nutrition.

The same or closely related substrates, including inulin-type fructans, fructo-oligosaccharides, galacto-oligosaccharides and raffinose-family oligosaccharides, have been interpreted differently across research fields. They may be added as prebiotics to stimulate selected microbial functions, restricted as FODMAPs to improve gastrointestinal tolerance, or reduced in food processing as flatulence-producing or antinutritional factors. The figure illustrates that these framings have coexisted in parallel rather than replacing one another despite different starting time points.

activity is closely linked to fermentation load and host tolerance [17–20]. Additionally, bifidogenic and lactogenic responses are important evidence of microbial responsiveness, but they are not sufficient on their own to establish host benefit as stipulated by the prebiotics definition [1]. A more complete evaluation of rapidly fermentable prebiotic substrates should consider not only changes in selected taxa, but also functional and metabolic outputs, cross-feeding, fermentation load, host–microbiome signalling and clinical tolerance [20–23]. This broader interpretation is consistent with recent microbiome research, which increasingly distinguishes taxonomic markers from functional, metabolic and host–interaction outcomes.

Thus, the rationale for the use of prebiotics should extend beyond taxonomic enrichment, as fermentation may alter short-chain fatty acid production, luminal pH, microbial metabolic activity and host–microbiome signalling. The prebiotic literature has therefore explored these substrates in relation to bowel function, immune regulation, allergy prevention, inflammatory conditions, metabolic health and gut–brain axis outcomes [1,5]. However, the strength of evidence varies considerably by substrate, dose, population and endpoint. Some reviews and intervention studies support potential benefit in defined contexts, whereas others highlight low certainty of evidence, methodological heterogeneity, limited clinical guidance and uncertainty regarding optimal dose, timing and target population [5,15,24–26].

Rapidly fermentable substrates should therefore not be treated as equivalent. Inulin, FOS, GOS and raffinose-family oligosaccharides differ in molecular size, degree of polymerisation, linkage type, solubility, microbial accessibility and fermentation kinetics (Figs. 2 and 3). These differences influence the types of organism that can utilise them, location, speed of utilisation, concentration of metabolites, and the host tolerance [4]. In conclusion, the benefit-oriented literature stream supports the principle that rapidly fermentable substrates can beneficially modulate the gut microbiota, but it also shows that such effects are substrate-specific and context-dependent rather than uniform across all prebiotics, hosts or clinical settings.

4. FODMAP restriction: when the same substrates become symptom triggers

In clinical nutrition, rapidly fermentable substrates are often viewed through a different perspective. The low-FODMAP diet restricts poorly absorbed and non-digested short-chain carbohydrates, including fructans, galacto-oligosaccharides and other fermentable substrates, to reduce luminal water, colonic fermentation and gastrointestinal symptoms [7,8,27,28]. This approach is most established in irritable bowel syndrome and related disorders of gut–brain interaction, where bloating, abdominal pain, flatulence, urgency and altered bowel habit are common clinical targets [7,9]. The clinical relevance of this restriction model is increasingly recognised, although the evidence also shows that its effects are not uniform [29–31]. Systematic reviews, trials and clinical practice studies generally report symptom improvement in the majority of patients with IBS, including reductions in abdominal pain, bloating and global symptom severity, with responses varying by IBS subtype, baseline microbiota, symptom profile, dietary implementation and comparator diet [28,31–33].

Evidence of the low-FODMAP benefits in paediatric DGBI, endometriosis-associated gastrointestinal symptoms and exercise-associated gastrointestinal symptoms is promising but more limited and context-specific [34–36]. Overall, the low-FODMAP related literature shows that the same fermentable properties that support prebiotic activity may become symptom-relevant in susceptible hosts. Rapid fermentation, gas

production, osmotic effects and luminal distension may be tolerated in healthy individuals, but may provoke symptoms in those with visceral hypersensitivity, altered transit, impaired barrier function or inflammatory activity [4,9,37–39]. Although the low-FODMAP clinical model is based on restriction, reintroduction and personalisation rather than permanent exclusion, it highlights a conceptual tension with the prebiotic-addition literature: substrates promoted for microbial benefit may need to be reduced, titrated or individualised when host tolerance is limited.

5. Food-processing removal: oligosaccharides as flatulence and antinutritional factors

A third framing comes from food-science and legume-processing research, where raffinose-family oligosaccharides are often treated not as desirable prebiotic substrates, but as tolerance-limiting compounds. Legumes are nutritionally valuable foods, but their wider use is commonly constrained by antinutritional factors, reduced digestibility and gastrointestinal symptoms, particularly flatulence. Within this literature, raffinose, stachyose and verbascose are frequently described as flatulence-causing oligosaccharides since humans lack small-intestinal α -galactosidase activity, allowing these substrates to reach the colon for microbial fermentation [13,14,40,41]. The practical relevance of this framing of oligosaccharides as flatulence factors depends on how much gas is considered excessive or poorly tolerated. In healthy adults, flatus frequency has been reported at approximately 10 times/day, with an upper normal limit of about 20 times/day. In a small study of healthy volunteers consuming their normal diet plus 200 g baked beans, 24-h flatus volume ranged from 476 to 1491 mL, with a median of 705 mL; a fibre-free diet reduced total flatus volume to a median of 214 mL/24 h and practically eliminated hydrogen production [42,43]. However, discomfort is not determined by gas volume alone as experimental flatulogenic diets can increase gas evacuation and mild discomfort even in healthy subjects, whereas patients with disorders of gut–brain interaction may experience substantial symptoms at similar gas volumes because of altered gas handling, visceral hypersensitivity and reduced tolerance to distension [39,44].

Concerns about flatulence, digestibility and product acceptability have led to extensive research on processing strategies designed to reduce raffinose-family oligosaccharide content. Traditional and household-level methods such as soaking, cooking, pressure cooking, autoclaving, germination, sprouting, malting, mashing and fermentation can lower raffinose-family oligosaccharides to varying degrees, although their effectiveness depends on the legume type, processing conditions, duration and whether soaking or cooking water is discarded [11,12,40,45,46]. More targeted modern approaches include the use of selected starter cultures, α -galactosidase-producing microorganisms and enzyme preparations to hydrolyse α -galactosidic linkages in raffinose, stachyose and verbascose [47–49]. Industrial membrane-based approaches, including ultrafiltration or diafiltration of aqueous soy or legume extracts, have also been investigated for removing low molecular weight oligosaccharides, although these are more relevant to processed ingredients such as soymilk or protein extracts than to ordinary whole-legume preparation [50,51].

Thus, the food-processing literature provides a third framing of rapidly fermentable oligosaccharides. In this context, raffinose-family oligosaccharides are not primarily valued for selective microbial stimulation, but are treated as tolerance-limiting compounds that may reduce digestibility, increase gas production and affect product acceptability. This contrast with the framing of oligosaccharides as prebiotic substrates is therefore important:

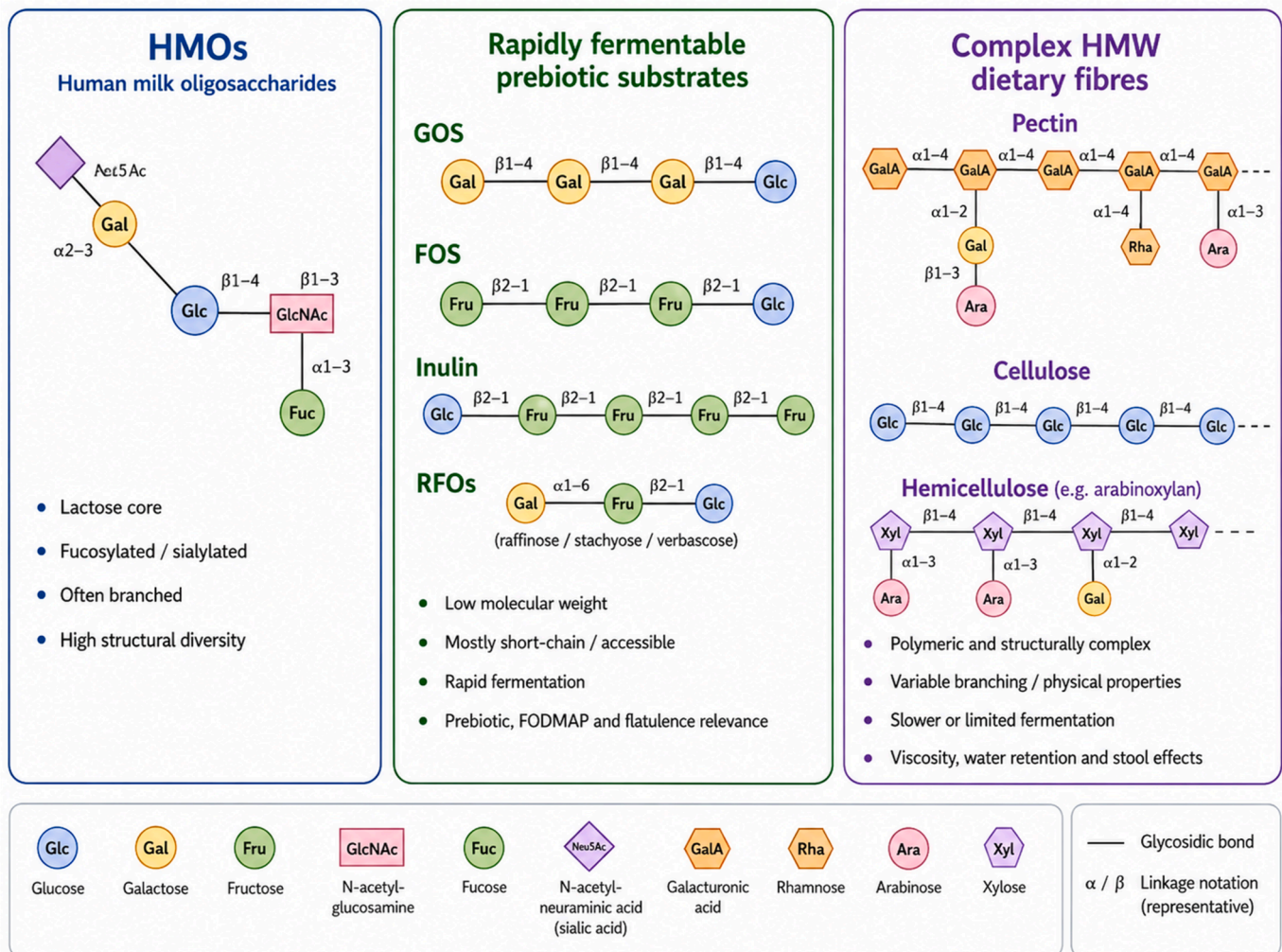


Fig. 2. Structural divergence between HMOs, rapidly fermentable prebiotic substrates and complex high molecular weight dietary fibres.

The figure contrasts representative structural motifs of human milk oligosaccharides, commonly used rapidly fermentable prebiotic substrates, and complex dietary fibres. It illustrates differences in structural diversity, molecular size, branching, microbial accessibility and physical properties relevant to fermentation kinetics, FODMAP effects, flatulence and host tolerance. Structures are schematic and not intended to represent exact molecular conformations.

substrates added in one field to stimulate microbial activity are reduced in another to limit gas formation and improve tolerance. This reinforces the need to evaluate rapidly fermentable substrates according to substrate type, dose, fermentation behaviour and host tolerance, rather than assuming that microbial accessibility is always beneficial.

6. Human milk oligosaccharides as a special case

Human milk oligosaccharides provide an important reference point for understanding the scientific rationale for using low molecular weight non-digestible carbohydrates within the prebiotic field. HMOs are abundant, structurally diverse glycans in human milk and contribute to early-life gut microbial ecology, immune development, intestinal barrier function and protection against pathogens [52–55]. Their effects are closely linked to their glycobiology, including fucosylation, sialylation, branching, linkage position and variation in maternal secretor status (Fig. 2). These features allow HMOs to act not only as microbial substrates, but also as pathogen decoys, immune-modulatory glycans and

components of a specific mother–infant developmental system. The practical difficulty of reproducing the full structural diversity of HMOs has contributed to the use of simpler substrates, including GOS, FOS and inulin-type compounds, in infant formula and functional foods. These substitutes may increase bifidobacteria, influence stool characteristics and have clinical or nutritional value in defined contexts [56,57].

Studies comparing individual HMOs with GOS and inulin show that fermentation is strongly structure-dependent. Kong et al. (2021) showed that 3-fucosyllactose was utilised slowly, whereas lacto-N-triose II was fermented more rapidly but produced a different microbial and metabolic profile. GOS and low-DP inulin fractions were also rapidly utilised. Fermentation digesta differed in their effects on epithelial adhesion of *Lactobacillus plantarum*, indicating that structural differences can translate into different functional consequences [58]. HMOs, particularly fucosylated and sialylated structures, have also been associated with early neurodevelopmental outcomes, including cognitive, language and motor domains [59–61]. By contrast, the evidence for plant-derived oligosaccharides such as FOS, GOS and inulin relates mainly to

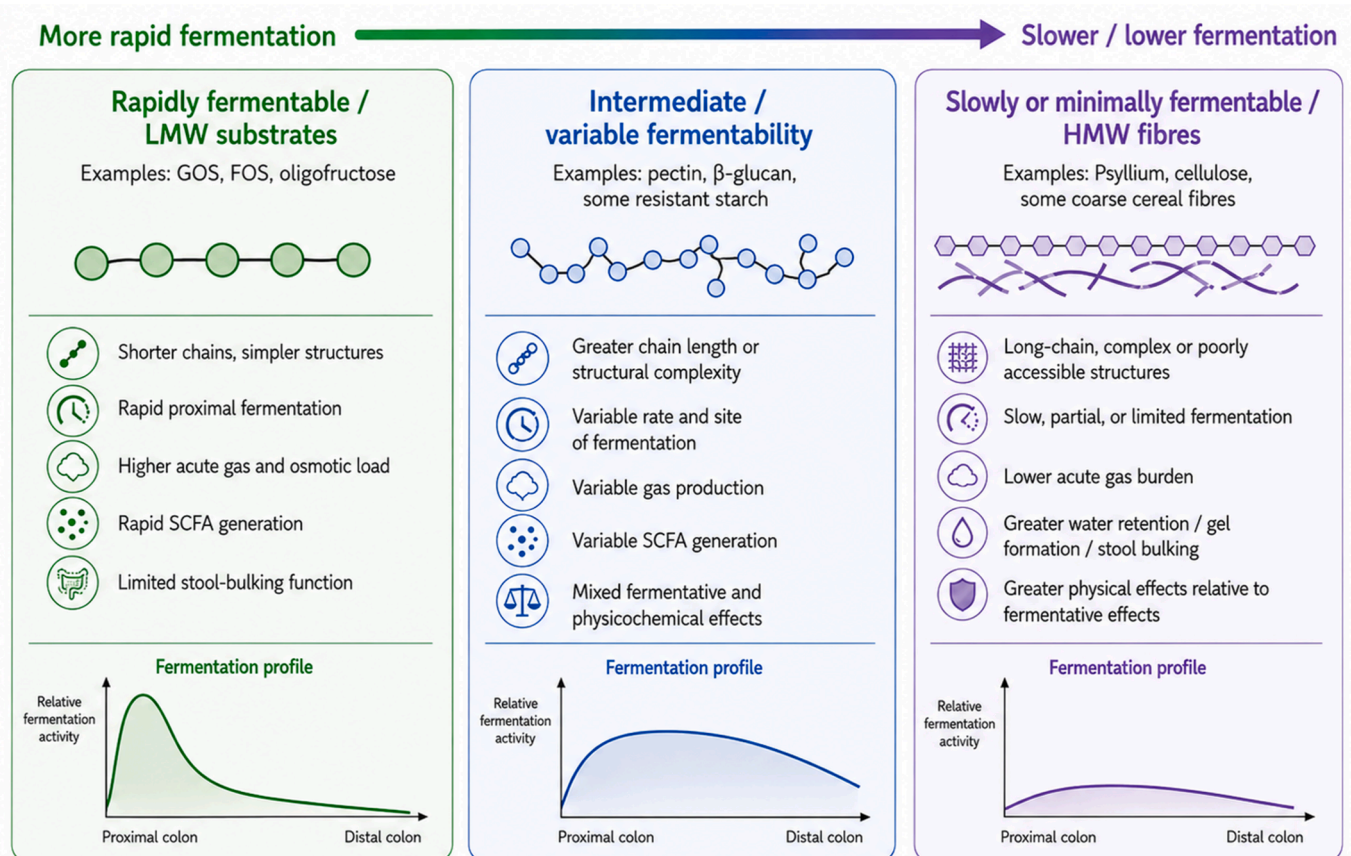


Fig. 3. Comparative fermentability of rapidly fermentable low molecular weight substrates and complex high molecular weight dietary fibres.

The figure illustrates a schematic fermentability continuum across selected non-digestible carbohydrates. Differences in molecular size, structural complexity and physicochemical properties may influence fermentation rate, gas production, short-chain fatty acid generation, and physical effects relevant to host tolerance and bowel function. The profiles shown are schematic and intended to illustrate general functional patterns rather than exact in vivo fermentation kinetics.

bifidogenic and gastrointestinal effects, rather than comparable HMO-like neurodevelopmental functions.

Overall, HMOs demonstrate that glycan effects are highly structure- and context-dependent. Accordingly, GOS, FOS and inulin should not be used as simplified models of HMO function, given their lower structural diversity and functional divergence from HMOs. Their potential benefits should instead be evaluated according to substrate structure, dose, fermentation behaviour, target population and host context.

7. Selectivity, host control, and ecological regulation

A key limitation of the prebiotic concept concerns the interpretation of microbial selectivity. As outlined above, recognised prebiotics, particularly inulin-type fructans and galactooligosaccharides, have been shown to reproducibly increase bifidobacteria and, in some contexts, lactobacilli. These findings support the concept of preferential microbial utilisation, but selectivity should be understood as relative and quantitative rather than absolute or exclusive. The carbohydrate-active enzymes, transporters and metabolic pathways required to utilise non-digestible carbohydrates are distributed across multiple gut microbial genera, and the products of primary fermentation can be further used through cross-feeding [21–23]. Therefore, the effect of a prebiotic substrate is not confined to the initially enriched taxa, but may extend across a wider microbial and metabolic network.

Bifidogenic enrichment is important evidence of microbial responsiveness, but it should not be treated as sufficient evidence of host benefit, ecological stability or clinical tolerance.

This point also aligns with the broader difficulty of defining a universal taxonomic core microbiome. Although early interpretations of microbiome health often implied that particular microbial groups could be classified as consistently beneficial across hosts, human gut microbiomes vary substantially between individuals, populations and environments, and no single taxonomic profile reliably defines health. Current concepts therefore place greater emphasis on functional capacity, ecological redundancy, host-microbe interactions and context-dependent microbial behaviour than on the consistent presence of fixed beneficial taxa [62–64]. Microorganisms should therefore be understood as conditionally beneficial members of a regulated ecosystem, whose effects depend on substrate availability, microbial competition, host physiology, immune tone, barrier integrity and transit time.

This broader view is consistent with an emerging host-control framework for understanding the gut microbiome. The colon contains a dense, competitive and metabolically active microbial ecosystem that must remain spatially organised and functionally contained [65–67]. Host-microbiome homeostasis therefore depends not only on microbial activity, but also on mucus integrity, epithelial barrier function, immune surveillance, antimicrobial peptides, secretory immunoglobulins, regulated transit and controlled access of microbes and metabolites to the mucosal

surface [68,69]. From this perspective, prebiotic benefit should not be equated simply with increased microbial growth or enrichment of selected taxa. A substrate that stimulates microbial activity may be beneficial when this occurs within a stable and well-regulated ecosystem, but the same increase in fermentative activity may be less desirable where barrier function, transit, immune regulation or symptom tolerance is impaired [9,19,24,37].

8. Complex dietary fibre, host tolerance and mopping effect

Structurally complex dietary fibres may contribute to host control through mechanisms that are not limited to microbial fermentation. High molecular weight fibres, including non-starch polysaccharides such as cellulose, hemicellulose, pectin, gums, mucilages and β -glucans, differ from rapidly fermentable low molecular weight substrates in molecular size, physical structure, viscosity, water-holding capacity, gel-forming potential and effects on stool bulk [3,4,70]. These properties influence microbial access to substrates, transit, stool consistency and the physical environment of the colonic lumen [18,19]. This distinction is particularly relevant to functional bowel disorders, where symptoms may depend not only on whether a substrate is fermented, but also on the speed and location of fermentation and the resulting gas production, luminal distension and osmotic effects. Rapidly fermentable low molecular weight substrates may be less well tolerated in individuals with visceral hypersensitivity, whereas more slowly fermented or minimally fermented high molecular weight fibres may offer advantages through stool normalisation, water retention and lower acute fermentative burden [9,18,39]. This proposed continuum between rapidly fermentable low molecular weight substrates and more structurally complex high molecular weight fibres is illustrated in Fig. 3.

While many recognised low molecular weight prebiotics are primarily valued for their microbial effects, complex dietary fibres may also exert direct physical and rheological effects. Psyllium, for example, is a gel-forming fibre with high water-holding capacity and has been described as stool-normalising, with the capacity to soften hard stool in constipation and firm loose stool in diarrhoea [18,70]. Such effects are not fully explained by selective microbial stimulation, but by the interaction between fibre structure, hydration, viscosity, transit and stool form. These properties provide a rationale for the “mopping effect” described by So et al. (2021). In their original use, this term referred to a putative mechanism by which physical changes in luminal contents may reduce mucosal or epithelial exposure to deleterious substances. They also noted that slowly and minimally fermented fibres with bulking characteristics, including psyllium and sterculia, inhibited hydrogen sulphide release in faecal slurries, possibly through absorption of gaseous compounds or by limiting microbial access for fermentation [18].

Building on this concept, future research should examine whether the “mopping effect” can be extended to a broader host-control hypothesis. Specifically, this would test whether structurally complex, slowly or minimally fermented fibres can reduce mucosal exposure to microbial biomass, microbial metabolites, secondary bile acids, proteolytic fermentation products and other potentially harmful luminal substances by sequestration, dilution, reduced diffusion, altered microbial access and enhanced faecal clearance. Emerging preclinical evidence that insoluble dietary fibre can adsorb particulate contaminants such as microplastics further supports the plausibility of fibre-mediated sequestration, although its clinical relevance in humans remains uncertain [71]. The expanded mopping-effect hypothesis therefore remains mechanistically plausible, and should be tested using direct measures of microbial load, mucosal microbial proximity, metabolite binding, epithelial permeability, immune activation and clinical tolerance.

9. The short-chain fatty acid paradox: an emerging hypothesis

Short-chain fatty acids are frequently presented as a central mechanism through which prebiotics and dietary fibres benefit the host, with acetate, propionate and butyrate implicated in epithelial metabolism, immune regulation, barrier function, luminal pH regulation and gut–brain signalling [3,72,73]. However, their effects should not be assumed to be universally beneficial under all conditions. Like many biological mediators, their physiological effects depend on concentration, site of production, timing, absorptive capacity and host context [74,75]. This creates a potential short-chain fatty acid paradox that may contribute to the broader fibre paradox. Slow and spatially distributed fermentation of structurally complex dietary fibres may generate physiologically useful concentrations of short-chain fatty acids across the colon. By contrast, rapid fermentation of low molecular weight prebiotic substrates may generate more abrupt, localised or excessive short-chain fatty acid exposure, particularly when intake is high or host tolerance is impaired [17,19,76].

At appropriate concentrations, butyrate supports epithelial integrity and contributes to anti-inflammatory signalling. At excessive or poorly localised concentrations, however, SCFAs may alter epithelial barrier function, influence immune signalling and affect gut sensorimotor activity [72,74,75]. This concentration-dependent interpretation is biologically plausible because SCFAs are weak organic acids. Weak organic acids are used as food preservatives. In their undissociated form, they can diffuse across microbial membranes and dissociate within the cell, releasing protons and anions that disrupt intracellular pH regulation, increase energetic stress and inhibit microbial growth; these effects depend strongly on acid concentration, pH and cell physiology [77,78]. Although this antimicrobial mechanism cannot be directly extrapolated to human intestinal cells, it supports the broader principle that weak organic acids can have different biological effects according to concentration and exposure conditions. This provides a plausible basis for considering whether excessive or poorly localised SCFA exposure in the gut may have effects that differ from normal physiological exposure.

The distinction between physiological and excessive or poorly localised SCFA exposure may be particularly relevant in individuals with diarrhoea-predominant symptoms, rapid transit or visceral hypersensitivity. In these contexts, increased luminal fermentation products, including SCFAs, may influence enteroendocrine and neural pathways involved in motility, secretion and visceral sensation, thereby contributing to urgency, pain or altered bowel habit [29,30,79,80]. Recent metabolotyping data further challenge the assumption that greater faecal SCFA enrichment is necessarily protective in all clinical contexts. In a post hoc analysis of baseline samples from a multicentre IBS-D trial, an SCFA-enriched faecal metabolotype was identified in almost one-third of participants and was associated with greater abdominal pain, urgency, increased stool frequency and faster whole-gut transit [81]. However, as emphasised in subsequent correspondence, faecal SCFA enrichment should be interpreted cautiously because it may reflect increased production, reduced absorption, altered microbial metabolism, rapid transit or differences in stool water content, rather than a direct causal mechanism [82].

Thus, a refined prebiotic framework should consider where SCFAs are generated, how rapidly, in whom and with what clinical consequences. This supports greater attention to fibre structure and fermentation kinetics, particularly when aiming for a slower, more spatially distributed and clinically tolerable metabolic response.

10. Conclusion and future research priorities

Rapidly fermentable low molecular weight carbohydrates occupy a contradictory position in human nutrition. Inulin-type fructans, fructo-oligosaccharides and galacto-oligosaccharides are widely promoted as prebiotic substrates, yet the same or closely related compounds may also be restricted as FODMAPs in clinical nutrition or reduced as flatulence-producing and tolerance-limiting components in food processing. Future research should aim to reconcile these concepts.

Microbial stimulation may be beneficial when it occurs within a stable, spatially contained and physiologically regulated gut ecosystem. However, rapid fermentation of low molecular weight substrates may contribute to gastrointestinal symptoms when fermentation load, gas production, luminal distension, SCFA accumulation or osmotic effects exceed host tolerance. This is particularly relevant in disorders of gut–brain interaction, altered motility, visceral hypersensitivity and inflammatory or barrier-compromised states.

Structurally complex high molecular weight dietary fibres differ from rapidly fermentable low molecular weight substrates because they may provide physical and rheological functions in addition to microbial effects. Their roles in stool normalisation, slower or more distributed fermentation, mucosal protection and the proposed mopping effect remain mechanistically plausible but require direct experimental and clinical testing.

Future studies should therefore compare rapidly fermentable low molecular weight substrates with structurally complex high molecular weight fibres using integrated outcomes, including microbial composition and load, metagenomic function, metabolomic output, gas production, SCFA profiles, transit, mucus integrity, epithelial barrier function, immune activation, stool rheology and patient-reported tolerance. Such studies are needed to determine when microbial stimulation represents host benefit, when it represents excessive fermentative burden, and when fibre supports health through host control rather than microbial enrichment alone.

Author contributions

Fandi Ibrahim: Conceptualisation, literature evaluation, writing – original draft, visualisation, writing – review and editing.

Declaration of generative AI and AI-assisted technologies in the manuscript preparation process

During the preparation of this work, the author used ChatGPT to support language refinement and improve readability. The author also used an OpenAI image-generation tool to create Figs. 1–3 based on text prompts and scientific instructions provided by the author. After using these tools, the author reviewed, edited and verified the generated content as needed and takes full responsibility for the content of the manuscript.

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Conflict of interest

The author declares no conflict of interest.

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