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**Dietary fibre in gastrointestinal health and disease**

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Fibre, microbiome, fermentation, prebiotics, IBS, IBD

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**Abstract**

Epidemiological studies consistently demonstrate the benefits of dietary fibre on gastrointestinal health through consumption of unrefined whole foods, such as wholegrains, legumes, vegetables and fruits. Recent mechanistic studies and clinical trials on isolated and extracted fibres have demonstrated promising regulatory effects (e.g., digestion and absorption, transit time, stool formation) and microbial effects (e.g. changes in gut microbiota composition, fermentation metabolites) that have important implications for gastrointestinal disorders. In this review, we detail the major physico-chemical properties and functional characteristics of dietary fibres, their importance, and current evidence for their use in the management of gastrointestinal disorders. It is now well established that the physico-chemical properties of different dietary fibres (e.g. solubility, viscosity, fermentability) vary greatly depending on their origin and processing and are important determinants of their functional characteristics and clinical utility. While progress in understanding these relationships has uncovered potential therapeutic opportunities for dietary fibre, many clinical questions remain unanswered. For example, clarity on the optimal dose, type and source required in both the management of clinical symptoms and prevention of gastrointestinal disorders. The use of novel fibres and (or) the co-administration of fibres is an additional therapeutic approach yet to be extensively investigated.

Key words: microbiota, fibre, fiber, solubility, viscosity, fermentability, prebiotics

**Introduction**

The term ‘dietary fibre’ was first coined in 1953, and following the first formal definition in 1976, the definition of dietary fibre has continued to be refined in light of research and the growing evidence demonstrating its health benefits. Finally, in 2009, following nearly twenty years of discussions from the scientific community across World Health Organisation (WHO) member nations, the CODEX Alimentarius definition of dietary fibre was accepted as ‘*all carbohydrates that are neither digested nor absorbed in the small intestine and have a degree of polymerisation (DP) of ten or more monomeric units*’1 2. The CODEX definition has subsequently been adopted by many countries and in doing so has encouraged international consistency in nutrition labelling, food composition tables and published research. However, there is flexibility allowing regional authorities to include carbohydrates with a DP of 3-9 monomeric units in the fibre definition. Subsequently, the European Food Safety Authority (European Union) 3 and the Food and Drug Administration (United States of America)4 adopted the wider definition of dietary fibre as all carbohydrates that are neither digested nor absorbed in the small intestine and have a DP of three or more monomeric units. In addition, the European Food Safety Authority and the Food and Drug Administration specify that synthetic and extracted fibres that are not intrinsic to plant cells must also demonstrate physiological effects to human health prior to be declared a dietary fibre. With this definition in mind, CODEX fibre includes non-starch polysaccharides such as cellulose, hemicelluloses and pectins, resistant starch and non-digestible oligosaccharides such as inulin and oligofructose, as well as lignins. Therefore, foods high in dietary fibre include wholegrains, legumes, vegetables, fruits, nuts and seeds.

Analytical methods to quantify dietary fibre have evolved alongside the updated definitions, with the most recent AOAC method (2011.25) considered the most reflective of the CODEX definition, capturing and enabling quantification of most dietary fibre entities, including total dietary fibre and its insoluble and soluble fractions by enzymatic-gravimetric assay, and molecular weight by size exclusion chromatography or high performance liquid chromatography quantification. Despite this, most food databases still include fibre values derived from outdated AOAC methods. Furthermore, investigating the health effects of fibre is complicated by variations in the interventions. Studies can investigate synthetic fibres consisting of only one type of molecule (e.g. fructo-oligosaccharides), extracted fibres from naturally occurring plant sources consisting of one, or a limited number of fibres (e.g. alginate or psyllium), single foods containing a limited number of naturally occurring fibres that are ‘intrinsic and intact’ in plant cells (e.g. prunes or wholegrain cereals), and high fibre diets consisting of a wide range of different naturally occurring fibres from a wide range of different foods. The variations in fibre interventions creates numerous challenges in interpreting and applying the findings. Firstly, *in vitro* and animal studies frequently use synthetic or extracted fibres in supplemental form but whose physico-chemical characteristics such as molecular weight and bioaccessibility may be different when consumed as whole foods and diets that can affect functional properties, and secondly because high fibre foods and diets contain other nutrients and food components (e.g. vitamins, polyphenols) that may be beneficial to health and thus identifying the effect of fibre alone can be challenging.

Dietary fibre has been shown in an extensive number of epidemiological and intervention studies to have important associations with the development and management of various diseases and with mortality. For example, the Scientific Advisory Committee on Nutrition (SACN)5 in the United Kingdom performed meta-analyses of epidemiological studies of fibre in the prevention of disease, showing that for each increase of dietary fibre intake from food of 7 g/d there was a statistically significant lower risk of cardiovascular disease (RR 0.91 [95% CI 0.88-0.94]; p<0.001), haemorrhagic plus ischemic stroke (RR 0.93 [0.88-0.98]; p=0.002), colorectal cancer (CRC) (RR 0.92 [0.87-0.97]; p=0.002), rectal cancer (RR 0.91 [0.86-0.97]; p=0.007) and diabetes (RR 0.94 [0.90-0.97]; p=0.001). More recently, a meta-analysis of 185 epidemiological cohort studies including just under 135 million person-years echoed these findings, showing that risk reduction was greatest when dietary fibre intake from food was between 25 g/d and 29 g/d, and higher fibre intake was associated with lower risk of all-cause mortality (RR 0·85 [0·79-0·91]), mortality from coronary heart disease (RR 0·69 [0·60-0·81]) and cancer (RR 0·87; 0·79 to 0·95); and the incidence of coronary heart disease (RR 0·76 [0·69-0·83]), stroke (RR 0·78 [0·69-0·88]), type 2 diabetes (RR 0·84 [0·78-0·90]), and CRC (RR 0·84 [0·78-0·89])6. Both observational analyses highlight the critical importance of the quantity of fibre required to elicit health benefits5,6.

These well-established associations between dietary fibre intake and health have resulted in the majority of countries recommending a daily intake of between 25-35 g/d. Despite this, the average intake of dietary fibre by adults across the globe remains low, typically under 20 g/d7.

As well as disease prevention, dietary fibre has potential to be used as a therapeutic intervention, in particular for disorders of the gastrointestinal tract. National and international guidelines provide some recommendations in relation to dietary fibre in the treatment of gastrointestinal disorders such as irritable bowel syndrome (IBS)8,9, inflammatory bowel disease (IBD)10, diverticular disease11,12, and in the management of specific gastrointestinal symptoms such as constipation13,12 and diarrhoea14. However, these recommendations are often limited, failing to provide specifics in terms of the type and dose of fibre and are sometimes even conflicting. The limited number and quality of studies as well as the variations in the fibre interventions (including fibre type, source, dose and duration of treatment) represent key challenges to providing recommendations for the therapeutic use of dietary fibre in the treatment of gastrointestinal disorders.

The impact of dietary fibre on gastrointestinal health and as a therapeutic agent in gastrointestinal disorders is attributed to its impact on nutrient digestion and absorption, improving glycaemic and lipaemic responses, regulating plasma cholesterol through limiting bile salt resorption, impacting gut transit, and microbiota growth and metabolism. Mechanistic research highlights the diverse physico-chemical characteristics of different dietary fibres, such as solubility, viscosity and fermentability, all of which determine their function in the upper and lower gastrointestinal tract.

The aim of this review is to discuss the physico-chemical and functional characteristics of dietary fibres and the impact of these on the clinical application of fibre in gastrointestinal disorders.

**Physico-chemical characteristics of fibres**

The majority of dietary fibres are the structural polysaccharide components of plant cell walls (Figure 1, Table 1). Cell walls contain multiple polysaccharides and the complexity in elucidating their functions results from the variety of sources and their functions within the cell. This is most evident in the variation in their molecular structure, which includes the composition of the polymer subunits, but also extends to the polymer linkages and side-chains (esterification). These differences in molecular structure of dietary fibres can significantly alter their physico-chemical properties and their behaviour in the gastrointestinal tract. For example, the resistance to intestinal digestion can result from the spacial orientation of polymer subunits, branching, or the presence of side-chains.

Food processing provides an additional level of complexity. Indeed, both milling and cooking can also be important determinants, improving starch digestibility and degradation of plant-derived compounds15. However, some digestible polysaccharides can also be classified as dietary fibre due to their inaccessibility to digestive enzymes within the food matrix, such as type-1 resistant starch (as in whole grains) or, type-3 resistant starch (retrograded), where resistance can be conferred following cooking and cooling. The consequence of these small variations in structure is that dietary fibres can have very different physico-chemical characteristics (e.g. viscosity, fermentability) that impact their functional effects (e.g. transit time, microbiome) in the gastrointestinal tract.

***Fibre solubility***

Solubility refers to the extent to which dietary fibres can dissolve in water. Unlike insoluble fibres that remain as discreet particles, soluble fibres have a high affinity for water. In cases where it is necessary to divide the dietary fibre content into soluble and insoluble fibre fractions, the enzymatic-gravimetric assay is often used for routine analysis (AOAC method 2011.25).

An example of the structural differences affecting solubility is starch (amylose and amylopectin) and cellulose, the former being composed of α-glucose monomers and the latter β-glucose. The corresponding secondary structures result in starch being soluble (the majority of which is digested in the small intestine) and cellulose being insoluble (therefore classified as dietary fibre). However, while β-glucose monomers linkages can result in β(1,4) cellulose being insoluble, they can result in β(1,3)(1,4)-glucan (mixed-linkages in beta-glucan) being soluble. Similarly, branching of the polymer structure such as amylopectin (starch), β-glucan or inulin can also affect solubility. Interestingly, the branching in amylopectin (starch) can result in increased solubility, whereas branching in β-glucan decreases solubility. Additionally, some fibres, such as pectin or methyl cellulose, contain side-chains along the polymer that provide resistance to digestion but alters solubility.

The majority of current evidence has focused on solubility as a characteristic of fibre in relation to its impact on the upper gastrointestinal tract through the regulation of gastric emptying and nutrient absorption. Indeed, early *in vitro* studies of isolated fibres allowed for the distinction between those that primarily impacted small intestinal lipid and glucose absorption and those that primarily impacted colonic function such as stool bulking and reduced transit time (insoluble fibres such as cellulose, wheat bran and lignin)16. Therefore, classifying fibres based upon solubility was for many decades used to allude to differentiation of their functional properties. However, the FAO proposed that these conventionally-classified terms relating to solubility should be phased out for a number of reasons17. Firstly, measuring and classifying fibre solubility *in vitro* is method-dependent. Secondly the varying pH conditions within the gastrointestinal tract (e.g. stomach *versus* colon) and between subjects may impact fibre solubility *in vivo*18,19. Thirdly, solubility alone does not predict the physiological effects of fibre and therefore its functional properties. For example, both psyllium (soluble) and cellulose (insoluble) have been shown to improve glycaemic control, transit time and stool output, albeit via different mechanisms. Glycaemic control is improved by psyllium20 through a mechanism involving increased viscosity of intestinal contents, whereas cellulose impacts glycaemia via inhibition of starch digestion by binding α-amylase21 thereby reduce glucose absorption22.

A further challenge to the use of fibre solubility as an indicator of functionality, is that in reality, whole fibrous foods are often a complex mix of soluble and insoluble fibres (e.g. resistant starch, hemicelluloses, cellulose, lignin) and therefore simultaneously exert different physiological effects in the gastrointestinal tract. For example, apples contain soluble (pectins) and insoluble (cellulose) fibre fractions. It has been suggested that the effects of both soluble (i.e. swelling via water absorption) and insoluble (i.e. bulking) fibres in the ileum may activate the ileal brake via mediators such as GLP-1 and GLP-223. Nonetheless, while solubility *per se* is a poor indicator of physiological function in isolation, it has a profound impact on other factors such as viscosity and fermentability that have since gained recognition for their specific physiological and microbial actions in the gastrointestinal tract.

***Fibre viscosity***

Viscosity is the degree of resistance to flow. It is generally associated with soluble dietary fibres (e.g. gums, pectins, β-glucans, psyllium) and relates to the ability of a fibre, when hydrated, to thicken in a concentration-dependent manner. Some forms of fibre, such as pectins, have the capacity to form gel networks. In the gastrointestinal tract, this process can begin in the mouth and continues throughout the digestive tract. There are several physico-chemical characteristics that contribute to the viscosity potential of fibre, including the length and structure of the polymer as well as its charge. These factors affect the ‘type’ of gel formed and the critical concentration required for the formation of a viscoelastic gel. Broadly, viscous fibres can be categorised into two groups, ‘random coil polysaccharides’ and ‘ordered assembly polymers’. Random coil polysaccharides increase viscosity through entanglement, thereby restricting the flow of the surrounding solvent24. Examples include the neutral polymers β-glucans, psyllium and guar galactomannan, where generally the longer the polymer (i.e. higher the molecular weight), the greater the entanglement occurs and therefore the lower the concentration required to increase viscosity25. In contrast, ordered assembly polymers, such as some pectins and alginate, form a gel network in the presence of divalent ions (i.e. Ca2+). Increasing gut luminal viscosity is proposed to have multiple health benefits. Consumption of viscous dietary fibre has been shown to alter transit time in the upper gut, including decreasing gastric emptying rate and modulating small intestinal transit. Increased luminal viscosity is postulated to play a role in significant regulatory effects of dietary fibre consumption, including delaying digestion, decreasing postprandial glycaemia26 and lipaemia27,28,29 and increasing satiety30. The effect of the visco-elastic properties in the small intestine are less well defined, particularly as the impact of digestive secretions that dilute luminal contents are difficult to replicate and test *in vivo*. Indeed, the effects of simulated gastric and small intestine digestion *in vitro* on the thickening ability of six soluble fibres of different sources, found significant differences in their viscosity profiles31.

Viscosity remains the accepted model for the cholesterol lowering capacity of β-glucan. Increased luminal viscosity decreases diffusion of bile salts preventing their resorption in the distal ileum32. Malabsorbed primary bile salts entering the colon can be de-conjugated by bacterial hydrolases to produce secondary bile acids, which have been shown *in vitro* to increase the risk of CRC33. While the mechanism remains unclear, as does the interaction between bile acids and dietary fibre34, this elevated CRC risk is not observed *in vivo*. Indeed there is substantial epidemiological evidence suggesting the opposite, that diets high in fibre are protective against CRC35. Additionally, *in vitro* studies suggest potential interactions of dietary fibres (e.g. rice bran fibre, cellulose) with digestive enzymes inhibiting the rate of nutrient digestion21,36,37 .More recently an additional mechanism has been proposed, where by an interaction between dietary fibres and the mucus layer results in localised increases in viscosity adjacent to the brush border38,39 regulating nutrient diffusion across it. In the colon, increased luminal viscosity alters water-holding capacity, which in turn can impact colonic bulk and transit time. The colonic contractions moving luminal content between compartments may also reduce localised viscosity by shear thinning and alter colonic transit, particularly with fibres that are able to form disordered networks when hydrated (e.g. pectins)40. The consequences of these changes are likely to influence the extent of fermentation occurring in the colon.

There are several mathematical equations and models, as well as rheological measurements to determine the viscosity of a solution. The two most common analytical techniques are rheometry (i.e. measures the flow of a fluid) and viscometry (i.e. measures the viscosity of a fluid)41, however, there is no standardised method of viscosity measurement.

***Fibre fermentability***

Observational studies have consistently observed differences between the faecal microbiota of industrialised and rural populations. This has been attributed to differences in the typical westernised diet consisting of foods that are highly refined and low in dietary fibre, particularly fermentable fibre42,43,44,45,46,47. Unlike mammalian cells, the gut microbiota is well equipped to degrade a range of dietary fibres packaged within plant-based foods48. Interestingly, a number of specialist primary degraders known as ‘keystone species’ have been identified within the large intestine. These species have a superior ability to degrade certain dietary fibres and release energy on which other bacterial communities depend. For example, *Ruminococcus bromii* has been shown to specifically degrade certain types of resistant starch49.

Examples of fermentable fibres include inulin-type fructans, galacto-oligosaccharides and resistant starch (Table 1). All natural plant fibres have some degree of fermentability, even cellulose and lignin (low fermentability), with only synthesized fibres such as methycellulose being completely non-fermentable (Table 1).

Fibre fermentability has traditionally been assessed using *in vitro* fermentation models of the digestive tract. Although this technique provides valuable mechanistic insight into fermentation patterns and behaviour of different dietary fibres, the findings are not always reflected *in vivo*. For example *in vitro* models have indicated psyllium is moderately fermented, whereas *in vivo* studies have demonstrated it to be poorly fermented50. This discrepancy is likely owing to the dilution and high speed mechanical blending that occurs *in vitro*, which destroys the gel network, artificially exposing the fibres to enzymatic degradation. *In vivo* methods of measuring fibre fermentability include quantification of short-chain fatty acids (SCFAs) in stools and hydrogen breath testing, although these also have limited interpretability. More recently, advances in functional magnetic resonance imaging (fMRI) have allowed measurement of colonic gas volumes in response to different fibres51,52. Although still in its infancy in terms of quantifying fermentation, fMRI overcomes many of the limitations of the above methods.

Intake of dietary fibre, particularly foods high in the fermentable fibres, has been associated with stool SCFA concentrations53. SCFAs play a number of key roles in the gastrointestinal tract. Animal studies have shown that SCFAs affect gastrointestinal motility by stimulating colonic contractile activity through increasing the number of excitatory cholinergic neurons54. SCFAs are also have a mediatory role, bridging communication between the mucosal microbiota and the mucosal immune system, with pre-clinical evidence suggesting significant anti-inflammatory and immunomodulatory effects with relevance to inflammatory disorders of the gut55. For example, SCFAs can influence intestinal adaptive immune responses through direct regulation of the size and function of the regulatory T cell pool, including proliferative capacity and gene expression56. SCFA have also been implicated in maintaining intestinal barrier integrity57 and regulating appetite via several mechanisms including the stimulation of gluconeogenesis in the liver58. Furthermore, SCFAs indirectly maintain gastrointestinal homeostasis *via* the reduction of luminal pH which may be important in preventing colonisation and inhibiting growth of acid-sensitive enteropathogens (Figure 2).

Many factors can affect the rate, site and extent of fibre fermentation including the composition of the microbiota (i.e. degradation capacity) with different microbes shown to preferentially metabolise different fibres59 and the availability of other substrates (i.e. proteins that have escaped digestion)60. Nonetheless, the key factor influencing fibre fermentability is thought to be its physico-chemical characteristics (e.g. solubility, viscosity, accessibility). This was exemplified by an *in vitro* fermentation study using human stool samples from three donors to investigate the impact of 15 dietary fibres on SCFA production. Despite marked inter-individual differences in microbiota composition between donors, SCFA production (concentrations and proportions) from the different fibres was reproducible between samples. More specifically, rhamnose followed by galactomannans produced the highest proportion of propionate while fructans and other α- and β-glucans produced the highest proportion of butyrate61.

The impact of fermentable fibres on SCFA production has been consistently shown *in vitro*62,63,64,65. In contrast, human intervention studies are somewhat limited in interpretation given that approximately 95% of SCFAs are absorbed by colonocytes, and therefore faecal SCFA only represent 5% of the total SCFAs produced. Therefore, faecal SCFA better reflect the dynamic processes of both SCFA production and SCFA absorption, the former being affected by fibre source, colonic transit time and the microbiome and the latter being greatly affected by colonic transit time66.

Reducing fermentable fibre intake may reduce bacterial diversity. To date, several animal studies have shown that gut microbiota deprived of fermentable fibres shift to degrading and extracting alternative energy from the glycoprotein-rich mucus layer that acts as a protective and mechanical barrier to pathogens67,68. Indeed, reduced availability of fermentable fibres leads to a thinner mucus layer69,70,71, which in turn may compromise intestinal epithelial integrity and increase pathogen susceptibility. Reduced availability of fermentable fibres also cause a shift from a stable microbial intestinal environment to one that is temporarily or permanently altered, often characterised by reduced bacterial diversity and richness, a state that is commonly referred to as dysbiosis72. A dysbiotic intestinal environment is a common feature of a number of gastrointestinal disorders, for example, decreases in Bifidobacteria73,74 and Firmicutes75,76,77 have been commonly observed in IBS and IBD, respectively.

***The food matrix: particle size, surface area and porosity***

The nature of the food matrix in which a dietary fibre is delivered will significantly influence the extent of its physiological function. More specifically, the particle size and integrity of the plant cell walls affects the dissolution of soluble fibre78. This has the potential to significantly affect luminal viscosity and reduce the rate of fermentation. Further, the cell wall integrity can also encapsulate the intracellular starch79, thus reducing digestion by endogenous enzymes and increasing the substrate available for microbial fermentation (RS-1, Figure 1).

Particle size can influence fibre fermentation, although to date, the majority of research has been conducted *in vitro*, with limited translational research investigating the effects in humans. Lower particle size of fibres has been associated with higher SCFA concentrations80,81, suggesting greater bacterial fermentation. This is likely due to smaller particles having greater external surface area exposure for bacterial enzymes. Similarly, the physical act of chewing and grinding high fibre foods can play a notable role in particle size kinetics by increasing surface area and total pore volume as well as structural modification. For example, reducing the particle size of coconut residue from 1127 to 550 μm led to an increase in hydration properties including water-holding, retention and swelling capacity82. Equally, the physical and mechanical effect of large/course insoluble fibre particles on the colonic mucosa has been shown to stimulate the secretion of water and mucus into the lumen, contributing to stool output (i.e. consistency and weight)83,84.

Porosity is the ability of enzymes or bacteria to diffuse into particles, which can significantly influence the fermentability of a fibre. Low porosity of a food matrix can result from maintenance of cell wall structures in the small intestine and therefore inability of digestive enzymes to access intracellular starch, thus leading to increased RS-1 resistant starch, such as the case with whole chickpeas. Low porosity of a food matrix in the large intestine, can also impede fermentative degradation85. Dietary fibre preparations high in insoluble fibres such as cellulose are likely to have low porosity whereas those containing more soluble fibres such as pectin have a higher porosity, which contributes to their differences in fermentability (Table 1).

**Functional characteristics of dietary fibre**

***Micronutrient bioavailability***

Dietary fibre can influence nutrient bioavailability beyond merely limiting micronutrient accessibility within a food matrix. Cereals containing dietary fibre (e.g. wheat) are a vital source of non-haem iron, however, other cereal components (e.g. bran fractions) contain concomitant factors such as phytate that reduce absorption of iron, zinc and calcium86. Additionally, in wheat, the aleurone contains the majority of the iron, but is encapsulated by the cell walls (predominantly dietary fibre). Recent studies have shown that micro-milling to disrupt these structures results in increase mineral bioavailability87,88. In contrast to iron, several studies have shown that consumption of specific fibres, such as fructans89 and galactoologosaccharides90, may increase absorption of dietary calcium. The proposed mechanism for these findings involves the decreased colonic pH resulting from the SCFA production, which in turn can increase calcium solubility, thereby increasing passive transport of calcium across the intestine.

The influence of dietary fibre consumption on vitamin absorption remains unclear. Several studies have demonstrated that “fibre” improves absorption of some water-soluble and fat-soluble vitamins91,92, others have shown no effect93. For example, fibre may enhance the net colonic bacterial biosynthesis of B vitamins such as folate94, while other studies report increased faecal excretion of vitamins following dietary fibre consumption95. The variety of fibre sources with varying physico-chemical properties demonstrates that further work is required.

***Gut transit time***

Abnormal whole gut transit time is found in some gastrointestinal disorders, which some types of fibre may normalise. For example, fermentable fibres may indirectly modulate contractile activity *via* production of SCFAs96, while non-fermentable fibres contribute to stool weight that increases colonic volume and thus stimulates contractility. Indeed, a systematic review and meta-analysis reported that transit time decreased in a dose-dependent manner by 0.78 h per additional 1 g/d of wheat fibre97. Additionally, wheat fibre particle size has been shown to influence stool output, whereby coarse wheat fibre resulted in higher stool weight (mean 219.4g/d) compared with fine wheat fibre (199 g/d) in healthy humans98.

Measuring gut transit time using radio-opaque markers and scintigraphy have shown dietary fibre interventions reduce gut transit time99,100,101,102. More recently, a wireless motility capsule (SmartPill®) which correlates with radio-opaque markers and scintigraphy103,104 has been shown to be a more practical and less invasive method in determining gastric emptying time and whole gut transit time. One study showed a decrease in transit time with wheat bran from 70 hours to approximately 46 hours105. One controlled cross-over trial in healthy subjects with background fibre intakes of between 14-15 g/d who consumed an additional 9 g/d of wheat bran or a low-fibre control for 3 days found whole gut transit time (-8.9 h) and colonic transit time (-10.8 h) were lower during wheat bran supplementation compared to the control group106. Insoluble, poorly fermented fibres (e.g. wheat bran) have a greater impact on reducing gut transit time in healthy controls and to a lesser extent in those with constipation107 than fermentable fibres that do not remain physically intact throughout the large intestine.

***Stool forming***

For a dietary fibre to exert effects on stool output (i.e. frequency, consistency and weight), it must possess certain physico-characteristics. Dietary fibre has been shown to increase stool frequency from 1/day to 1.5/day with wheat bran supplementation105, although the type of bowel movement (e.g. complete or spontaneous) was not reported, while further studies have observed increased stool frequency with cellulose/pectin combination108, psyllium109 and high-fibre breakfast cereals110.

Both soluble, viscous fibres and insoluble, non-viscous fibres can contribute to improvements in stool consistency and stool weight (bulk). Soluble, viscous fibres (e.g. psyllium) with a high water-holding capacity that are resistant to fermentation and form a viscoelastic substance in the gastrointestinal tract, contribute to softening hard stool and increasing stool bulk, making them easier to pass111,112. These properties also assist with diarrhoea by firming loose stools and slowing transit time113,114,115. In contrast, insoluble, non-viscous fibres (e.g. coarse wheat bran) can contribute to improvements in stool consistency and stool weight *via* the mechanical stimulation of the intestinal mucosa116,84. Previous trials have shown improvements in stool consistency in adults with self-reported constipation and higher intakes of rye bread consumption117 and higher fibre bread consumption118. Theoretically, fermentable fibres may contribute, in part, to stool weight *via* increasing microbial mass, but the effect size is limited compared with non-fermentable fibres. For example, results from six trials showed that resistant starch (RS2) supplementation increased stool weight (+38g/d, 95% CI 23, 53; p<0.001) in doses ranging from +21.5 to +37 g/day5. Outcomes from a review of intervention trials concluded that fermentability determined the role of fibre in total stool weight, with less fermentable fibres from cereals contributing most to stool weight119.

***Microbial specificity (prebiotics)***

Some fermentable fibre are also classed as prebiotics, a term whose definition has been updated to ‘a substrate that is selectively utilized by host microorganisms conferring a health benefit’120. Examples of prebiotic fibres include the inulin-type and galactooligosaccharides.

Prebiotic fibres are known for their rapid fermentative capacity and subsequent release of SCFAs, in particular acetate, but selectively stimulate the growth of only a specific range of genera/species (i.e. Bifidobacterium and Lactobacillus) 121,122,123. This is due to specific gene clusters within the bacterial genome that dictate the saccharolytic enzymes they produce and their phenotypic ability to selectively metabolize the prebiotic substrate124,125.

The first landmark study demonstrating the prebiotic effect in healthy volunteers reported supplementation of 15 g/d of oligofructose or inulin increased luminal bifidobacterial by almost +1 log10126. Subsequent research has demonstrated a dose-dependent effect on luminal Bifidobacterium with oligofructose109 and galactooligosaccharides127. Most recently, a systematic review and meta-analysis of 64 randomised controlled trials in humans demonstrated fibre, and in particular fructans and galactooligosaccharides and other candidate prebiotic fibres, increased abundance of Bifidobacterium and Lactobacillus121.

However, large inter-individual variability in gut microbiota responses to dietary fibre have been reported128,129,130,131,132. A randomised controlled crossover trial in 34 healthy participants found that those with a habitually high dietary fibre intake had a greater gut microbial response to prebiotics (e.g. increases in Bifidobacterium and Faecalibacterium, and decreases in Coprococcus, Dorea and Ruminococcus) compared to those with habitually low dietary fibre intake, suggesting those following a habitual high fibre diet are more likely to benefit from an inulin-type fructan prebiotic133.

**Dietary fibre in gastrointestinal disorders**

Given the significant inter-condition and inter-individual variability in response to dietary fibre, there remains the complex challenge of unravelling which fibres are most appropriate for which gastrointestinal disorders. Consideration of the diverse physio-chemical characteristics of fibre and how these translate to functional characteristics is fundamental to optimising any clinical benefit (Figure 3). Consideration should also be given to maintaining the balance between optimising symptom benefit (i.e., management and maintenance) and limiting symptom exacerbation (i.e., tolerance). The manipulation of dietary fibre is a common approach in clinical practice for many gastrointestinal disorders and is commonly recommended as first line therapy in the management of several gastrointestinal symptoms (Table 2), despite the limited number of randomised controlled trials across gastrointestinal disorders, all of which use heterogenous methodologies (e.g. fibre type, amount, duration).

***Irritable bowel syndrome***

IBS is a functional gastrointestinal disorder characterised by recurrent abdominal pain and change in stool habits (i.e. constipation, diarrhoea or both), often alongside abdominal bloating and distension. The mechanisms underpinning IBS symptoms include visceral hypersensitivity, alterations in gut-brain interactions, immune activation, gut motility and the gut microbiota. Since the revised Rome IV criteria was introduced in 2016, estimated prevalence of IBS has decreased from 11.7% (Rome III) to 5.7% (Rome IV)134.

Guidelines for fibre consumption in IBS vary, with the National Institute for Health and Care Excellence8 in the UK recommending reducing resistant starch intake, whereas the World Gastroenterology Organisation Global Guidelines135 suggest encouraging fibre-rich foods or fibre supplements (e.g. psyllium), while limiting insoluble fibres that may exacerbate symptoms.

To date, a number of systematic reviews and meta-analysis have concluded that some fibres are beneficial in reducing IBS symptoms and improving stool frequency and consistency, although results are inconsistent with wide variation in responses136,137,138. Benefits appear to be limited to soluble fibre (relative risk of having continued symptoms after supplementation, RR=0.83; 95% CI 0.73–0.94)139, compared to other fibres such as bran (RR=0.90; 95% CI 0.79–1.03). The ongoing changes in diagnostic criteria (e.g., Rome Criteria for IBS), and lack of standardisation in outcome measures reported between studies presents major challenges when attempting to compare findings of previous studies. Further rigorous and long-term randomised controlled trials are required and there is an urgent need to assess the different functionalities of dietary fibres in sub-groups of IBS to allow for a better understanding of its therapeutic potential.

Interestingly, a recent 3-period, crossover mechanistic study revealed that despite similar physiological responses to prebiotics between patients with IBS and controls, only those with IBS experienced symptoms when challenged with 40 g of fructose or inulin compared to healthy controls, suggesting that visceral hypersensitivity to colonic gas is involved in the induction of symptoms, rather than excessive gas production, *per se*140.

A limited number of clinical trials investigating prebiotic supplementation (e.g., oligofructose, fructo-oligosaccharide, and β-galacto-oligosaccharides) ranging from 3.5-20 g/d over 4-12 weeks have been conducted in IBS populations with mixed results141,142,143,144. Results from a recent systematic review and meta-analysis of 11 randomised controlled trials in 729 patients showed that although bifidobacteria increased following prebiotic supplementation, no differences in response rates, or severity of abdominal pain, bloating, flatulence, or in quality of life were observed145. Notably, sub-group analysis showed a low prebiotic dose (≤6g/d) and non inulin-type fructans (e.g. GOS, guar gum) improved flatulence (SMD: -0.35; 95% CI: -0.71, 0.00; p=0.05 and SMD: -0.34; 95% CI: -0.66, -0.01; p=0.04, respectively). While there is little current evidence for the use of prebiotics in IBS management146,145, emerging evidence suggests that second generation candidate prebiotics such as pectin and partially hydrolysed guar gum have bifidogenic properties147 with potential therapeutic use in IBS148, possibly due their viscosity characteristics and therefore slower fermentation rate.

***Inflammatory bowel disease***

Inflammatory bowel disease (IBD) encompasses Crohn’s disease (CD) and ulcerative colitis (UC), both chronic, relapsing gastrointestinal disorders of which the pathogenesis remains incompletely understood, although a dysregulated mucosal inflammatory response in genetically susceptible individuals is responsible for the initiation and maintenance of IBD149. This involves alterations in immunological (e.g. T and B cell regulation) and microbial factors (e.g. diversity and functionality of bacteria such as *Faecalibacterium prausnitzii*). The prevalence of IBD exceeds 0.3% across North America, Oceania and across Europe150. The goal of IBD treatment is to halt disease progression, maintain remission and prevent reoccurrence of inflammatory episodes.

There continues to be debate on the clinical benefits of dietary fibre in IBD151 despite plausible mechanisms for therapeutic potential, including the production of SCFAs (particularly butyrate) that may attenuate intestinal inflammation through upregulation or downregulation of cytokine expression (e.g. IL-10, IFN-γ and IL-1β) by colonic epithelial cells56,152,153. SCFAs may also affect leukocyte activity including alterations in cell signalling and metabolism55. A number of studies have observed alterations in the gut microbiota (e.g. lower concentrations of Bifidobacteria and *Faecalibacterium prausnitzii*) in IBD, both of which may be amendable to prebiotic fibre supplementation154,155. Previous studies have generally reported lower stool SCFA concentrations in IBD compared to healthy controls156,157,158, suggesting a lower fermentative capacity and an impairment of SCFA production in this patient group.

Collectively, it is conceivable that fibre (dietary or supplement form) may prevent IBD, maintain or restore intestinal epithelial integrity in IBD, although trials of fibre in the prevention, maintenance and treatment of IBD are extremely limited151.

In terms of risk of developing IBD, a prospective cohort study of 170,776 women in the Nurses’ Health Cohort Study followed up over 26 years identified 269 and 338 cases of CD and UC, respectively. Compared to women with the lowest energy-adjusted dietary fibre intake, intake in the highest quintile (median of 24.3 g/d) was associated with a 40% reduction in risk of CD (Hazard ratio for CD, 0.59; 95% confidence interval, 0.39-0.90), but not UC159. On the contrary, a meta-analysis of 14 case-control studies observed an association of higher intakes of vegetables with lower risk of UC (odds ratio, OR=0.71, 95% CI 0.58–0.88), but not CD (OR=0.66, 95% CI 0.40–1.09), whereas a higher consumption of fruits was associated with lower risk of both UC (OR=0.69, 95% CI 0.49–0.96) and CD (OR=0.57, 95% CI 0.44–0.74)160. However, a large prospective study of 401,326 participants recruited from across eight European countries found no associations with intakes of total fibre or fibre from specific sources and the development of IBD161, however, this was in a population of people in their middle ages, beyond the age at which IBD commonly develops. Furthermore, given the nature of these observational studies, recall bias is likely.

Only a limited number of clinical trials have been undertaken of fibre in the maintenance and treatment of IBD, that have been summarised in a systematic review151. For maintenance of remission, of the four studies included in UC (n=213), two reported positive results on disease activity; one larger randomised controlled trial observed continued remission at 12 months across all groups (psyllium vs. mesalamine vs. psyllium plus mesalamine), whereas a smaller randomised controlled trial reported lower relapse rates at 12 months for psyllium plus mesalamine vs. mesalamine alone. Four studies in CD (n=465) observed equivalence in the number of patients with deteriorating disease at 24 months in high fibre (mean intake 27 g/d) vs. low fibre (mean intake 15 g/d) groups, one study reported negative outcomes for patients consuming a high fibre diet (33.4 ± 1.8 g/day, of which 2.9 ± 0.3 g/day was from raw fruit and vegetables) with significantly higher treatment failure and shorter time to relapse (1.4 *versus* 2.8 months) compared with patients on a low-fibre exclusion diet151. In contrast, an observational cohort study of 1619 patients with IBD reported that a higher fibre intake was associated with reduced risk of flare in CD (adjusted OR, 0.58, 95% CI 0.37–0.90), but not in UC (adjusted OR, 1.82; 95% CI, 0.92–3.60), although intakes were measured using 26-item self-reported, retrospective dietary survey162.

For treatment of active disease, the systematic review reported five trials in UC (n=114) observing positive effects of fibre (e.g. germinated barley, oligofructose/inulin) on disease activity. While five trials in CD (n=193) showed no positive effect, three studies showed equivalent effects of a high fibre diet compared with another dietary intervention in active, inactive, or mixed disease stage cohorts151.

Collectively based on these results, while there is limited evidence for dietary fibre in maintenance or treatment of IBD, dietary fibre should not be unnecessarily restricted in IBD, unless intestinal strictures are present and there is risk of obstruction. Overall, results suggest that UC may be more amendable to dietary fibre interventions than CD, potentially due to the formation of SCFAs at the site of disease151. Historically, it has been common in clinical practice to recommend reducing high-fibre foods during relapse, although this is not evidence-based, and patients should be monitored in regard to their tolerance to fibre during both remission and relapse.

***Diverticular disease***

Diverticular disease refers to herniation of the mucosa and submucosa through the muscular layer of the colonic wall163. The pathogenesis of diverticular disease relates to colonic smooth muscle over-activity, alterations in the colonic wall structure and/or genetics, in addition to lifestyle factors such as fibre intake and physical activity, although associations remain inconsistent164. The incidence of diverticular disease is highest in economically developed countries with cases continuing to increase165. Although, the majority of patients with diverticular disease remain asymptomatic (~80%), inflammation of a diverticulum presents as diverticulitis that can vary in duration and severity, but can be complicated by fistulae, abscesses, obstruction and perforation.

For risk of developing diverticular disease, a recent prospective cohort study of 46,295 men reported a positive association between a Western dietary pattern and an increased risk of diverticulitis. In particular, a higher consumption of red meat and lower consumption of dietary fibre166. Similar results were observed in another prospective cohort study of 690,075 UK women (part of the Million Women Study) whereby the relative risk for diverticular disease for a 5 g/d increase in fibre intake was 0.86 (95% CI 0.84 to 0.88). Notably, the source of fibre appeared to influence disease risk whereby fruit and cereal fibre had significant reductions in risk compared to vegetable and potato fibre167. Similarly, another cross-sectional study in 539 individuals found that a low-fibre diet was associated with an increased risk of diverticular disease168. The proposed mechanisms include low fibre intake leading to reduced stool bulk, promoting small, hard stools and increased colonic pressure and therefore herniation. In contrast, one observational study of 2104 participants undergoing colonoscopy reported that a high-fibre diet was not protective against the risk of diverticular disease169, in fact the highest quartile of fibre intake experienced an increased prevalence of diverticular disease (prevalence ratio = 1.30; 95% confidence interval, 1.13–1.50). However, it is important to note that dietary intake was measured up to 12 weeks following colonoscopy results and it is not possible to exclude alterations in diet as a consequence, rather than a cause, of the diverticular disease diagnosis.

During active diverticulitis, there is no consensus on fibre intake. Some guidelines suggest a low fibre diet to ‘minimise irritation’12 and a gradual increase to 20-30g/d through diet or as fibre supplements once inflammation has resolved170. These recommendations are often used in clinical practice, although are based on physiological rationale or uncontrolled studies and not robust clinical trials. Most recently, a systematic review recommended that patients with uncomplicated diverticulitis should be placed on a liberalised diet (e.g., solid food, no bowel rest or nil by mouth), as opposed to dietary restrictions, and a high fibre diet which meets individualised nutrient requirements, with or without fibre supplementation. However, it was recognised that recommendations were based on a limited number of low quality studies171.

In 2012, a systematic review172 of the effectiveness of fibre (dietary and supplement form) in diverticular disease found only three RCTs of sufficient quality, although yielding inconsistent findings173,174,175, while a more recent systematic review of 19 studies of varying design reported that both dietary fibre and fibre in supplement form appeared were effective in reducing abdominal symptoms and preventing diverticulitis. However, the studies presented were of low quality with methodological limitations and with significant heterogeneity between research design176. A further systematic review and meta-analysis of 20 studies concluded that dietary fibre in the prevention of diverticulitis remains unknown177.

***Functional constipation***

Functional constipation is one of the most common functional bowel disorders and is characterised by symptoms of difficulty or infrequent stool passage, or incomplete defaecation, without structural cause. Unlike IBS in which abdominal pain must be present, functional constipation does not have abdominal pain as a predominant symptom, and although not considered to be a serious condition, it can lead to complications such as faecal impaction, bowel perforation, haemorrhoids178 and the symptoms experienced are varied and place a burden on the patient179. At present, there is limited data available on the pathophysiology of functional constipation, although lifestyle factors appear to play a role in its aetiology including a low fibre intake and low levels of physical activity. The majority of studies have focused on chronic constipation where the estimated global prevalence is 14%111, although variations in symptoms used in self-reporting constipation result in varying numbers presenting to their doctor179.

A number of large cohort studies have observed positive associations between high intakes of dietary fibre and stool frequency180,181. A systematic review and meta-analysis of seven RCTs concluded that fibre is effective in treating chronic constipation in adults compared with placebo. For example, fibre (dietary (e.g. wheat bran) and supplement form (e.g. psyllium) including prebiotics (e.g. inulin)) was associated with increased stool frequency (SMD = 0.39; 95% CI 0.03–0.76; P = 0.03) and more normalised stool consistency (SMD = 0.35; 95% CI 0.04–0.65; P = 0.02). In particular, sub-group analysis suggested that a high dose (>15g/d) of psyllium was the most effective in increasing stool frequency and improving stool consistency112. However, fibre can induce other gastrointestinal symptoms such as flatulence compared to placebo (SMD 0.56, 0.12–1.00, P = 0.01).

This meta-analysis further highlights the importance of choosing fibres with the most appropriate physico-chemical characteristics to provide the preferred functional benefit. For example, non-viscous, highly fermentable fibres such as prebiotic fibres (inulin and GOS) did not consistently have any benefit over placebo in increasing stool frequency and stool consistency. This is due to these fibres being almost completely fermented in the colon and water‐holding capacity therefore being lost. In contrast, viscous and poorly fermented fibres such as psyllium retain their physico-chemical characteristics (e.g. high-water holding capacity and gel-forming) throughout the gastrointestinal tract. Indeed, while insoluble fibres containing large/coarse particles can provide a regulatory benefit83,182, there are fibres (wheat dextrin and finely ground wheat bran) that have been shown to contribute only to the dry mass of stool, resulting in decreased stool water content and a constipating effect, potentially exacerbating symptoms in those with constipation183. This may, in part, explain the disparities in previous studies reporting laxative effects with insoluble fibre184.

The effect of different quantities of different fibres on health have been previously highlighted5. One review that summarised the research of Burkitt, who advocated that >50 g/d of dietary fibre is required for chronic disease prevention185, noted that fibre intakes >35 g/d appeared to be more effective in reducing chronic disease than lower intakes. Moreover, it has been suggested that many of the human intervention trials on fibre supplementation to date showing minimal effects on health outcomes could simply reflect the insufficient quantity of fibre supplement provided in those studies185

**Future research: fibre co-administration and novel natural fibres**

Currently, extracted and isolated fibres (e.g. inulin, psyllium) from various sources are commonly added to food products to enrich the fibre content, in order to assist people achieving the dietary recommendations for dietary fibre.

It is plausible that co-administration of different fibres (e.g. combined isolated fibres) may provide a ‘dual treatment’ by driving different functionalities that target separate gastrointestinal features (i.e. the correction of dysbiosis, normalisation of stool form and transit time), potentially maximising the impact of clinically meaningful symptom improvement. Yet to date, there is limited research in the area of co-administration. A 3-week randomised crossover block-design study in 19 healthy volunteers found that wheat bran plus resistant starch (12g/d and 22g/d, respectively) produced greater benefits (e.g. higher stool output, shorter transit time, lower faecal pH, higher concentration of acetate and butyrate) above wheat bran (12g/d) alone186. Furthermore, an animal study demonstrated that by combining wheat bran with resistant starch versus wheat bran alone that fermentation can be shifted distally, thus potentially improving the luminal environment and offering protective effects further along the colon187. More recently, an *in vitro* study using faecal microbiota from healthy donors to compare degradation profiles of single fibres (arabinoxylan, chondroitin sulphate, galactomannan, polygalacturonic acid, xyloglucan) versus a combination of these observed slower utilisation of some soluble dietary fibres when present in a mixture, suggesting this strategy may be used as a means of delivering fibres to the more distal regions of the colon188. Further research is required to determine whether fibre combinations may be efficacious in the management of gastrointestinal disorders and beyond. As our understanding of the physico-chemical characteristics of dietary fibre advances, co-administration of known fibres with different functional characteristics are likely to offer greater therapeutic utility.

Natural sources of dietary fibre are increasingly used as they often contain many different fibres. For this reason, they may hold some of the benefits of co-administration, have synergistic effects and offer diverse functional characteristics that may offer therapeutic potential in the management of a range of gastrointestinal disorders. Examples of novel plant-based fibres include prickly pear, galactomannan, plantain peel, ivy gourd, Gnetum africanum, yacon root, Moringa oleifera, which have unique combinations of different fibres and rich source of other bioactive compounds (e.g., polyphenols) that have potential anti-inflammatory and anti-bacterial properties. However, there are limited studies of natural fibres in the management of gastrointestinal disorders.

**Conclusion**

Manipulation and/or increasing fibre intake is a promising therapeutic strategy in the prevention and management of many gastrointestinal disorders. Physico-chemical characteristics such as solubility, viscosity and fermentability drive different functionalities in the gastrointestinal tract, and thus underpin their therapeutic potential. Current guidelines and recommendations reflect earlier studies that have used a wide range of dietary fibres with different physico-chemical and functional characteristics. The lack of consistency and reporting of these characteristics in studies to date has limited the clinical utility of dietary fibre for managing gastrointestinal disorders. There is an urgent need for well-designed, placebo-controlled clinical trials to determine which physico-chemical characteristics and therefore which fibre sources and in what dose and duration is optimal for clinically meaningful gastrointestinal health benefits. The utility of co-administration of different fibres with differing physiological effects, or novel, naturally-occurring dietary fibres with duel physiological properties has yet to be explored and holds promise as a therapeutic strategy across several gastrointestinal disorders.

**Table 1: Physico-chemical characteristics of common dietary fibres**

|  |  |  |  |  |
| --- | --- | --- | --- | --- |
| **Fibre Type** | **Common Sources** | **Physico-chemical characteristic\*** | | |
|  |  | **Solubility** | **Viscosity** | **Fermentability** |
| Cellulose | All green plant cell walls | Insoluble | Non-viscous | Low |
| Lignins | All green plant cell walls | Insoluble | Non-viscous | Low |
| Arabinoxylans | Wheat, psyllium\*\* | Low to Medium | Medium | High\*\* |
| B-glucans | Oat, barley, fungi | Low to Medium | Medium to High | High |
| Galactomannans | Guar gum, fenugreek | Medium to High | Medium to High | High |
| Pectins | Fruits, vegetables, legumes | High | Medium to High | High |
| Inulin | Cereals, fruits, vegetables | High | Low to High | High |
| Galacto-oligosaccharides | Pulses (e.g. beans, peas, lentils) | High | Low | High |
| Dextrins | Cereals (e.g. wheat dextrins) | High | Non-viscous to low | High |
| Alginate | Seaweed | High | High | Low |
| Methylcellulose | Synthesized | High | High | Non-fermentable |
| Resistant Starch |  |  |  |  |
| RS 1 (physically inaccessible) | Whole grains, legumes, raw fruits, vegetables | Insoluble | Non-viscous | High |
| RS 2 (starch conformation) | Cereals, raw legumes, raw fruits, vegetables | Low | Non-viscous | High |
| RS 3 (retrograded) | Cooking and cooling of any starch source | Low | Non-viscous to low | High |
| RS 4 (chemically modified) | Synthesized (e.g. acylated starches) | Low to High | Low to Medium | High |
| RS 5 (starch-lipid complex) | Synthesized (e.g. amylose and stearic acid) | Low | Low | Low |

\*Physico-chemical characteristics are not distinct entities but represent a continuum or gradient, and will vary depending on botanical origin, chemical structure and molecular weight, or whether isolated fibres rather than as part of the cell wall matrix (Figure 1).

\*\* Due to its structural features, wheat and psyllium sources of arabinoxylans are considered of only low fermentability.

**Table 2: Guidelines and recommendations for the use of fibre in gastrointestinal disorders**

|  |  |  |
| --- | --- | --- |
| **Gastrointestinal disorder** | **Dietary fibre recommendations** | **Level of evidence** |
| IBS-C | Soluble fibre in supplement form (e.g. psyllium, methylcellulose, partially hydrolysed guar gum) 8,12,135,136,138 | Meta-analyses of RCTs |
|  | Adjust fibre intake according to symptoms including adding naturally occurring sources (e.g. oats or linseeds)8 | Professional consensus |
|  | Ground linseeds (6–24 g/day). Increase gradually over a 3-month period9 | Small number of RCTs |
| IBS-D | Reduce intake of insoluble fibre, such as wholemeal or high-fibre flour and breads, cereals high in bran, and whole grains such as brown rice8 | Systematic review of RCTs |
|  | Soluble fibre in supplement form (e.g. psyllium)135,138,189 | Meta-analyses of RCTs |
|  | Adjust fibre intake according to symptoms8 | Professional consensus |
| Inflammatory bowel disease | Encourage a varied diet to meet energy and nutrient requirements, including dietary fibre, including a wide variety of fruit and vegetables, cereals, grains, nuts and seeds10 | Professional consensus |
|  | Consider limiting fibre and fibrous foods in patients with strictures12 | Professional consensus |
|  | Dietary fibre should not be restricted, unless intestinal obstruction12 | Professional consensus |
|  | Ulcerative colitis may be more amenable to fibre supplement interventions than Crohn’s disease151 | Systematic review of RCTs |
| Diverticular disease | Low fibre diet during active diverticulitis to ‘minimise irritation’12 | Professional consensus |
|  | High fibre diet from mixed sources to prevent diverticulitis. | Case-control studies and professional consensus |
| Functional constipation | Encourage gradual increases (weeks rather than days) in fibre (or by adding fibre supplements) to minimise gastrointestinal discomfort including bloating and flatulence aiming for 20-30 g/day, being aware that beneficial effects may be seen after several weeks12,13 | Meta-analyses of RCTs |
|  | Encourage whole fibre foods with additional components with laxating effects (e.g. sorbitol) such as prunes or apricots13 | Small number of RCTs and professional consensus |
|  | High dose psyllium (>15g/day)112 | Meta-analysis of RCTs |

Guidelines are based upon research and professional consensus, but frequently the evidence is not from high quality clinical trials.

**Figure titles**

**Figure 1 Physico-chemical characteristics of dietary fibre and their location within the plant cell**

**Figure 2 Mechanisms by which different dietary fibres impact the gastrointestinal tract**

**Figure 3 Spectrum of physico-chemical characteristics of dietary fibre**

The physicochemical characteristics of fibre solubility, viscosity and fermentability exist along a continuum and work in concert to determine its functional properties in the gastrointestinal tract. The combination of these three physico-chemical characteristics determine the functional effects of fibres in the gut. For example: fibres in the left-hand, bottom, near corner (insoluble, non-viscous, non-fermentable) have functions relating to gut transit time; fibres in the right-hand, bottom, far corner (soluble, non-viscous, fermentable) have functions relating to microbiome and fermentation; and fibres in the right-hand, top, far corner (soluble, viscous, fermentable) have functions relating to microbiome, fermentation and nutrient bioavailability. Fibres in intermediate positions would be predicted to have intermediate functional properties.

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