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The interconnection between dietary fibre, gut microbiome and psychological well-being

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Abstract

This review highlights the importance of dietary fibres (DF) intake and its interconnection with the gut microbiome and psychological well-being, while also exploring the effects of existing DF interventions on these aspects in adults. The gut microbiota is a complex and diverse ecosystem in which microbial species interact, influencing the human host. DF are heterogeneous, requiring different microbial species to degrade the complex DF structures. Emerging evidence suggests that microbial fermentation of DF produces short-chain fatty acids (SCFA), which may play a role in regulating psychological well-being by affecting neurotransmitter levels, including serotonin. The effectiveness of DF interventions depends on factors such as baseline gut microbiota composition, the dosage and the source of DF consumed. Although the gut microbiota of adults is relatively stable, studies have shown that the abundance of the species in the gut microbiota can change within 24 h of an intervention and may return to baseline following the termination of DF intervention. This review underscores the need for larger and well-powered dietary clinical trials incorporating longitudinal biological sample collections, advanced sequencing and omic techniques (including novel dietary biomarkers and microbial metabolites), validated subjective questionnaires and dietary records. Furthermore, mechanistic studies driven by clinical observations are crucial to understanding gut microbiota function and its underlying biological pathways, informing targeted dietary interventions.

The human gut is home to trillions of microorganisms. These microbes, collectively known as 'gut microbiota', are composed of eukarya (0·01 %), protozoa (0·01 %), fungi (0·1 %), archaea (0·1 %), viruses (6 %) and bacteria (93 %)[1–3]. The gut microbiota is vastly diverse, where the number of microbes is 10 times higher than germline and somatic cells of the human body[2,4–6]. Further, the number of microbial genes (gut microbiome) is over a hundredfold more than the human genes[2,4,5]. It has also been estimated that a normal healthy gut harbours over 1000 microbial species, with at least 160 microbial species shared among individuals[2,6].

The metabolic interactions between the gut microbiota and host involve complex interactive processes[7]. The gut microbiota undertakes anaerobic fermentation activities to produce a wide range of metabolites, including short-chain fatty acids (SCFA), branched-chain fatty acids, neurotransmitters and others[8,9]. Each metabolite is an information messenger between microbes and host cells that can affect these interactions[8,9].

Furthermore, diet, particularly dietary fibre (DF) intake, is a modifiable lifestyle factor associated with the modulation of the gut microbiota composition and function[10–12]. Intake of fermentable DF may diversify the gut microbiota community, subsequently increasing the production of SCFA[13]. SCFA are considered neuroactive bacterial metabolites of DF degradation and fermentation[14] and also regulate systemic inflammation and oxidative stress in the gut[15]. Several reviews show that SCFA can affect other neuroactive metabolites[16], including serotonin[17]. Therefore, the interactions between DF intake and the gut microbiota are likely crucial for the psychological well-being of the human host[18].

The current understanding of how different types of DF affect the human body has been gleaned from studies on prebiotics, whole food or isolated fibre interventions[10]. While several reviews have established the protective role of DF against chronic diseases and gut dysbiosis[19–22], critical gaps remain. Most studies focus on broad DF categories rather than the distinct effects of specific fibre types on gut microbiota composition, function and metabolite production. Moreover, the interconnection among DF, the gut microbiome and psychological well-being remains largely underexplored. Given that microbial and metabolic responses to DF are dictated by its chemical structure[23,24], this review addresses the following key questions: (1) How do different DF types impact gut microbiota composition, metabolic activity and psychological well-being? (2) What gaps remain in the current understanding and what future research directions are needed to further investigate these relationships? By addressing these questions,

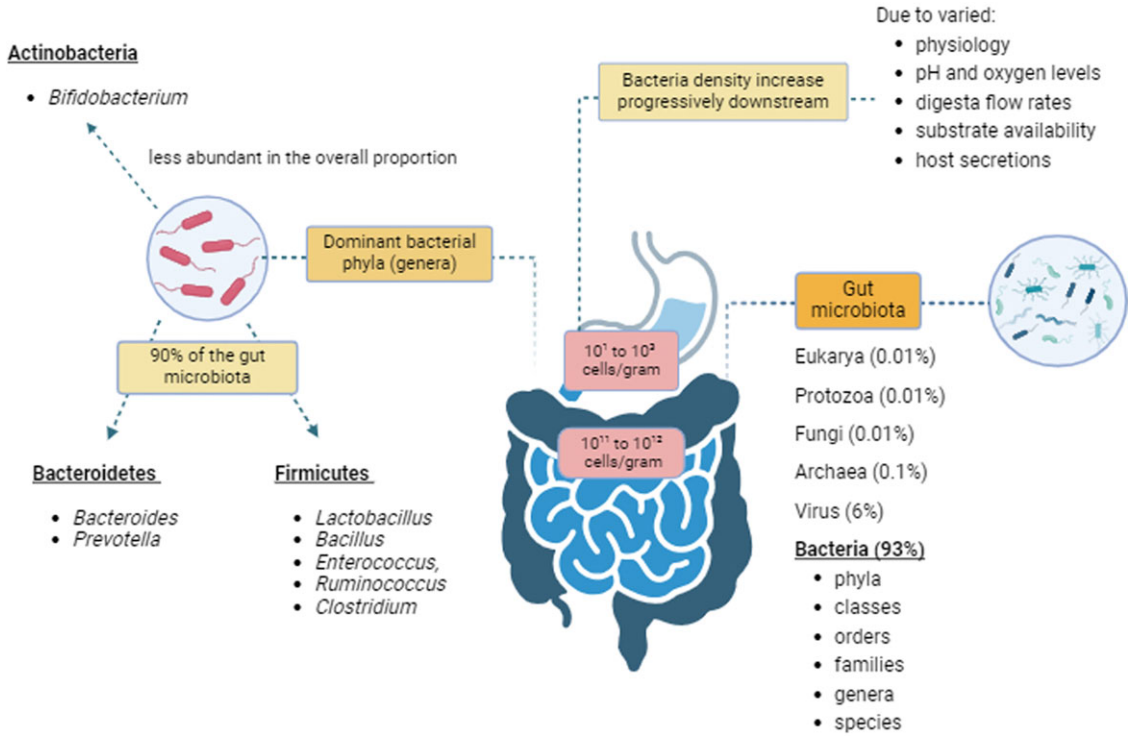

Figure 1. Gut microbiota composition. Created with *BioRender.com.*

this review aims to provide a more nuanced understanding of how specific fibre types influence the gut microbiome and psychological well-being in adults.

Gut microbiota composition

Gut microbes are classified according to their taxonomy: phyla, classes, orders, families, genera and species[25]. Gut microbial density increases progressively downstream from the stomach and duodenum (ranging from 10^1 to 10^3 cells per gram) to the large intestine (10^{11} to 10^{12} cells per gram)[26]. The gut microbiota differs across different regions of the intestine due to variations in physiology, pH, oxygen levels and digesta flow rates, substrate availability and host secretions[27]. The small intestine has relatively short transit times (three to five hours) and high bile concentrations. In contrast, the large intestine has slow flow rates, a neutral to mildly acidic pH and a higher concentration of obligate anaerobic microbes[4,5,27–29]. The two most abundant gut microbial phyla, which represent 90 % of the gut microbiota, belong to Bacillota (formerly Firmicutes) (*Lactobacillus, Bacillus, Enterococcus, Ruminococcus* and *Clostridium* genera) and Bacteroidota (formerly Bacteroidetes) (*Bacteroides and Prevotella* genera)[4,5,28,29]. The phylum Actinomycetota (formerly Actinobacteria) is less abundant in the overall proportion of the gut microbiota but mainly consists of the *Bifidobacterium* genus[4,5,28,29] (Figure 1). For this manuscript, former taxonomic names, Firmicutes, Bacteroidetes and Actinobacteria will be used.

What is a healthy gut microbiota

Due to the high degree of inter-individual variability[30], there is no consensus on the definition of a 'healthy gut microbiota'. Nonetheless, it can be described as a state in which the gut microbiota exhibits diversity, stability and resilience to potential gut disturbances[9,31] or achieves a state of homeostasis[32].

Gut homeostasis is a complex interconnection involving the gut microbiota, specialised epithelial cells and the host immune system[33]. These specialised epithelial cells are part of the gut barrier and regulate cross-communication between commensal microbial communities and mucosal immune cells[34]. Consequently, this helps protect the gut against pathogens[34]. The state of homeostasis occurs when there is neither an overgrowth of pathogenic microbes nor a loss of beneficial gut microbes and diversity[32]. The most well-known diversity indices are alpha (richness and evenness of gut microbial species within an individual) and beta diversity (similarity and differences of microbial community between individuals)[35–37]. Nourishing the gut microbiota by consuming a diverse diet containing substrates such as DF may help enhance gut microbiota diversity, stability and resilience[9,31] and/or homeostasis[32], promoting a healthier gut microbiota.

Dysbiosis, on the other hand, occurs when gut homeostasis is disrupted[32]. A dysbiotic microbiota is often related to a loss of microbial diversity[38], which can compromise the functional resilience of the gut microbiota. This subsequently will increase susceptibility to dysbiosis-related diseases, including irritable bowel syndrome (IBS)[33] and IBS-associated psychological issues, including anxiety and depression[39]. Emerging evidence suggests that disruptions in microbial functions, called the 'functional core', may be more critical than microbial composition changes in establishing a healthy gut microbiota and psychological health[40,41].

Determining microbial functions is achieved using -omic approaches[42]. For instance, metagenomics characterises the various gut microbial species and their gene abundances depending on the depth of sequencing[7]; metatranscriptomics allows the

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comparison of the microbial gene expression profile among individuals[42,43]; metabolomics and proteomics provide insights into the functional relationship between the gut microbiota and host[7,43] using mass spectrometry (MS) instruments[7,44]. These -omic approaches help improve our understanding of the complexity of the gut microbiota and its role in human health and diseases.

Definition of dietary fibre

Diet, particularly DF intake, is a modifiable lifestyle factor associated with modulating the gut microbiota composition and function[10–12]. The ability of the gut microbes to metabolise DF depends on the chemical structure, including chain length and branching of DF[45].

Defining DF has been controversial for multiple reasons. Firstly, DF are a collection of related chemical compounds[46]. The physicochemical mechanisms that provide health benefits are not fully elucidated. Secondly, whether and how DF affects peristaltic gut movement and microbial fermentation is unclear[47]. Thirdly, compounds embedded in the DF matrix, such as antioxidants and polyphenols, may produce different physical, chemical and physiological effects in the gut[48]. It is also unclear if these effects are contributed by DF *per se* or occur only within the food matrix[46]. In a food industry context, DF are further characterised based on physiological effects[46,49]. These complexities around defining DF have led to separate organisations proposing their own definitions.

Despite DF complexities, a uniform and accurate definition is warranted. In 2009, the CODEX Alimentarius Commission proposed a definition and classified DF into three distinct categories[50]. The first category covers DF derived from naturally occurring foods as part of a healthy diet. The second and third categories include extracted DF and synthetic carbohydrate polymers, which have demonstrated beneficial health effects[50]. These effects may include increased gut transit time and faecal bulk, colonic fermentation and modulation of blood glucose and cholesterol levels[46]. The CODEX definition allows officials worldwide to decide whether to include oligosaccharides and/or carbohydrates of three to nine monomeric units within the DF definition[50,51]. While no uniform definition is used worldwide, CODEX definition adaptation could be an initial step towards achieving global DF definition consensus.

Prebiotics

Most prebiotics are DF, but not all DF are prebiotics[45]. Prebiotics are usually carbohydrates, and only a few functional carbohydrates have been accepted as prebiotics. These include inulin-type fructans, fructo-oligosaccharides (FOS)[20,52,53] and galacto-oligosaccharides (GOS)[54,55] that effectively stimulate the growth of species from the *Bifidobacterium*[56,57] and *Lactobacillus* genera[58]. Resistant starch (RS)[59,60] and arabinoxylans[59,61,62] also have prebiotic effects[59,61,62]. However, most experimental methods and mechanisms of RS and arabinoxylans have been investigated either *in vitro*[63] or in animal models, which require further exploration to confirm their role as a prebiotic for improving human health.

The current concept of prebiotics has been criticised as ill-defined and in need of revision[64]. Initially, prebiotics were described as 'selective' and 'specific' toward beneficial gut microbial groups, which shifts the gut microbiome to a 'healthier' state[64]. However, this concept lacks clarity, as many dietary compounds could meet these criteria. Moreover, gut microbial species can share degradation features via horizontal gene transfer, allowing a broad range of species with the necessary degrading enzymes to degrade DF[65]. Compounds such as polyphenols and polyunsaturated fatty acids with evident therapeutic effects may also qualify as prebiotics[53]. This further challenges the notion of selectivity and identification of beneficial microbes within the definition[64].

Categorising gut microbes as beneficial or non-beneficial may be oversimplified[64]. Different gut microbes can be both beneficial and detrimental to the human host depending on environmental factors such as diet, gut microbiota or host genetic predisposition[64]. Prebiotic research often focused on bifidobacteria and lactobacilli, as these genera are widely known for their beneficial role[64]. However, other genera, such as *Faecalibacterium*[54], *Anaerostipes* and *Bilophila*[55], as well as the previously considered harmful genera *Clostridia* and *Bacteroide*[66,67], may also be beneficial to the human host.

Microbiota accessible carbohydrates

The inter-individual variation of gut microbiota makes defining DF and prebiotics more complex. Gut microbiota composition varies between individuals and populations of different physical and health statuses and lifestyles[59]. These may impact the degree of metabolism and health effects of DF on the human body. A new term, microbiota-accessible carbohydrates (MAC), was coined to address these challenges. This term classifies carbohydrates into dietary (prebiotics and DF) and host-derived MAC (mucosal glycans)[68]. MAC does not include non-fermentable DF and depends on the presence of gut microbial species in the gut to metabolise the different types of DF[69,70]. For example, individuals who possess the important gut microbial species *Rumonococcus bromii* can metabolise Resistant Starch (RS) type 3[70]. Therefore, RS type 3 would be considered a MAC for these individuals[70]. Additionally, if a MAC provides health benefits to the human host, it would also be a prebiotic[71]. This concept helps contextualise how different DF types interact with the gut microbiota, setting the stage for a deeper discussion on their metabolic activities and implications for host health.

Metabolic activities of the gut microbiota

This section describes how DF metabolism can influence the relationship between the gut microbiota and the human host. The effectiveness of the existing DF interventions on the gut microbiota and psychological well-being is subsequently reviewed.

DF are usually favoured over other nutrients as a substrate for microbial anaerobic fermentation[8,13]. Gut microbes can undertake two types of fermentation: saccharolytic and proteolytic. Saccharolytic fermentation mainly occurs in the proximal colon[13,72]. This part of the colon is more acidic (pH 5·5–6·5) than the distal colon (pH 6·5–7·0)[73] and has a greater availability of highly fermentable DF, such as inulin, which produces SCFA[13,72]. Saccharolytic fermentation increases faecal biomass, bulk, weight and frequency[13,72] and are considered beneficial to a certain extent[8]. Proteolytic fermentation, on the other hand, occurs in the distal colon, where there is are lower amount of fermentable DF[13,74]. In this process, the high amount of undigested dietary protein is broken down by proteolytic bacteria and subsequently used in proteolytic fermentation[13,74]. This fermentation process potentially results in toxic compounds, such as branched-chain fatty acids, ammonia, amines, phenols, thiols and indoles[13,74] (Figure 2). Therefore, consuming a wide range of

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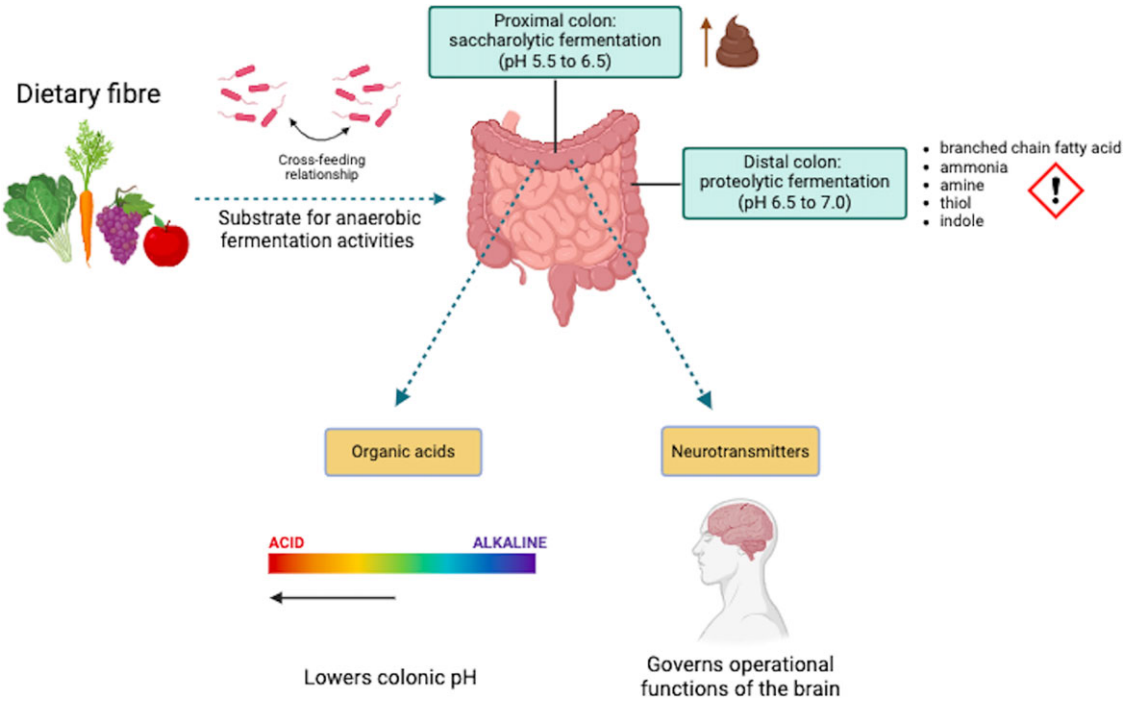

Figure 2. Metabolic activities of the gut microbiota. Created with *BioRender.com.*

DF will likely benefit the human host as it promotes saccharolytic fermentation and lowers proteolytic fermentation[72].

The degradation of DF by the gut microbiota provides substrates supporting a symbiotic relationship with the human host and other microbes[9,45]. Gut microbial species utilise energy from the degradation process for survival, growth, reproduction, provision and maintenance of cellular functions for the human host[14,75]. Highly specialised microbial species that degrade DF directly are referred to as primary degraders or keystone species[64,76]. Degradation of DF by these keystone species results in partial breakdown products, including SCFA, which can lower the colonic pH[45,77]. This acidic condition can benefit the primary degraders and potentially other microbial species. However, it may also inhibit the growth of another microbial species[45,77]. For example, the lower colonic pH promotes butyrate producers that thrive under acidic conditions and reduces acid-sensitive species such as members of the *Bacteroides* genus[45]. Additionally, SCFA produced during DF degradation can affect neurotransmitter levels[16,78], such as serotonin, highlighting the link between gut microbial activity and brain function.

Further, secondary degraders taking up metabolites released by primary degraders[79] is generally termed 'cross-feeding'[80]. This process can occur within the same species, between different species or in a complex system where species depend on each other[81,82]. A cross-feeding relationship can be observed in a co-culture experiment where *Ruminococcus bromii*, a primary degrader, metabolises RS type 2 and RS type 3, which subsequently stimulates metabolite utilisation by species *Eubacterium rectale*, *Bacteroides thetaiotaomicron* and *Bifidobacterium adolescentis*[70]. Therefore, DF degradation products are part of a complex web of interactions that influences the symbiotic relationship between the gut microbiota and the human host highlighting the crucial role of this ecosystem in human health.

Organic acids

The degradation of DF by microbial species produces organic acids[45,77]. Among the organic acids are SCFA that are saturated fatty acids containing aliphatic carboxylic acid tails of up to six carbon atoms[83]. In ascending order of carbon atoms, the six SCFA are formate, acetate, propionate, butyrate, valerate and caproate[83]. Acetate, propionate and butyrate are the primary SCFA as they are produced at a higher rate than other SCFA[8,22,84].

SCFA are often measured from faecal or plasma samples[85]. However, their levels may vary depending on the production and absorption into gut epithelial cells. Acetate concentrations then propionate and butyrate concentrations are considered more prevalent, as they are utilised by colonocytes[9,22,86], taken up by hepatocytes[9,22,87], enter peripheral circulation[9]. Therefore, it has been suggested that the concentrations of SCFA in faecal and plasma samples might not accurately represent their *in vivo* production[88].

Recent studies found that colonic transit time affects SCFA production[89]. A longer transit time in the descending colon was associated with lower plasma acetate concentrations but not butyrate or propionate concentrations[89]. More SCFA are released from the distal colon into the circulation compared to the proximal colon[88], potentially due to greater proximal gut mucosal metabolism[90]. Additional factors include variations in SCFA production by the microbiota between proximal and distal colon[1] and the differences in apical and basolateral sides of epithelial cells uptake and transport across gut segments[91]. Therefore, faecal

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SCFA levels are suggested to primarily reflect production and/or absorption in the distal colon rather than the proximal colon[85]. Nonetheless, the production and absorption of SCFA are dynamic. These processes can change based on factors, including the consumption of different types and doses of DF[84] and the presence of specific microbial species capable of metabolising the DF[13].

Neurotransmitters

The gut microbiota also produces neuroactive metabolites, including neurotransmitters such as serotonin, which influence gut motility, secretion and neurological functions related to behaviour and mood[78,92–96]. Serotonin is primarily produced by enterochromaffin cells in the gut[78], with tryptophan, a dietary amino acid, as a precursor[97]. Tryptophan metabolism and serotonin production are also modulated by specific commensal microbes[97] that degrade tryptophan or convert it into serotonin via tryptophan synthetase enzyme[98]. These include genera such as *Clostridium, Ruminococcus, Blautia*, *Lactobacillus*[99], *Lactococcus, Streptococcus, Klebsiella* and species like *Escherichia coli*[98,100].

Dietary fibre may influence serotonin pathways indirectly by shaping the composition and metabolic activity of these microbes. Microbial degradation of DF produces SCFA, which help maintain gut barrier integrity and modulate immune responses[45,77], thereby influencing tryptophan metabolism and serotonin production[17,101]. Low DF intake and the resulting dysbiosis have been linked to disrupted serotonin signalling, especially in disorders of gut-brain interaction[102–104] such as IBS[33,78], which frequently co-occur with anxiety and depression[39]. For instance, mice lacking serotonin reuptake transporter in the gut mucosal cells exhibit alternating bouts of diarrhoea and constipation[103]. In humans, reduced expression of this transporter has been reported in individuals with IBS or inflammatory bowel disease[105], potentially leading to increased mucosal serotonin exposure and desensitisation of serotonin receptors[105]. Subsequently, these reduce reflex activity, luminal secretion and gut motility[105]. In the brain, dysfunction of the serotonin reuptake transporter is associated with mood disorders[106], reinforcing the gut-brain interaction influenced by DF-microbiota interactions.

Moreover, SCFA can affect neurotransmitter levels[16,78], including serotonin[17,101]. For instance, propionic acid and butyric acid help regulate host gut cell gene expression[107], which can have downstream effects on neurotransmitter synthesis. However, it remains a challenge to understand whether these changes in SCFA levels are directly associated with the development of diseases or are a consequence of disease-related changes in the gut microbiota[108] or whether they are primarily diet-dependent[7].

Together, these findings suggest that DF can modulate neurotransmitter signalling via gut microbiota mediated pathways. This highlights the potential of DF-based strategies to modulate the gut microbiome and psychological well-being.

Impact of dietary fibre interventions on the gut microbiome

Different types of DF induce varied changes in the gut microbiome, whether post-intervention compared to baseline and/or between intervention groups. Most studies from more than a decade ago used a metagenomic approach, while one recent study used a culture-based experiment to explore the microbial community following asafoetida-curcumin complex in turmeric capsules administration[109]. It is not surprising that this culture-dependent study only explored a small subset of bacteria strains, specifically taxa from *Bifidobacterium* and *Lactobacillus* genera, due to the limited capability of the technique to explore a broader spectrum of bacterial strains and detect fine detail changes in the gut microbiota composition[13]. Nonetheless, this study showed that a asafoetida-curcumin complex intervention increased the abundance of *Bifidobacterium* and *Lactobacillus* genera compared to the control group[109].

However, studies using metagenomics of faecal samples found varied impact of DF and prebiotics consumption. Following a mixed fibre intervention changes in gut microbiota composition occurred as rapidly as four days[110]. However, in other studies, the relative abundances of microbial species changed either after seven days of a low fermentable oligo-, di- and mono-saccharides and polyols (FODMAP) diet and oligofructose[111] or after four weeks of prebiotic oligofructose and prebiotic candidate 2'fucosyllactose[112]. An intervention with GOS also showed increased Shannon (alpha) diversity and relative abundances of *Bacteroidetes*, *Clostridia* and *Bifidobacterium* genera compared to the control group[113]. Similarly, a prebiotic mixture containing different prebiotics and DF increased the relative abundance of the Actinobacteria phylum and *Bifidobacterium* species but showed no differences in SCFA concentrations compared to the control group[114].

Similarly, other types of DF intervention led to mixed results on the gut microbiota. The consumption of 14 gram (g) resistant dextrin for four weeks did not affect the gut microbiota composition and predictive function[115]. However, this result was not seen in studies with resistant maltodextrin. A three-week intake of 15 g or 25 g of resistant maltodextrin increased the relative abundance of *Fusicatenibacter saccharivoran*[116], but only 25 g of resistant maltodextrin increased *Bifidobacterium* counts after four weeks[117] and increased that of species *Akkermansia muciniphila* and *Faecalibacterium prausnitzii* after three weeks[116]. Further, a three-week intervention with 4·5 g chitin-glucan changed several microbial species abundances[118,119] and faecal SCFA concentrations, particularly butyric and caproic acids[119]. A 24-week rice bran intake increased the relative abundances of Firmicutes phylum and *Lactobacillus* genus compared to rice powder intake[120]. Similarly, there were changes in gut microbiota composition after the consumption of broccoli and daikon radish for 18 d[121], of crackers containing RS for 10 d[122], bread and biscuits containing refined or wholemeal amylose wheat for four weeks[123], a snack bar containing 7 g of chicory inulin-type fructans daily for four weeks[123] and biscuits enriched with olive pomace for eight weeks compared to control group[124]. These findings overall highlight the diverse and duration-dependent effects of different types of DF interventions on the gut microbiota.

In terms of gut microbiota diversity, there was no difference in the reviewed studies. This includes a four-week study providing either chitin-glucan[119], rice bran[120], mixed prebiotics[114], fruit pomace[125] or polydextrose[126], a two-week study using potato RS, maize RS or chicory root inulin[127], two-week of a whole grain diet[128], an eight-week intervention of olive pomace enriched biscuits[124], a 10-week high DF diet[129] or 18 d of cooked broccoli and daikon radish[121]. Interestingly, a diet containing six serves or more of fermented foods increased alpha diversity, but not a diet containing 20 g or more fibre[129]. The alpha diversity was also lower following a 12-week wheat bran-derived arabinoxylan oligosaccharides intervention, possibly resulting from softer stools, selective stimulation and growth of the *Bifidobacterium* genus[130].

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Therefore, this alpha diversity reduction might not correlate with gut microbiota instability[130].

Overall, the reviewed studies that explored the effect of DF on the gut microbiome showed mixed results. Different study durations and washout periods, high inter-individual variability and limited research examining different DF types examined might have been contributing factors. Comparing these studies directly is challenging, which warrants better designed clinical trials. Future studies on DF should consider baseline dietary intake and individual gut microbiota composition prior to intervention. The gut microbiota of individuals with low DF intake and limited baseline microbial diversity may respond more to the intervention as compared to their counterparts[131]. Therefore, a personalised nutrition approach may be more effective, allowing improved adherence to the dietary intervention.

Impact of dietary fibre interventions on psychological well-being

A handful of studies have explored the effects of DF on psychological well-being in healthy adults. These studies used either resistant dextrin[115], chitin-glucan[119], wheat bran-derived arabinoxylan oligosaccharides[89], GOS[113], polydextrose[126], snack bar containing chicory inulin-type fructans[132] or a low FODMAP diet supplemented with prebiotics[133]. These studies found that the interventions did not affect well-being, quality of life or mood. However, GOS supplementation tended to decrease anxiety symptoms in participants with anxiety[113]. These findings may be influenced by a ceiling effect, where individuals with already high well-being levels have limited potential for further improvement. This suggests that the effectiveness of these interventions may depend on participant selection criteria, with greater effects potentially observable in those with lower baseline well-being[134,135].

Further, co-administration of a prebiotic consisting of oligofructose and 2'fucosyllactose, demonstrated a decrease in depression, anxiety and negative affect schedule scores in a double-blind, placebo-controlled, randomised controlled trial[112]. Despite research on co-administering DF or prebiotics is limited, combining isolated fibres might offer a 'dual treatment' for gut-related and psychological outcomes[20]. This was shown where the co-administration of isolated oligofructose and 2'fuscosyllactose increased the relative abundances of several beneficial butyrate-producing microbes, including taxa from the *Lactobacillus* and *Blautia* genera, after four weeks of intervention[112]. The authors observed several positive correlations between several mental health scores improvement and presence of the genera *Bifidobacterium*, *Roseburia*, *Anaerostipes*, *Blautia* and the species *Faecalibacterium prausnitzii* in in their cohort[112]. The authors speculated that gut microbiota manipulation may influence mental health by regulating neurological pathways[16,112]. This can be explained by the ability of some DF types to promote microbes involved in serotonin precursors production or tryptophan metabolism, linking gut microbial activity to serotonin signalling and mood regulation[17,45,77,97–101]. Nonetheless, as these findings are based on associations rather than mechanistic evidence, further research is needed to establish causal pathways.

There is no conclusive evidence on how specific DF types or food items modulate the gut microbiota to improve psychological well-being. However, some prebiotic fibres, such as GOS, oligofructose and 2'fucosyllactose have shown promise in modulating microbial composition and mood-related outcomes, particularly among individuals with lower baseline well-being. Therefore, consuming a variety of DF-rich foods, including those high in prebiotic fibres may help support a beneficial gut microbiome and potentially improve psychological well-being.

Future directions

Determining the most suitable food or dietary pattern is crucial to help manage specific diseases or disorders. Furthermore, this may also help in designing personalised nutrition strategies to alter the gut microbiota and optimise the health and metabolism of humans[136]. However, several reviews and intervention studies have suggested that factors, including season, age[137], both baseline gut microbiota and well-being as well as habitual dietary patterns can influence gut microbiota composition and overall response to a dietary intervention[138,139].

Baseline microbial richness, diversity and stability might be able to predict dietary interventions' effectiveness[140–142]. A systematic review proposed that individuals with low DF intake and limited baseline microbial diversity may show more gut microbiome changes following intervention[131], particularly after acute dietary interventions[143]. However, taxonomy changes may be relatively temporary, subtle and inconsistent compared to the effects of habitual, sustained diet[11]. The authors of a review further argued that a stable gut microbiota may suggest a stable response or that there may not be a change to the diet[142]. An unstable gut microbiota may indicate a flexible response to the 'optimal diet'. This may require constant re-evaluation of the diet[142], which can be challenging to determine the effectiveness of the dietary intervention.

The taxonomic composition of the adult gut microbiota is relatively stable over extended periods[11]. The abundance of individual taxa may be susceptible to alterations in dietary patterns and geographic area[10,144]. With the diverse dietary and lifestyle choices available, there is a tendency for each individual to have a highly individualised gut microbiome[145]. Large phylum-level adjustments in response to dietary changes may be more pronounced in animal models involving rodents or pigs due to the highly controlled study environment[11]. To further substantiate this, a one-year longitudinal study of two individuals showed that 75 % to 88 % of bacteria were relatively stable for several months. However, relative abundances of specific taxa, including *E. rectale*, *F. prausnitzii, Eggerthella, Clostridium, Ruminococcus, Blautia* and *Bifidobacteriales* shifted within a day following a change in geographical and DF intake in one of the participants[144]. Following travel for 51 d, the gut microbiota of this participant returned to its pre-travel state within 14 d. The authors suggested that this reversal was partly due to temporarily adopting the local diet abroad[144]. Nonetheless, the range of behavioural choices was limited to these two individuals and the shift in gut microbiota was observed in only one participant[144]. In this context, migration and intervention studies will be particularly valuable in understanding the long-term impact of dietary and environmental changes on gut microbial composition, warranting further clinical research in this area.

The addition of certain types of DF may lead to unwanted negative gut symptoms. Tolerances between individuals vary, but a sudden increase or a drastic change in DF intake may result in bloating, discomfort and increased flatulence[146]. These potential

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adverse effects should also be considered in future studies to ensure both efficacy and tolerability of DF interventions.

Conclusion

Taken together, evidence remains inconclusive on how habitual intake of DF supplementation through whole foods influence the gut microbiome and psychological well-being in adults. Key knowledge gaps include the effects of specific DF types, inter-individual variability and the impact of habitual *v*. acute intake. Baseline microbial composition, dietary patterns and psychological status may influence dietary intervention outcomes, highlighting the potential for stratified or personalised approaches. To address this, there is a pressing need for larger and well-powered clinical trials that incorporate longitudinal biological sample collections, advanced sequencing techniques and other -omics techniques (including novel dietary biomarkers and microbial metabolites), validated subjective questionnaires and comprehensive dietary records. Integrating these data can enhance understanding of the gut microbiome, while mechanistic studies driven by clinical observations are essential for identifying underlying biological pathways. These insights may ultimately support the development of targeted interventions that can be applied to the general public and clinical practices.

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