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Diet-Gut Microbiota Interactions in Metabolic and Neuroinflammatory Diseases

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ABSTRACT

This study looks at how eating habits, gut microbes and the body work especially when it comes to metabolic and brain inflammation problems. Nutritional foods change the diversity of gut microbes and the ways they work while gut byproducts such as short chain fatty acids, bile acid derivatives, tryptophan metabolites and trimethylamine related compounds affect the lining of the gut hormone cells, immune cells and brain cells. Human studies and animal experiments link problems in gut microbes. Altered microbial work, to obesity, insulin resistance, type 2 diabetes, fatty liver disease, heart risks and brain inflammation conditions. The gut–brain link sets up two way communication through nerves the system, hormones and metabolism. The study reviews diet changes, prebiotics, probiotics, postbiotics and other gut microbe tactics while also talking about the parts of proving cause and effect making standards and using these tactics in real health care. Overall, the evidence supports getting food quality and more diverse gut microbial work and it warns against simple claims that say one certain bacterial species is the key. Future research should look at clinical trials custom nutrition plans and regular ways of analyzing data to find out cause and effect. The goal is to check if the treatments that affect the microbiota are safe work well and matter in life for different groups of people.

Keywords: *Gut microbiota; Metabolic disease; Neuroinflammation; Diet; Gut–brain axis; Nutrition; Short-chain fatty acids; Microbial metabolites; Personalized nutrition; Dysbiosis*

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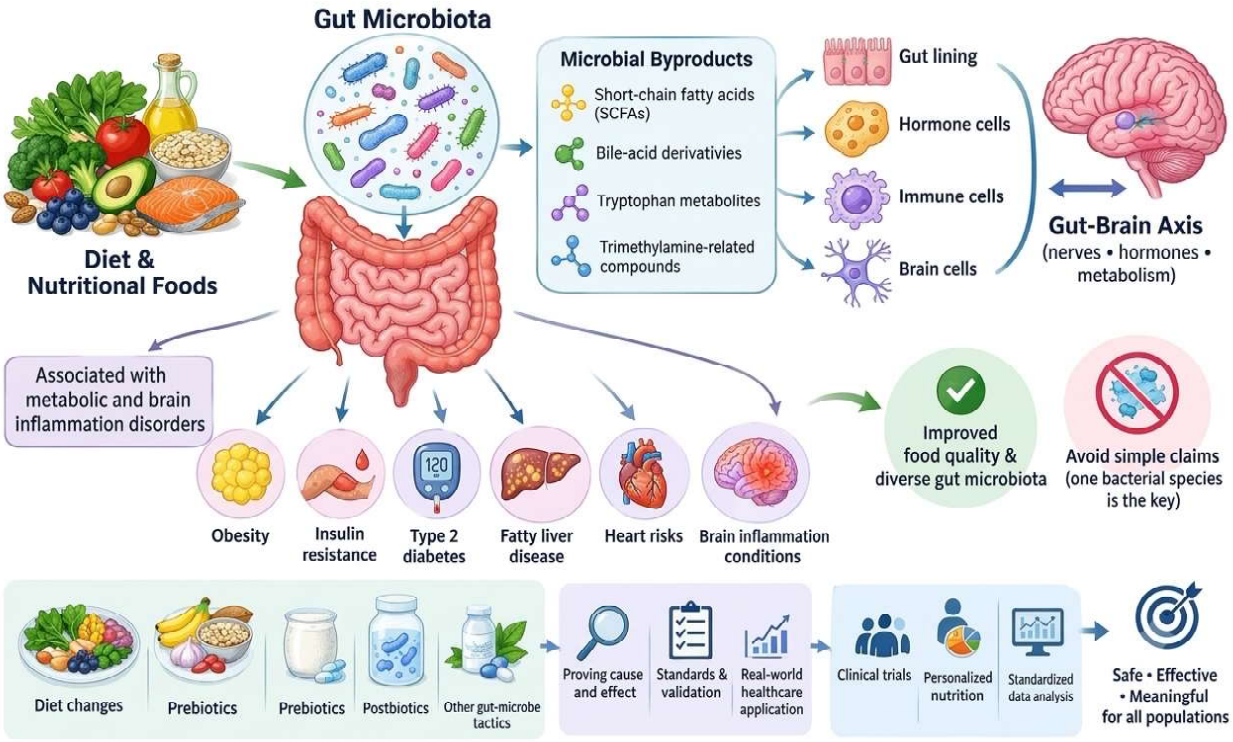

Graphical Abstract

INTRODUCTION

The human digestive system houses a living community of organisms. These tiny creatures help with breaking down food building the system keeping the inside of the stomach healthy sending signals through the body and helping with how the body uses energy (Asnicar et al., 2021). Many people use the words gut flora and gut microbiota as if they mean the thing. Now we know more. It's not about the kind of bacteria that live there. The gut microbiota is like a world made up of genes, chemicals and parts that always work with the body's tissues. The Human Microbiome Project found that these communities can be very different from one part of the body to another and from one person to another (Wang et al., 2021). They often do similar jobs. This changed how we look at food. Of thinking that food directly feeds the body we now see a group of three parts working together: what we eat the activity of the microbes and the body's own way of handling it. Metabolic disorders show how closely connected the body's systems are. Things like diet, inflammation, energy balance and what the gut microbes do can affect conditions such as obesity, insulin resistance, type 2 diabetes mellitus (T2DM) metabolic dysfunction-associated fatty liver disease and heart disease (Wastyk et al., 2021). Studies in people have found that low diversity in microbial genes and certain changes in microbial functions are linked to metabolic problems. Lab experiments have also shown that substances made by gut microbes can damage the gut lining lead to metabolic endotoxaemia and cause low-grade inflammation throughout the body. Signals travel through the vagus nerve the bloodstream, immune molecules and microbial compounds (Agus et al., 2021). The microbes in the gut

can influence how microglia develop affect the blood–brain barrier change how neurotransmitters work and impact hormone signals in the brain. Because of these links, imbalances in gut microbes. Known as dysbiosis. Have been looked at in diseases like sclerosis, Parkinson's disease, Alzheimer's disease and some neurodevelopmental conditions. Still just because there's a link doesn't mean one causes the other. Most human evidence is not as strong as the data, from animal or lab studies, which often provide mechanisms (Wu et al., 2021). The gut wall is very important for controlling what moves from the gut into the blood. If this wall is damaged more exposure to bacteria parts can cause inflammation and problems with metabolism. Also the link between the gut and the brain can influence hunger, stress, thinking and the way the brain reacts to issues. These effects vary depending on a persons genes their age how they live what medicines they take and their current health. Because of this results from studies about the microbiota must be looked at carefully especially when trying to tell if something is causing a change or just happening at the time. Combining what people eat studies of the microbiome and health records can help us understand these relationships better. This review looks at what's already known about how food and the gut microbiota work together, in metabolic and neuroinflammatory diseases focusing on the ways they work what food does possible treatments and the problems of using research into real medical situations(Becher et al., 2024)

DIET–MICROBIOTA INTERACTIONS: BIOLOGICAL BASIS

The link between what you eat and the tiny living things in your gut starts with what's available for them to use. People break down starches, proteins and fats with their tools but a lot of hard-to-digest carbs, some special starches and parts of plant walls reach the lower part of the digestive system mostly unchanged. These things act as food for the microbes. Fiber in the diet affects more than just how solid your stool's. It decides which paths the microbes take to get energy and what kinds of products they make (Valles et al., 2023). When the carbs that are available get broken down they usually make three products: acetate, propionate and butyrate. The exact results depend on what kind of carbs are there how the microbes are arranged and how they help each other (Rinott et al., 2022). The world of microbes changes a lot when you are eating habits change. Experiments where people ate controlled foods have shown that changing what you eat can change the mix of microbes in just a few days. This shows that what you eat is not something that affects you over a long time. The system has parts, like competition, teamwork and sharing food (Wang et al., 2022).

Fat in the diet also changes the world indirectly by affecting bile secretion the speed of food movement through the gut and the reaction of the gut lining. Studies in animals and people have found that high fat diets change the microbes that live in the gut make the gut more leaky and increase signals of inflammation. The kind of fat can be as important as the amount of fat. Unsaturated fats and plant based diets usually come in foods that also contain fiber and polyphenols (Meyer et al., 2022). Looking at what microbes can do might tell us more, than noting if a species is present or missing. Many species can make the same good products and communities that look similar can produce metabolites. That is why

scientists now use metagenomics, transcriptomics and metabolomics together (Jang et al., 2023).

GUT MICROBIOTA AND METABOLIC HOMEOSTASIS

Studies on people and on animal models show that the types and jobs of these bacteria are connected to being overweight and type 2 diabetes. However, how strong and in what direction these links work can vary from one group to another (Chen et al., 2023). A first idea was that bacteria linked to being overweight might get energy from food that others can't break down. Experiments on mice showed that the bacteria from people can affect how much energy is absorbed and how fat builds up. Later studies found that moving bacteria from twins with different body weights could pass on some of their metabolic traits to mice that have no bacteria. These findings suggest that the bacteria play a role in experiments. They don't mean that certain types of bacteria directly cause being overweight in people. Many things affect being overweight in humans, such as what's eaten how much exercise is done how much sleep happens how hormones work, where someone lives and genes (Gong et al., 2023). A main way that bacteria connect to inflammation is through a process called metabolic endotoxaemia. In experiments eating a lot of fat can increase levels of a substance from bacteria in the blood, make inflammation worse. Changes in the bacteria may help reduce some of these effects. Pathways that involve receptors can start immune reactions leading to the release of signals and changes in how insulin works. Lasting small amounts

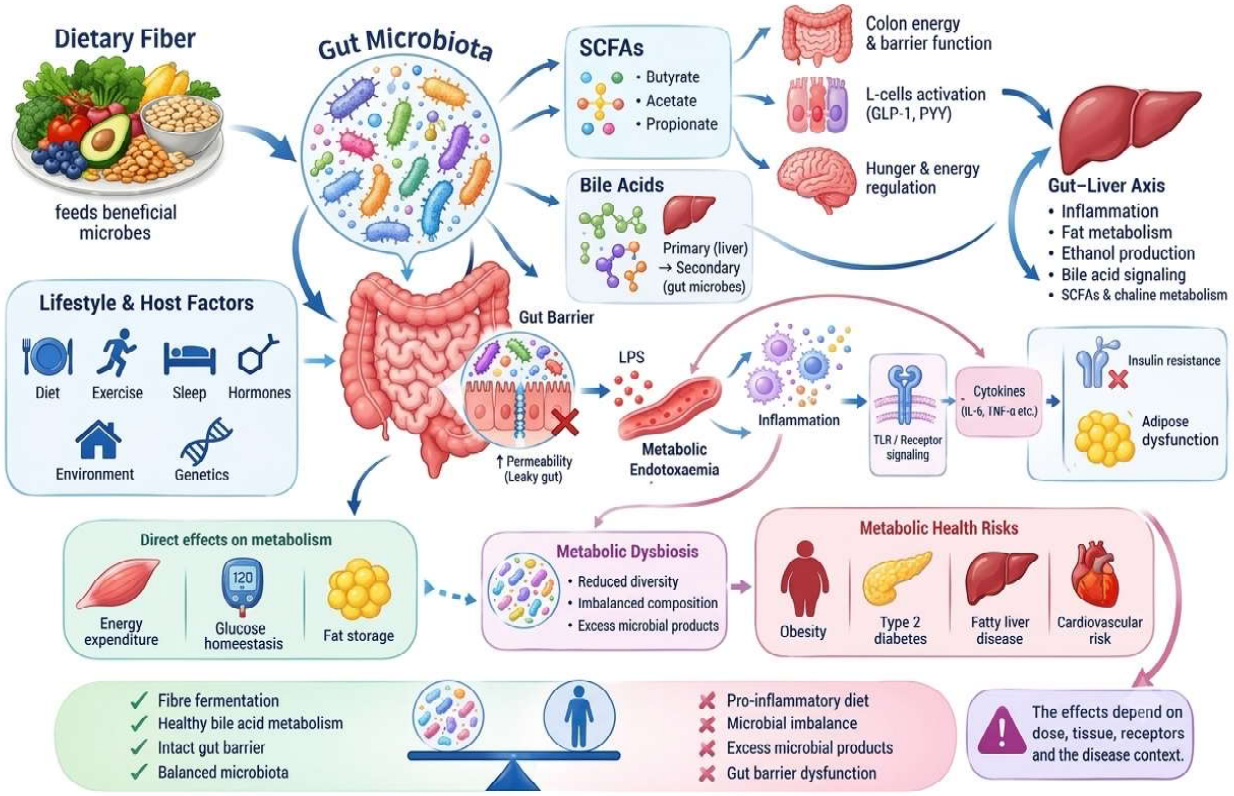

Fig. 1: Metabolic from Gut Microbiota and Gut-Liver Communication, in the Control of Metabolic Balance

of inflammation are tied to problems in fat tissue and resistance to insulin. People need control of diet, body weight, medicines and how samples are taken before saying that bacteria directly cause these problems (Frias et al., 2023).

In below shows how dietary fiber influences gut microbiota and metabolic health through metabolites, bile acids and the function of the gut barrier (Fig. 1). It also shows how microbial dysbiosis and inflammation can contribute to obesity type 2 diabetes, fatty liver disease and cardiovascular risks.

SCFAs provide another way that microbial metabolism can help keep the body in balance. Butyrate helps with the energy use of cells in the colon. Keeps the lining of the colon strong. Acetate and propionate are part of signals that travel through the body. SCFAs can turn on L-cells in the intestines. Signals made by the microbes can also influence the nerves that manage hunger and how the body uses energy. So, fiber in the diet can change how the body works by feeding the microbes of being used directly by the body (Eveleens et al., 2024). Bile acids are another system for signaling in the body. Primary bile acids start in the liver. Then gut microbes change them into secondary bile acids. Changes in bile acid levels have been seen in obesity and other metabolic problems (Gao et al., 2024). The liver is very sensitive to substances from the gut because the blood from the intestines goes to the liver. Changes in how tight the gut's how microbes work can then affect liver inflammation and how fats are handled. The link between the microbes and liver disease that comes with metabolism issues is complex. It includes things like making ethanol signaling from bile acids handling SCFAs managing choline and dealing with inflammation. This shows that the gut-liver connection is very important for health (Wang et al., 2024). Metabolic balance comes from a network, not one single process. Fibre being broken down helping the gut barrier SCFA signals and good changes to bile acids can all be good. Bad effects might come from eating patterns that cause inflammation, an unbalanced mix of microbes and too much of certain products from microbes. It is important to know that a substance can have effects depending on how much there is, which part of the body it is, in what receptors are present and what kind of disease is happening (Song et al., 2024).

MICROBIOTA–BRAIN COMMUNICATION AND NEUROINFLAMMATION

The gut contains an enteric nervous system. It has routes that link to the central nervous system. The vagus nerve provides a way for signals to travel from the gut to the brain. At the time metabolites and immune factors move through the bloodstream. These travel slowly but still connect the gut and brain. This back-and-forth communication affects gut movement, secretion, blood flow and the types of microbes living in the gut. This means stress and other brain activities can influence the gut environment (Segev et al., 2026). The blood-brain barrier and the intestinal epithelial barrier are parts of this system. They control what moves between areas of the body. They help protect tissues from microbial substance. Inflammation can change the gut environment. The term " gut" is often used. It only describes part of a much larger and complex process. Germ-free animals, which have no gut microbes have microglial shapes and functions. Short-chain fatty acids or SCFAs may help restore

some microglial development (Rodriguez et al., 2024).

Tryptophan metabolism gives another way. Dietary tryptophan can be turned by the body into serotonin and kynurenine. Changed by gut bacteria into indole compounds. Some of these indoles can turn on the aryl hydrocarbon receptor (AhR) a factor involved in keeping the lining of the body fighting infections. Changes in these processes can affect how the nervous system communicates with the system. Tryptophan metabolism depends a lot on the situation. Having more or less of a substance can't always be seen as good or bad (Voigt et al., 2022). Neuroinflammatory conditions give a reason to look at immune pathways. In diseases like sclerosis, Parkinsons disease and Alzheimers disease changes in the microbes and the products they make have been seen, but the results are different in different groups. The connection between the microbiota and the brain should be viewed as a set of parts rather than just a direct route from the gut to the brain. Understanding this group could show things that can be changed. Treatments need to tell the difference between things that are linked and things that are actually causing problems. Also the mix of microbes, in people is very different so that has to be taken into account (Pereira et al., 2022). The microbiota and the brain are connected in a complex way. It is not one path. There are different parts to this connection. The way we think about it should be different. We should not see it as a simple path. The microbiota and the brain are linked through parts. This makes the connection more complicated. The idea is not a single line from the gut to the brain. There are different elements involved. The relationship is more complex than we thought. Each part has its role. This helps us understand the connection better (Lubomski et al., 2022). The microbiota and the brain are connected in a complex way. It is not one simple path. There are parts to this system. The way we look at it should be different. We should not see it as a path. The microbiota and the brain are linked through parts. This makes the connection more complicated. The idea is not one simple line from the gut to the brain. There are different elements involved. The relationship is more complex, than before. Each part has its job. This helps us understand the connection better (Kim et al., 2022).

DIETARY PATTERNS AND METABOLIC DISORDERS

Eating habits shape metabolic health in two ways. Eating habits have an impact, on metabolic health guiding it along two clear paths. They directly affect the body. Also work through the microbiota. A typical Western diet is usually high in carbs, processed foods, saturated fats and calorie-dense foods. This type of diet has fiber diversity. It has been connected to obesity and metabolic problems. On the hand Mediterranean-style diets and other plant-based diets focus on vegetables, fruits, legumes, whole grains, nuts, seeds and unsaturated fats. These types of diets offer a range of microbial substrates. The microbiota can explain some of the benefits linked to these types of diets (Chen et al., 2022).

In below Fig. 2 shows how various eating habits affect the gut microbiome and metabolic well-being (Fig.2). A diet rich, in fiber and plant-based foods encourages microbes keeps the energy balance and manages glucose levels while Western diets and very processed diets can produce microbial imbalance increase obesity and trigger inflammation.

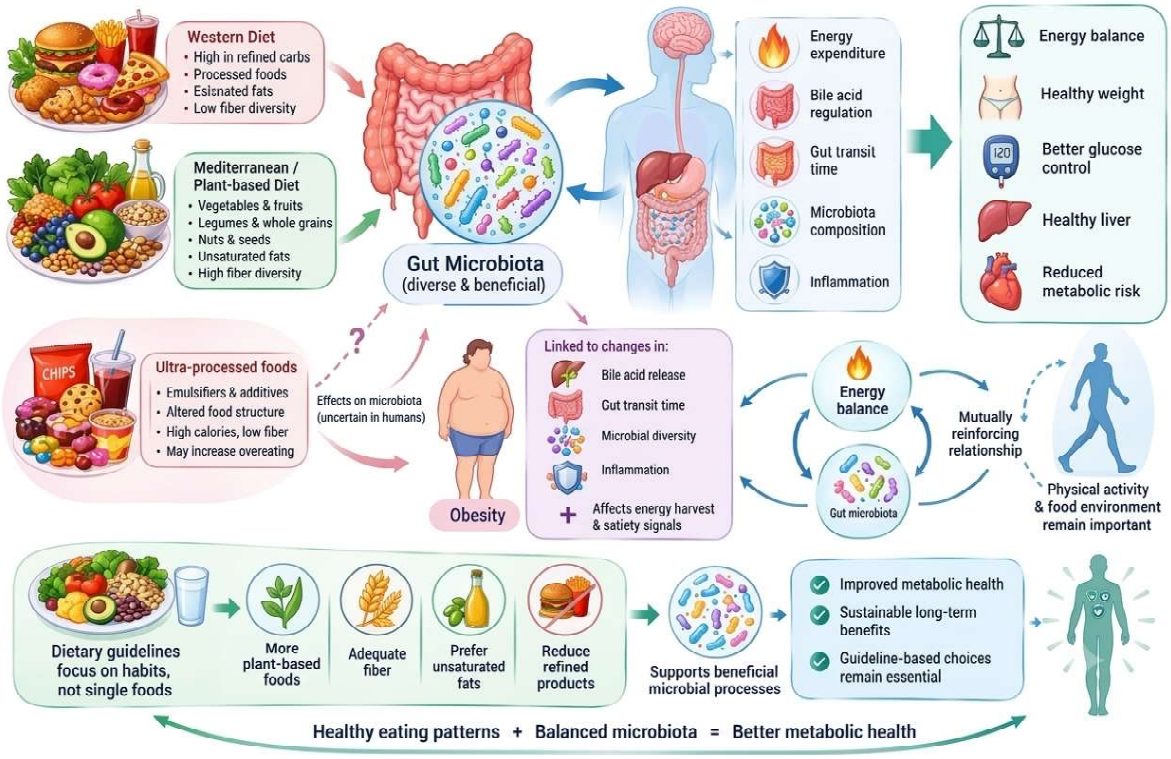

Fig. 2: Dietary Habits, Gut Microbiome Interactions and Their Consequences, for Metabolic Wellness and Illness

The consumption of ultra-processed foods is receiving focus as processing can change the food's structure and the way nutrients are provided. Emulsifiers, sweeteners and various additives can change the environment in laboratory experiments. It is not clear how they affect real people. The main worry, for health is that ultra-processed foods usually have a lot of calories taste great and have very little fiber, which can lead to overeating. Therefore, it is crucial not to attribute all health impacts of these foods solely to the gut microbiota (Metcalfe et al., 2024). Obesity shows how the balance of energy and the microbes, inside the body are linked. Obesity reveals that when a person eats calories than they burn it can affect many things. Obesity can change how bile acids are released. It can also change how fast food moves through the gut. It changes the number of microbes. It changes how inflammation there is. At the time the microbes in the gut can influence how much energy is taken from food. It can also influence how full a person feels. Studies suggest these two factors—energy balance and the microbiota—can support each other. With all this changes, in the microbiota do not take away the importance of energy balance. Physical activity or the overall food environment remain important (Scheperjans et al., 2024). Dietary guidelines should focus on habits than on the specifics of individual microbiome foods because habits shape long term health more, than any single food item. Increasing the variety of processed plant based foods making sure to eat enough fiber choosing unsaturated fats often and reducing excess refined products can help metabolic health and give resources for beneficial microbial processes. The evidence does not show that a specific probiotic or food

can stop diabetes or obesity. Microbiota science confirms the basis, for well known healthy eating patterns and gives new insights into how healthy eating patterns might work. Dietary guidelines remain the way to guide the choices dietary guidelines make (Lee et al., 2023).

GUT DYSBIOSIS IN NEUROINFLAMMATORY DISEASES

Dysbiosis has been seen in neuroinflammatory and neurodegenerative conditions and can show up as fewer of some taxa changes, in gene routes different metabolite production or weaker ecological balance(Hoffman et al., 2023). Experiments show that signals from microbes can shift the balance between T cells and the inflammatory Th17 cells. Short chain fatty acids and other microbe products can help keep the system balanced but when the mix of microbes changes the body may become more prone to inflammation. People with Multiple sclerosis, in places have shown different microbial patterns but those patterns are not the same everywhere (Schumacher et al., 2025). Parkinson's disease is an example of how the gut and brain can talk to each other. In Parkinson's disease gastrointestinal issues such as constipation often appear long before any movement problems are noticeable. Scientists have observed that changes in the gut wall's barrier function and in signals are linked to changes, in the gut's bacterial community. Research suggests that bacterial products might influence processes that involve alpha synuclein and could trigger the brain's response yet the exact cause and effect remain unclear. It is possible that gastrointestinal issues can alter the gut bacteria and at the time the altered gut bacteria could influence how Parkinson's disease progresses (Montgomery., 2024).

Alzheimer's disease has also been linked to changes in the kinds of microbes that live in the body and the substances they produce. Chronic inflammation throughout the body problems with blood vessels, weak barriers and broken immune signals might all be ways that Alzheimer's disease happens. The microbes might influence these problems by sending out substances that travel in the blood and by affecting the system. However research on Alzheimer's disease in people is often limited by age many medicines and big differences, in diet and other health problems which makes it hard to prove cause and effect (Ayten et al., 2024). Neurodevelopmental disorders have drawn a lot of attention in microbiota research. Studies on autism spectrum disorder have found differences in the makeup and digestive problems in some people. When the microbiota from people with these conditions was moved into germ- animals the animals showed changes, in behavior. These findings show that it is possible biologically. They don't back up simple claims that gut bacteria cause autism. Brain development is influenced by genes what happens during pregnancy the way the immune system develops and many other factors (Schneider et al., 2024). A link between balance of gut bacteria and inflammation in the brain is the breakdown of the gut barrier. The gut lining usually controls how exposure there is to bacteria parts. If there are changes in how tight the connections between cells are in the mucus layer or in how the lining cells work then more bacteria parts could get into the body. The brain has its own protective barriers and signals, from the gut bacteria may change how open these barriers are and how the immune system works. The idea is therefore best shown as a group of barriers connecting the gut the blood and the brain (Zhou et al., 2024).

Another idea people think about is that SCFAs might run out. SCFAs like butyrate and other similar chemicals can change how cells that control inflammation and brain cells called microglia behave. It is important to know that SCFAs are not always good for everyone. Their effect depends on how many receptors are on cells, where, in the body they are and the overall state of the body's metabolism (Lvanich et al., 2025). Another concern is directionality. Neurological disorders can change appetite, food choices swallowing ability, motility and physical movement. Patients may therefore follow diets or experience changes in intestinal transit which can cause secondary changes, in the microbiome. Drugs can produce effects. Consequently, longitudinal studies that begin before the disease starts or in its stages are especially important (Svendsen et al., 2024). One clear point is that gut dysbiosis can make sense as a reason for neuroinflammation. It is unlikely to be the only cause in every case. Future research should move beyond listing bacteria. It should look at what those bacteria can do like the genes they carry the chemicals they make the signals they trigger and how well the gut lining works. By doing this, scientists may find ways the disease works, which could help doctors treat it better than, by focusing on a single bacterial species (Rebeaud et al., 2022).

MICROBIAL METABOLITES LINKING METABOLISM AND BRAIN HEALTH

Microbial metabolites form a connection between the gut microbiota the body's metabolism and brain health. Unlike taxonomy, which focuses on types of bacteria metabolites represent the actual functional molecules produced by these microbes. These molecules can interact with the host's receptors, enzymes and immune cells leading to ranging effects. Important groups include short-chain fatty acids, bile-acid derivatives, tryptophan metabolites, phenolic compounds, substances derived from trimethylamine and various products made from amino acids. One single metabolite can affect than one organ meaning signals, from the gut may travel to influence both the liver and the brain. This shows how deeply linked the gut is to bodily function (O Riordan et al., 2022). Bile acids create another way for the body to work. Bile acids that are made in the liver are sent to the intestine. Changed by tiny living things. Bile acids that are changed then send messages through FXR, TGR5 and other receivers. Because bile acids manage sugar, fat, swelling and how the body uses energy changes in how tiny living things change bile acids can have an effect on diseases related to metabolism. Maybe even on swelling in the brain. Bile acids and the receivers they use are being studied closely as parts of the mix, between tiny living things and the body (Yang et al., 2021). Microbes make special chemicals called metabolites by changing polyphenols. Each chemical acts differently. How each persons microbes work can change the amount and type of these chemicals made. This shows that we need to look at what's, in food and what the microbes can do together (Zhao et al., 2021).

TMAO show a link between what we eat the microbes in our gut and the health of our heart and brain. Microbes can turn foods such as choline and carnitine into Trimethylamine and liver enzymes can then turn Trimethylamine into TMAO. Higher levels of TMAO have been linked to heart disease. Have been studied for their role in inflammation of blood vessels. Because the health of blood vessels has an impact on brain health this

pathway creates an indirect connection between diet, microbial activity and risk, to the brain. Still TMAO is not a poison; TMAO is an active substance that can be good or bad depending on the context (Kim et al., 2025). A common organism might not have much effect in patients if the substance it needs is missing. Metabolomics can show if an action actually changes the chemical processes that are thought to bring about benefits. This is important for moving research about the microbiome from seeing connections, to understanding real causes (Sun et al., 2024). In the end tiny substances made by microbes can be used as signs of health or sickness and as ways to treat diseases. These substances should not always be seen as good or bad. How much of them is there when they are present where in the body they are found, if the body has the receptors for them and the overall health of the person all affect what happens in the body. Because of this future studies with patients should include records of what people eat information about the genes of the microbes in the body measurements of the substances, in the body and checks of the immune system. These ways of looking at different types of data could find patterns of substances that show which people will benefit the most from a certain diet (Aljada et al., 2021).

NUTRITIONAL AND MICROBIOTA-BASED INTERVENTIONS

Nutritional and microbiota based interventions aim to restore a gut microbial environment. Nutritional and microbiota based interventions also work to improve metabolic outcomes immune outcomes and neurological outcomes. I notice that dietary modification, prebiotics, probiotics, synbiotics, postbiotics and fecal microbiota transplantation (FMT) all act through mechanisms. These mechanisms include modulation of composition production of beneficial metabolites strengthening of the intestinal barrier and regulation of inflammation.

TRANSLATIONAL AND PUBLIC HEALTH PERSPECTIVES

Translating science into public health requires a balance between enthusiasm and evidence. The microbiota offers a reason, for how diet can influence distant organs. Yet marketing claims often go beyond the evidence. Therefore public health guidelines should focus on well established principles while slowly adding new microbiome ideas. At the population level making sure there are lots of processed foods available might be more important than encouraging people to buy expensive products that claim to improve the microbiome. Foods like fiber, whole grains, legumes, vegetables, fruits, nuts and seeds provide different kinds of nutrients and substances that the body needs. Policies that make these foods easier to get can help reduce health risks related to metabolism through ways that are already known even if we don't fully understand how the microbiome works. This approach also stops people from thinking about nutrition in terms, like just "good bacteria" and "bad bacteria" (Heidelbaugh et al., 2023). Dietary patterns are heavily influenced by factors. When creating guidelines it's important to take into account the cost of food how easy it is to get peoples cultural food preferences and the kind of cooking equipment they have available.

We must be careful not to let microbiome research create a kind of inequality, where only wealthy people can afford personalized genetic testing and expensive supplements. If

Table 1: The major inventions, their mechanisms, potential applications and limitations are summarized.

Intervention	*Main target*	*Proposed mechanism*	*Potential application*	*Major Limitation*	*References*
High-fibre diet	SCFA-producing bacteria, Gut microbial fermentation	Increase SCFAs (butyrate, acetate, propionate), Increase fermentation of complex carbohydrates	Enhanced gut barrier function, Weight management, Improved insulin sensitivity	Tolerance and GI symptoms (bloating, gas), Individual variation in response	(Barber et al., 2023)
Mediterranean diet	Beneficial commensals (*e.g., Bifidobacterium, Lactobacillus*), Overall microbiome ecology	Anti-inflammatory effects, High fibre, polyphenols, healthy fats	Improved lipid profile, Reduced chronic inflammation, Cardiometabolic health	Cultural and economic differences, Adherence and lifestyle factors	(Kiani et al., 2022)
Prebiotics	Beneficial gut bacteria (*e.g., Bifidobacterium, Lactobacillus*)	Increased SCFA production, Modulation of gut environment, Selective fermentation	Improved gut function, Immune regulation, Metabolic health	Dose-dependent effects, GI intolerance (bloating, flatulence)	(Du et al., 2024)
Probiotics	Specific strains (*e.g., Lactobacillus, Bifidobacterium*)	Immune modulation, Barrier strengthening, Competitive exclusion	Metabolic benefits, Reduced inflammation, IBS and gut disorders	Variable efficiency, Survival colonization challenges, Strain specificity	(Du et al., 2024)
Synbiotics	Beneficial bacteria + prebiotic substrate	Increased SCFA production, Enhanced survival and activity of probiotics	Gut barrier support, Immune modulation, Metabolic health	Optimal combination not well defined, Limited-high quality evidence	(Alli et al., 2022)
Postbiotics	Microbial metabolites (*e.g., SCFAsindoles, peptides*)	Anti-inflammatory and antioxidant actions, Direct signalling effects	Immunomodulation, Metabolic and neurological benefits, Gut barrier protection	Limited clinical data, Dosage and formulation challenges, Standardization issues	(Scott et al., 2022)
Fecal Microbiota Transplantation (FMT)	Complete microbial community from healthy donor	Restoration of microbial diversity, Ecological replacement	Experimental for metabolic and neuroinflammatory disorders, Established for recurrent *C. difficile* infection	Long-term effects unknown, Ethical and practical challenges, Safety and infection risk	(Singh et al., 2022)

microbiome-based nutrition turns out to be clinically effective then making it affordable and accessible, to everyone will be essential. A different challenge in translation is standardization. Many laboratories might use sequencing technologies, databases, sample-processing techniques and definitions of dysbiosis. Stool specimens give insights but might not fully show mucosal communities or microbial functions in different intestinal areas. Measurements of metabolites are affected by the methods used for collection, storage and analysis. Therefore standard operating procedures are necessary for comparisons, among studies (Knight et al., 2022). Long-term studies hold importance. Long-term studies can reveal associations. Cannot ascertain if microbiome changes occur before disease or are a consequence of it. Ultimately translation research needs teamwork from fields. Nutrition researchers, microbiology experts, medical practitioners, immunology specialists, neuroscience scholars, epidemiology analysts, statisticians and public health officials all need the clear standards in research. Animal research, on how things work's still useful but it must be followed by well planned research trials in humans. Human research must include different groups because microbiomes and diet habits change with place and culture (Ye et al., 2026).

CONCLUSIONS

Diet and gut microbiota work together to shape how Diet and gut microbiota influence how body uses energy, how body's immune system fights off germs and how brain receives signals. Our Diet and gut microbiota change the kinds of microbes that live in our gut. These microbes these microbes produce substances that affect the body's signals and these body signals influence obesity, diabetes, heart disease risk and brain inflammation. Key ideas include that diet, diet impacts the makeup of gut microbes; substances made by these microbes substances made by these microbes like short chain fatty acids influence body processes; problems in these processes are linked to diseases; changing what people eat can shift how microbes function but people react differently to the same changes; and there is stronger proof that diet, diet affects metabolism more than it affects neuroinflammation. Future research should focus on human studies, consistent methods and clear outcomes while considering individual differences. In the end boosting diversity and the body's ability to adapt metabolically through food choices is a public health strategy, than chasing an "ideal microbiome." This approach supports nutrition, better metabolism and stronger brain health (Perler et al., 2023).

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