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ORIGINAL ARTICLE

THE GUT–BRAIN AXIS: MICROBIOTA-MEDIATED REGULATION OF NEUROINFLAMMATION AND COGNITIV

Mirza Mohammad Zakwan kausar baig[1],

Group - 120, Samarkand state medical University[1]

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Abstract

The gut–brain axis represents a complex bidirectional communication network connecting the gastrointestinal tract and the central nervous system through neural, immune, endocrine, metabolic, and microbial pathways. Growing evidence indicates that the intestinal microbiota participates in the regulation of neuroinflammation, neuronal activity, synaptic function, stress responses, and cognitive processes. Microorganisms residing in the gastrointestinal tract produce metabolites, including short-chain fatty acids, tryptophan-derived compounds, bile acid derivatives, and other bioactive molecules that can influence systemic immunity and brain physiology. At the same time, intestinal barrier dysfunction and microbial imbalance may increase inflammatory signaling and contribute to activation of microglia and astrocytes. Persistent neuroinflammation has been associated with impaired neuronal communication and cognitive dysfunction and may participate in the pathophysiology of several neurological and psychiatric disorders. This article examines the relationship between intestinal microbiota, neuroinflammatory mechanisms, and cognitive function, with particular attention to microbial metabolites, intestinal and blood–brain barrier integrity, immune signaling, and potential therapeutic interventions. Understanding these mechanisms may contribute to the development of microbiota-oriented strategies for maintaining neurological health and preventing cognitive decline.

Keywords: gut–brain axis, intestinal microbiota, neuroinflammation, cognitive function, microbiome, microglia, short-chain fatty acids, intestinal barrier, blood–brain barrier, brain health.

1. Introduction

The human gastrointestinal tract contains a highly diverse microbial ecosystem composed of bacteria, archaea, fungi, viruses, and other microorganisms. This microbial community interacts continuously with intestinal epithelial cells, immune cells, dietary components, and host metabolic pathways. Although the majority of research initially focused on the role of intestinal microorganisms in digestion and metabolism, it is now evident that the gut microbiota can influence physiological processes far beyond the gastrointestinal system.

The concept of the gut–brain axis describes the multidirectional communication between the gastrointestinal tract and the central nervous system. This communication involves several interconnected pathways, including the vagus nerve, enteric nervous system, hypothalamic–pituitary–adrenal axis, immune system, microbial metabolites, and circulating

endocrine mediators. Through these pathways, intestinal microorganisms can influence brain function, while psychological stress and neurological processes can simultaneously alter gastrointestinal motility, secretion, permeability, and microbial composition.

The gut microbiota is not a static population. Its composition and functional activity can change in response to age, diet, medications, physical activity, infection, stress, sleep patterns, environmental factors, and underlying disease. Such changes may influence the production of microbial metabolites and the interaction between microorganisms and host immune cells.

One of the most important mechanisms linking the microbiota to brain function is regulation of inflammation. Under physiological conditions, the intestinal barrier limits uncontrolled movement of microbial products into the circulation. The epithelial layer, mucus, antimicrobial peptides, and immune mechanisms work together to maintain controlled communication between the intestinal contents and host tissues.

When intestinal barrier integrity is impaired, microbial components and metabolites may gain greater access to the systemic circulation. This can influence peripheral immune activity and potentially alter signaling at the blood–brain barrier. Persistent peripheral inflammation may contribute to changes in central immune responses and activation of resident glial cells.

Microglia are the principal innate immune cells of the central nervous system. They continuously monitor the local neural environment and respond to injury, infection, metabolic disturbances, and inflammatory signals. Controlled microglial activation is important for maintaining tissue homeostasis, whereas persistent or excessive activation can contribute to production of inflammatory mediators and neuronal dysfunction.

Astrocytes also participate in communication between systemic physiology and the nervous system. These cells contribute to maintenance of the blood–brain barrier, neurotransmitter regulation, metabolic support, and immune signaling. Changes in systemic inflammatory status may therefore influence astrocyte behavior and alter the neural microenvironment.

Microbial metabolites provide another important pathway through which the gut can influence the brain. Short-chain fatty acids, particularly acetate, propionate, and butyrate, are generated through microbial fermentation of dietary components. These compounds can affect immune function, epithelial integrity, cellular metabolism, and signaling pathways relevant to neurological physiology.

Butyrate has received particular attention because of its potential effects on intestinal barrier integrity and immune regulation. It can influence gene expression through epigenetic mechanisms and may affect inflammatory signaling. However, the biological effects of microbial metabolites depend on their concentration, tissue distribution, metabolic context, and interactions with host cells.

Tryptophan metabolism represents another important component of the gut–brain connection. Intestinal microorganisms can transform tryptophan into several bioactive molecules that interact with host signaling pathways. These pathways may influence serotonin metabolism, immune responses, and aryl hydrocarbon receptor signaling, thereby connecting intestinal microbial activity with neurological and immunological processes.

Bile acid metabolism also contributes to gut–brain communication. Intestinal microorganisms modify primary bile acids into secondary derivatives that can interact with host receptors involved in metabolism, immune regulation, and cellular signaling. Alterations in microbial bile acid metabolism may consequently influence systemic and neurological physiology.

Neurotransmitter-related mechanisms have also attracted significant scientific interest. Certain intestinal microorganisms can produce or influence molecules associated with neurotransmission, including gamma-aminobutyric acid, serotonin-related compounds, dopamine-related metabolites, and other signaling molecules. Nevertheless, the direct contribution of microbial neurotransmitter production to human brain neurotransmission remains complex because many of these molecules do not simply cross the blood–brain barrier.

The vagus nerve provides a major neural pathway connecting the gastrointestinal tract with the brain. Signals generated by intestinal epithelial cells, immune cells, and microbial interactions can influence vagal activity. Conversely, central nervous system signals can modify gastrointestinal function and potentially affect the microbial environment.

Stress represents an important example of bidirectional gut–brain communication. Activation of the hypothalamic–pituitary–adrenal axis can alter gastrointestinal motility, secretion, immune function, and intestinal permeability. These changes may modify the microbial ecosystem. In the opposite direction, microbial metabolites and inflammatory mediators can influence stress-related neural

pathways.
Cognitive function is particularly relevant to the study of the gut–brain axis. Memory, attention, learning, executive function, and emotional regulation depend on coordinated neuronal activity, synaptic plasticity, adequate metabolic support, and controlled immune signaling. Chronic inflammatory processes may interfere with these mechanisms.
Neuroinflammation has been implicated in cognitive dysfunction associated with aging and several neurological disorders. Excessive inflammatory signaling can influence synaptic function, neuronal survival, neurotransmitter systems, and neurovascular processes. Because the intestinal microbiota participates in systemic immune regulation, it has become an important target of research into mechanisms underlying cognitive decline.
Age is an important factor in this relationship. Both the intestinal microbiome and immune system undergo significant changes during aging. Reduced microbial diversity, altered microbial metabolic activity, changes in intestinal barrier integrity, and chronic low-grade inflammation may contribute to increased vulnerability of the aging brain.
Diet represents one of the most modifiable factors affecting the gut microbiota. Diets rich in diverse plant-derived fibers generally provide substrates for microbial fermentation and production of beneficial metabolites, whereas diets characterized by excessive amounts of highly processed foods may alter microbial diversity and metabolic function.
Antibiotics can also produce substantial changes in intestinal microbial communities. Although antimicrobial therapy is often essential for treating bacterial infections, disruption of the microbiota may temporarily or persistently alter microbial composition and metabolic activity. The neurological implications of such changes remain an active area of investigation.
Probiotic, prebiotic, synbiotic, dietary, and microbiota-directed interventions have therefore attracted considerable interest. However, their effects are not uniform across individuals, and clinical outcomes may depend on baseline microbiota composition, diet, age, genetics, disease status, and treatment duration.
The relationship between microbiota and cognitive function should consequently not be interpreted as a simple cause-and-effect interaction. The gut microbiome is part of a larger biological network in which genetic, metabolic, environmental, behavioral, immune, and neurological factors interact continuously.
The aim of this study is to examine the potential mechanisms through which intestinal microbiota influence neuroinflammation and cognitive function, with emphasis on microbial metabolites, intestinal barrier integrity, immune signaling, glial activation, and the therapeutic potential of microbiota-targeted interventions.

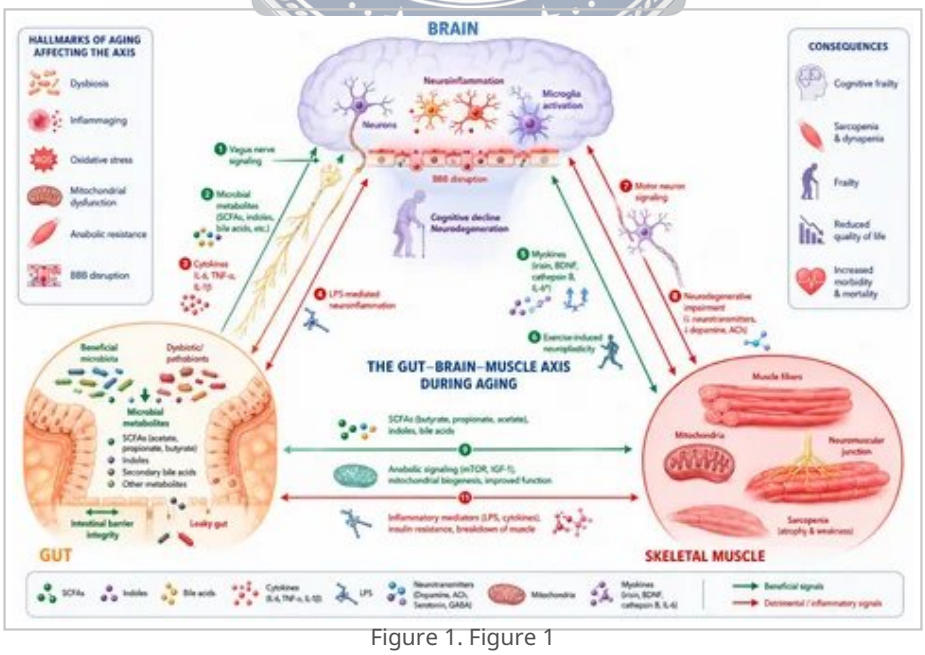

Figure 1. Figure 1

2. Materials and Methods

A descriptive analytical approach was used to examine current evidence concerning the relationship between intestinal microbiota, neuroinflammation, and cognitive function. Scientific information concerning gut microbial composition, microbial metabolites, immune signaling, neurological pathways, and cognitive outcomes was analyzed to construct an integrated model of gut–brain communication.

The study framework included evidence from experimental research, observational investigations, clinical studies, and mechanistic literature addressing interactions between intestinal microorganisms and the central nervous system.

The principal areas of analysis included intestinal microbial diversity, intestinal barrier function, systemic inflammatory signaling, blood–brain barrier integrity, microglial activation, astrocyte responses, microbial metabolite production, and cognitive performance.

Particular attention was given to short-chain fatty acids, tryptophan-derived metabolites, bile acid derivatives, microbial cell-wall components, and other biologically active substances capable of influencing host immune or metabolic pathways.

Studies examining cognitive outcomes were evaluated according to measures of memory, learning, attention, executive function, behavioral performance, and global cognitive status when such parameters were available.

Potential confounding factors were considered, including age, sex, diet, body composition, physical activity, medication exposure, antibiotic use, metabolic disease, sleep quality, psychological stress, and pre-existing neurological conditions.

The analysis also considered experimental evidence involving changes in gut microbial composition and subsequent effects on inflammatory signaling or neurological function. Animal and cellular studies were interpreted primarily as mechanistic evidence rather than direct proof of clinical effects in humans.

Evidence concerning microbiota-targeted interventions was evaluated according to intervention type, including probiotics, prebiotics, dietary modification, synbiotics, fermented foods, and other microbiome-directed approaches.

The primary analytical outcomes were the association between microbial alterations and neuroinflammatory signaling and the potential relationship between microbiota-associated mechanisms and cognitive performance.

Secondary outcomes included changes in intestinal barrier integrity, circulating inflammatory mediators, glial activation, microbial metabolite profiles, and behavioral or neurological measures.

Because microbiome composition can vary substantially between individuals and populations, emphasis was placed on functional microbial activity and metabolic pathways rather than on the presence or absence of individual bacterial species alone.

The available evidence was interpreted using a mechanistic and translational perspective, distinguishing established biological mechanisms from associations that require further clinical confirmation.

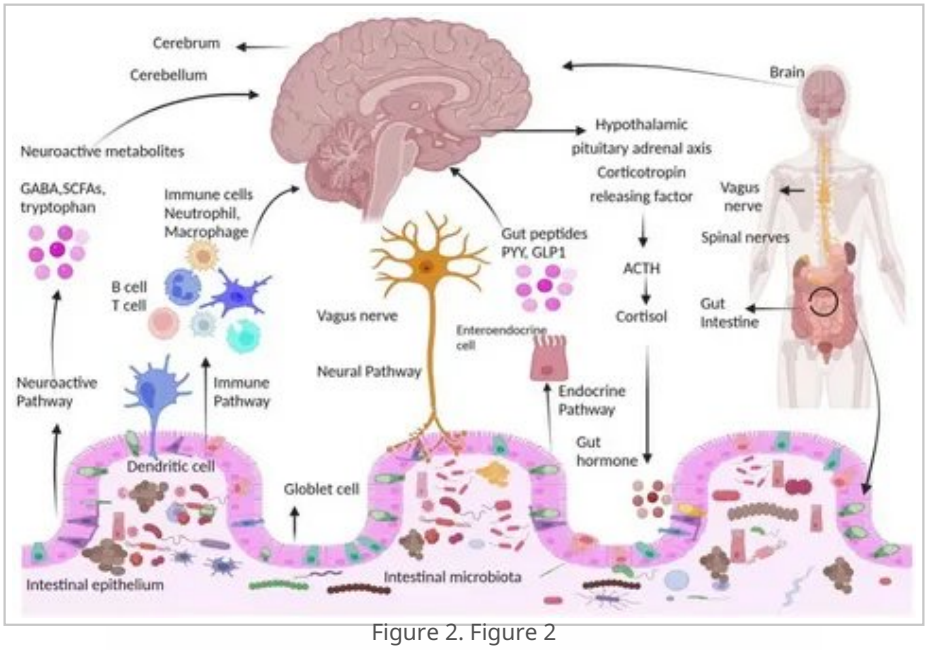

Figure 2. Figure 2

3. Results

The analysis demonstrated that the gut microbiota is closely connected with several biological pathways involved in the regulation of neuroinflammation and cognitive function. Although the composition of the intestinal microbiome varies considerably between individuals, changes in microbial diversity and metabolic activity were repeatedly associated with alterations in immune signaling, intestinal barrier function, and neurological physiology.

One of the most consistent observations was the relationship between intestinal microbial imbalance and inflammatory activity. Alterations in microbial composition may increase exposure to microbial products capable of stimulating innate immune receptors. This can promote the release of inflammatory mediators and contribute to a persistent low-grade systemic inflammatory state.

Changes in intestinal barrier integrity were also identified as an important component of gut–brain communication. Under physiological conditions, intestinal epithelial cells and intercellular junctions restrict uncontrolled movement of microbial products into the circulation. When barrier function becomes compromised, bacterial components and other microbial molecules may enter the systemic environment more readily and influence immune responses.

The analysis further indicated that systemic inflammatory signaling may affect the central nervous system. Circulating inflammatory mediators can interact with endothelial and immune mechanisms at the blood–brain barrier and may influence glial cells. Increased inflammatory signaling may contribute to activation of microglia and astrocytes, particularly when the inflammatory stimulus is prolonged.

Microglial activation was identified as a potential mechanistic link between peripheral immune changes and cognitive function. Moderate microglial responses are essential for maintaining neural homeostasis, but prolonged activation may increase the production of inflammatory mediators and reactive molecules. These processes may interfere with synaptic communication and neuronal plasticity.

Microbial metabolites represented another major pathway identified in the analysis. Short-chain fatty acids, particularly acetate, propionate, and butyrate, have multiple biological functions relevant to gut and brain physiology. Their effects include modulation of immune activity, regulation of intestinal barrier function, interaction with cellular receptors, and epigenetic regulation of gene expression.

Higher availability of beneficial microbial fermentation products was generally associated with a more favorable intestinal environment. However, the relationship between individual metabolites and neurological outcomes was not uniform. Metabolite effects depend on microbial composition, dietary substrate availability, host metabolism, and the physiological context in which these molecules act.

Tryptophan metabolism also emerged as an important component of microbiota-mediated signaling. Intestinal microorganisms can transform dietary tryptophan into multiple metabolites that interact with host immune and metabolic pathways. These compounds may influence inflammatory regulation

and communication between intestinal and neural tissues.

The analysis indicated that microbial regulation of tryptophan metabolism may affect pathways involving the aryl hydrocarbon receptor and other signaling systems. These mechanisms provide a plausible connection between intestinal microbial activity and immune regulation within the nervous system.

Microbial modification of bile acids was another potentially relevant mechanism. Intestinal bacteria participate in conversion of primary bile acids into secondary metabolites with biological signaling properties. These compounds can interact with host receptors involved in metabolism and immune regulation and may indirectly influence neurological homeostasis.

Neural communication through the vagus nerve was also identified as an important pathway. Signals generated in the gastrointestinal tract can influence vagal activity, providing a rapid route through which intestinal conditions may affect central nervous system function. This pathway may interact with immune and metabolic signals rather than functioning independently.

Several experimental studies demonstrated associations between alterations in gut microbial communities and behavioral or cognitive changes. These observations suggest that the microbiota can influence processes related to learning, memory, stress responsiveness, and emotional behavior. However, the strength of these effects varied considerably between experimental models.

Human studies generally demonstrated associations rather than definitive causal relationships. Individuals with certain neurological or cognitive conditions may exhibit altered microbial profiles, but it remains difficult to determine whether microbial changes contribute directly to disease development or arise as a consequence of altered diet, medication use, reduced physical activity, disease-related physiology, or other factors.

Age-related changes in the microbiota were associated with several processes relevant to cognitive decline. Reduced microbial diversity, changes in metabolite production, intestinal barrier dysfunction, and chronic low-grade inflammation may collectively increase vulnerability to neuroinflammatory processes in older individuals.

Dietary patterns appeared to have a substantial influence on the gut–brain relationship. Diets providing diverse sources of dietary fiber support microbial fermentation and production of short-chain fatty acids. In contrast, diets characterized by low fiber diversity and high consumption of highly processed foods may produce a less favorable microbial metabolic environment.

The analysis of microbiota-targeted interventions suggested that probiotics, prebiotics, synbiotics, and dietary modification may influence selected markers of intestinal and systemic physiology. Some studies reported improvements in inflammatory parameters, stress-related symptoms, or aspects of cognitive performance, but results were inconsistent across populations.

The greatest variability was observed among probiotic interventions. Different microbial strains can have substantially different biological effects, meaning that the term “probiotic” cannot be interpreted as a single uniform therapeutic intervention. Dose, strain characteristics, treatment duration, host factors, and baseline microbiota composition may all influence outcomes.

Overall, the results support the existence of a biologically plausible connection between intestinal microbiota, systemic immune regulation, neuroinflammation, and cognitive function. However, the available evidence also indicates that these relationships are complex and influenced by multiple interacting variables.

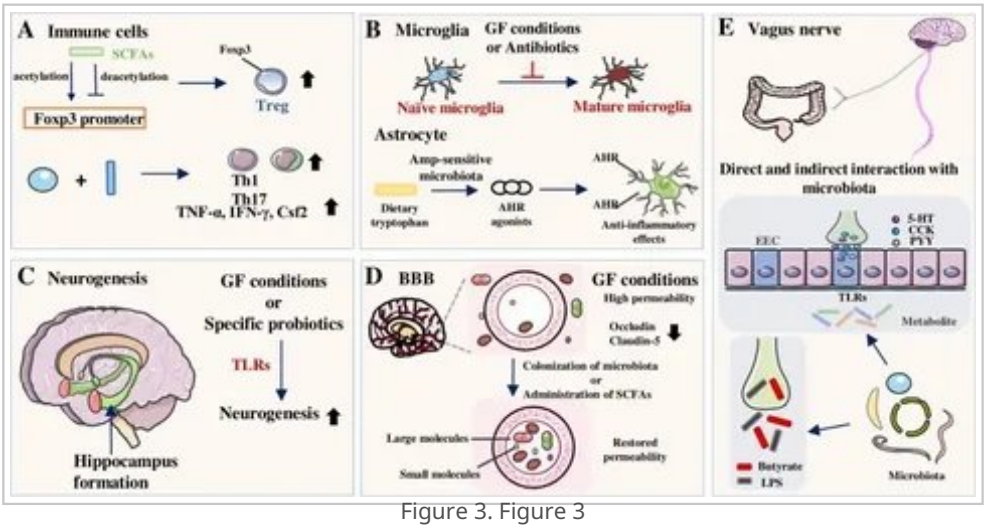

Figure 3. Figure 3

4. Discussion

The findings support the concept that the gut–brain axis represents an integrated biological network rather than a single communication pathway. Microbial microorganisms interact with intestinal epithelial cells, immune cells, metabolic systems, and neural structures, while signals generated by the brain can simultaneously influence gastrointestinal physiology.

One of the most important mechanisms appears to involve regulation of inflammatory tone. The intestinal microbiota participates in maturation and regulation of the immune system. A balanced microbial ecosystem may contribute to controlled immune responses, whereas disruption of microbial homeostasis can favor excessive inflammatory signaling.

The intestinal barrier plays a central role in this process. A healthy barrier separates the microbial ecosystem from the internal environment while permitting selective exchange of nutrients and signaling molecules. Impaired barrier integrity may increase systemic exposure to microbial components and potentially amplify inflammatory responses.

The relationship between intestinal permeability and neuroinflammation should nevertheless be interpreted cautiously. Increased intestinal permeability does not automatically produce neurological disease. The outcome depends on the nature and duration of the inflammatory stimulus, host immune status, blood–brain barrier integrity, metabolic state, and other physiological factors.

Microglia provide an important connection between systemic inflammation and brain function. These cells respond to changes in their microenvironment and participate in synaptic remodeling and immune surveillance. Persistent inflammatory stimulation may alter their functional state and interfere with processes required for normal neuronal communication.

Chronic neuroinflammation may affect cognition through several mechanisms. Excessive inflammatory signaling can influence synaptic plasticity, neurotransmitter metabolism, mitochondrial function, neuronal survival, and neurovascular regulation. Therefore, microbiota-mediated modulation of systemic inflammation could theoretically influence cognitive performance through multiple interconnected pathways.

Short-chain fatty acids deserve particular attention because they provide a direct metabolic connection between dietary intake, microbial activity, intestinal physiology, and systemic signaling. Butyrate, for example, contributes to intestinal epithelial health and can influence immune responses and gene regulation. However, the assumption that increased levels of any individual short-chain fatty acid will always produce neurological benefits would be overly simplistic.

The biological effects of microbial metabolites are context-dependent. Their concentration, site of production, receptor expression, metabolic conversion, and interaction with other molecules all influence their physiological activity. Consequently, future microbiome research should focus increasingly on functional metabolic profiles rather than simply counting bacterial species.

Tryptophan metabolism illustrates another level of complexity. The same dietary precursor can enter several metabolic pathways involving both host and microbial enzymes. Products of these pathways may influence neurotransmitter-related processes, immune regulation, and epithelial signaling. Changes in microbial metabolism may therefore modify the balance between different biologically

active compounds.

The bile acid pathway provides an additional example of microbial regulation of host physiology. Microorganisms alter the chemical structure of bile acids, generating compounds capable of interacting with receptors involved in metabolism and immune signaling. Because these receptors are expressed in multiple tissues, including tissues relevant to neurological function, bile acid signaling may contribute to systemic communication.

The vagus nerve provides a rapid neural pathway through which gastrointestinal signals may reach the central nervous system. However, the vagal pathway operates together with endocrine and immune mechanisms. It is therefore more appropriate to view the gut–brain axis as an integrated network than to attribute microbiota-related neurological effects to a single pathway.

Cognitive function is particularly sensitive to systemic physiological changes. Sleep disruption, metabolic disease, chronic stress, inflammation, physical inactivity, and nutritional deficiencies can all influence cognition. Many of these factors simultaneously affect the intestinal microbiota, making it difficult to separate direct microbial effects from broader lifestyle-related influences.

This issue is particularly important when interpreting human microbiome studies. Associations between altered microbial composition and cognitive impairment do not necessarily demonstrate that the microbiota caused the cognitive change. Reverse causality is possible, as cognitive impairment may lead to dietary changes, reduced activity, altered medication use, and changes in daily routines that subsequently influence the microbiome.

Medication exposure is another important confounding factor. Antibiotics can substantially modify microbial communities, while other commonly used medications may also affect intestinal microbial composition or metabolism. Future clinical studies should therefore carefully document medication exposure.

Diet must likewise be considered. Individuals with cognitive impairment may have different dietary patterns compared with healthy individuals. Since diet is one of the strongest determinants of microbial composition, dietary differences may partially explain observed associations.

The therapeutic implications of gut–brain research are nevertheless promising. Dietary strategies that increase the diversity of plant-derived fibers may support microbial fermentation and production of beneficial metabolites. Such interventions may provide a relatively accessible component of broader strategies for maintaining metabolic and neurological health.

Probiotic and prebiotic interventions may also have potential, but their clinical application requires greater precision. Different strains and formulations produce different effects, and a beneficial intervention for one population may have limited effects in another.

The concept of personalized microbiome therapy is therefore increasingly important. Future interventions may be designed according to individual microbial profiles, metabolic characteristics, dietary patterns, genetic factors, and clinical conditions.

Prebiotics, probiotics, and synbiotics should not be regarded as universal treatments for neurological disease. Their potential benefits should be evaluated using well-designed clinical trials with standardized cognitive outcomes, microbiological measurements, inflammatory markers, and sufficiently long follow-up.

Other approaches, including fecal microbiota transplantation and microbiota-derived metabolite supplementation, require careful investigation before broad neurological application. Safety, long-term ecological consequences, donor-related variables, and patient selection remain important considerations.

The gut–brain axis may also provide opportunities for prevention. Maintaining a diverse and metabolically active microbiota through balanced nutrition, regular physical activity, adequate sleep, appropriate antimicrobial use, and healthy lifestyle practices may contribute to overall physiological resilience.

Future research should increasingly combine microbiome sequencing with metabolomics, immunological measurements, neuroimaging, cognitive testing, and longitudinal clinical assessment. Such integrated approaches may clarify which microbial changes are biologically meaningful and which are simply markers of other disease processes.

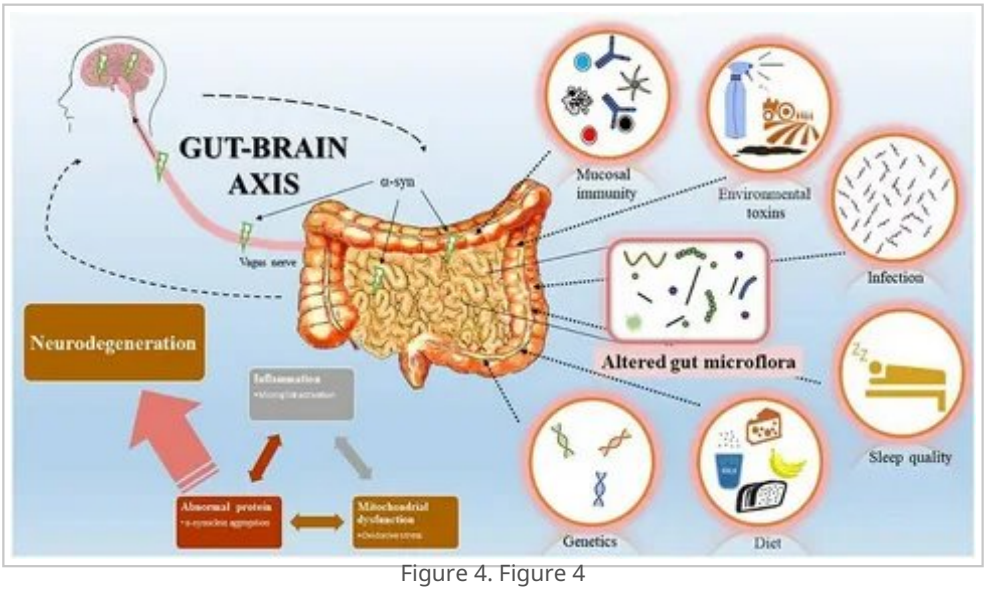

Figure 4. Figure 4

5. Conclusion

The gut–brain axis represents a complex communication system through which intestinal microorganisms can influence immune regulation, metabolism, neural signaling, and potentially cognitive function. Microbial effects are mediated through several interconnected mechanisms, including intestinal barrier integrity, microbial metabolites, immune signaling, vagal communication, tryptophan metabolism, bile acid transformation, and modulation of glial activity.

The available evidence supports a plausible relationship between intestinal microbial imbalance and neuroinflammatory processes. Persistent systemic inflammation and altered microbial metabolism may influence microglial and astrocytic activity and consequently affect neuronal communication and cognitive performance.

However, the relationship is bidirectional and highly individualized. Age, diet, medications, physical activity, metabolic health, psychological stress, sleep, and pre-existing neurological conditions can all modify the gut–brain connection. Therefore, microbiota alterations should not be interpreted as an independent cause of cognitive impairment without considering the broader physiological context.

Future therapeutic strategies may include dietary optimization, targeted prebiotics, selected probiotic strains, synbiotic formulations, microbiota-derived metabolites, and other personalized interventions. Nevertheless, stronger longitudinal and randomized clinical evidence is required before these approaches can be routinely recommended for prevention or treatment of cognitive disorders.

A deeper understanding of microbial metabolism and host–microbe interactions may ultimately enable the development of personalized strategies aimed at reducing chronic inflammation and supporting brain health. The gut microbiota should therefore be viewed not simply as a digestive component but as an active biological partner capable of influencing systemic and neurological homeostasis.

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