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THE ROLE OF GUT MICROBIOTA IN AGING RELATED DISEASES: A NARRATIVE REVIEW

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ABSTRACT

Background: Gut microbiota influences metabolic, immune and neuroendocrine functions throughout life. In older adults, microbiota instability and loss of beneficial taxa are associated with chronic low-grade inflammation and an increased risk of aging related diseases.

Aim: To summarize current evidence on the role of gut microbiota in aging related diseases and to review microbiota targeted interventions in older adults.

Methods: A narrative review was performed. PubMed, Scopus, Web of Science and Google Scholar were searched for full text peer reviewed articles in English published between 2017 and 2025. Keywords included gut microbiota, aging related diseases, inflammaging, frailty, longevity and microbiota interventions. Sixty four studies met the inclusion criteria. Only English peer reviewed studies were considered and animal only studies were excluded.

Results: The literature shows consistent associations between dysbiosis, reduced short chain fatty acids, increased intestinal permeability and systemic inflammation in older adults. Altered microbial profiles correlate with frailty, neurodegenerative, gastrointestinal, musculoskeletal and cardiometabolic disorders. Interventions such as probiotics, prebiotics, dietary modification and fecal microbiota transplantation demonstrate potential to restore microbial diversity, improve metabolic and inflammatory markers and support cognitive and physical functions.

Conclusions: Sixty-four studies showed that aging is associated with reduced microbial diversity, depletion of beneficial SCFA-producing genera and enrichment of pro-inflammatory taxa. These changes correlated with frailty, inflammation and higher prevalence of neurodegenerative, gastrointestinal, musculoskeletal and cardiometabolic

disorders. Microbiota-targeted interventions, including probiotics, prebiotics, diet and fecal microbiota transplantation improve microbial composition and metabolic and immune parameters in older adults.

Gut microbiota is an important modifiable factor in aging related diseases. Microbiota based interventions may contribute to healthy aging, but evidence remains preliminary. Large clinical studies in older adults are required to determine long term safety and effectiveness.

Keywords: gut microbiota, aging, older adults, dysbiosis, frailty, inflammation

INTRODUCTION

Human gut microbiota consists of a diverse and dynamic group of microorganisms located in the gastrointestinal tract. It is crucial for host health through regulation of metabolism, immune system, and intestinal barrier properties [1, 2, 3]. Disturbances during life stages, involving antibiotic use, environmental exposure, inappropriate diet or inadequate diversification of microorganisms often results in an increased risk of inflammatory and metabolic disease later in life. Disrupted microbiota is capable of forming lifelong consequences to health [4, 5]. In older people, reduction in beneficial bacteria and increase in potentially pathogenic species is common [6]. Such shifts might lead to dysbiosis and result in inflammation and predispose to age-related pathologies [7]. Moreover, changes in gut microbiota composition correlate with frailty in older adults [6]. The term "age-related diseases" (ARDs) and "aging-associated diseases" includes chronic illnesses primarily developing in elderly humans and associated with aging [8]. Geriatric health is affected by gut microbiota because it plays a crucial role in metabolizing nutrients, immune system and gut-brain axis, while aging deranges the gut microbiome and leads to inflammaging [9, 10]. Recent studies establish a connection between gut microbiota in older adults and metabolic, digestive, neurological and cardiovascular diseases [11, 12]. The aim and objective of this study is to further clarify the role played by gut microbiota in aging-related diseases. Understanding the implications is essential to broaden therapies towards healthy aging and possible chances for diverse anti-aging therapies targeting gut microbiota.

RELEVANCE

The growing proportion of older adults and the rising prevalence of cardiometabolic, neurodegenerative and inflammatory conditions have intensified interest in the gut microbiota as a modifiable factor influencing healthy and pathological aging. Despite rapid scientific progress, current evidence remains fragmented. Studies report heterogeneous microbial patterns, inconsistent mechanistic explanations and limited clinical validation across different age-related diseases. There is no consensus profile of the aging microbiota and no unified interpretation of its role in systemic inflammation, frailty and multimorbidity. This creates a clear need to synthesize recent findings and assess the therapeutic potential of microbiota-targeted strategies in older adults.

NOVELTY

This review integrates recent evidence published between 2017 and 2025, with particular emphasis on the most current studies. It compares microbial alterations across multiple age-related disease groups and highlights the gut microbiota as both a potential biomarker and a therapeutic target for healthy aging. The review also summarizes emerging microbiome-based interventions in older adults and outlines their clinical implications.

AIM

To clarify the role of gut microbiota in the development and progression of aging-related diseases and to evaluate microbiota-targeted interventions relevant to older adults.

Research objectives:

1. To describe characteristic microbiota alterations in older adults
2. To summarize links between dysbiosis and major groups of aging-related diseases
3. To outline the mechanisms underlying inflammation and metabolic dysfunction
4. To review current microbiota-modulating interventions and their reported effects

METHODOLOGY

This article was designed as a narrative review. The literature search was carried out in PubMed, Scopus, Web of Science and Google Scholar. The time frame covered publications from 2017 to 2025.

Only full-text, peer-reviewed publications in English were considered. Representative search strings included the PubMed query (“gut microbiota” AND “aging” AND “age-related diseases”) AND (“inflammaging” OR “frailty” OR “older adults”), and Scopus query TITLE-ABS-KEY (“gut microbiota” AND “older adults” AND “inflammaging”). As this

was a narrative review, no formal risk-of-bias or quality assessment tools were applied; articles were selected based on thematic relevance and methodological clarity.

The synthesis prioritizes studies published between 2021 and 2025, with earlier literature incorporated selectively to support conceptual continuity, mechanistic framing, context of the field and important previous studies. The following keywords and their combinations were used: "gut microbiota alterations", "aging-related diseases", "chronic low-grade inflammation (inflammaging) in elderly", "Implications of the Gut Microbiota in Aging-Related Diseases", "Frailty in Older Adults", "achieving extreme longevity by centenarians", "Interventions targeting the gut microbiota". In the initial screening phase, 125 studies were evaluated. After applying predefined inclusion and exclusion criteria and assessing thematic relevance, 64 articles were ultimately included in the qualitative synthesis. Inclusion criteria were full-text, peer-reviewed publications in English, original studies, clinical trials, case series, systematic and narrative reviews, mechanisms, diagnosis, treatment, prevention, or outcomes of Aging-Related Diseases. PRISMA procedures and flow diagrams were not applied because the review was narrative and not designed as a systematic analysis. Exclusion criteria were articles dealing exclusively with animal-only trials, young adult populations, non-microbiota-focused interventions, editorials, were excluded. Full texts of potentially relevant papers were reviewed, and disagreements were resolved by consensus. References from selected articles were also screened to identify additional relevant studies. Data were organized by aligning results within convergent thematic domains and critically evaluating the degree of consistency and reproducibility across the included studies. The extracted information was organized into thematic domains: Age-Related changes in Gut Microbiota of Older Adults, Frailty, Achieving extreme longevity by centenarians, Inflammation, Implications of the Gut Microbiota in Aging-Related Diseases and Interventions to modulate Gut Microbiota in Older Adults. Findings were summarized descriptively and compared across studies to highlight areas of consensus, controversy, and knowledge gaps.

RESULTS

1.AGE-RELATED CHANGES IN THE GUT MICROBIOTA OF OLDER ADULTS

Throughout the human life cycle, gut microbiota evolves dynamically in accordance with physiological need and environmental influence, where typically specific dominant bacterial groups vary as a consequence of type of childbirth delivery, diet, medication, and ageing [5, 13]. By adult life, gut microbiota is relatively constant, consisting of mostly butyrate-producing and anti-inflammatory genera like Faecalibacterium prausnitzii, Roseburia, and Ruminococcus, regulating functions related to intestinal and systemic health by producing short-chain fatty acid (SCFA). Older persons are more likely to exhibit mainly species of Proteobacteria and Enterobacteriaceae, and the Bacteroidetes to Firmicutes ratio is higher as well [1, 3, 6, 13]. These alterations are typically linked to decreases in bacteria that produce SCFA. There is a further alteration in older people in the form of lower concentration of Bifidobacterium and Lactobacillus [2, 10, 14].

1.1 Frailty in Older Adults

Impaired microbiota is linked to frailty syndrome, weakness and other dysfunctions of various parts of the body. Frail older adults exhibited decreased diversity of gut microbiota, increased levels of markers of inflammation, disturbed composition of gut microbiota, increased content of potentially pathogenic genera, and decreased content of commensal beneficial bacteria [15]. Older adults with frailty often exhibit increased levels of IL-6, TNF-α, and CRP, which correlate with reduced gut resilience. These disturbances interfere with intestinal barrier integrity, cause gut permeability ("leaky gut") and provoke systemic low-grade inflammation (inflammaging) [16, 17]. Composite results reveal that at phylum level, frailty was characterized by increased relative abundance of Actinobacteria and Proteobacteria and decreased Firmicutes and Fusobacteria, reflecting higher inflammation and reduced SCFA production [Table 1]. At genus or species level, concordant results showed that abundances of butyrate-producing bacteria that are involved in gut health such as Akkermansia, Prevotella, Faecalibacterium prausnitzii, Clostridium cluster XIVa, and Roseburia were decreased in frail older adults to significant degrees, their depletion is a sign of gut dysbiosis in frailty [6, 10, 18, 19]. Studies suggest that there may be roles for members of the Coriobacteriaceae, which appear to form a pro-inflammatory group of other bacteria [18, 20, 21].

Table 1. Microbiome bacterial abundance in older adults with frailty.

Taxonomic Level	Higher in Frailty	Lower in Frailty	Inflammatory Role	Reference number
Phylum	Actinobacteria, Proteobacteria	Firmicutes, Fusobacteria	↑Inflammaging	[1, 3, 6, 15, 16, 17]

Genus	Klebsiella, Parabacteroides	Faecalibacterium, Roseburia, Prevotella, Akkermansia	Barrier dysfunction	[6, 10, 18, 19]
Family	Coriobacteriaceae, Enterobacteriaceae	Ruminococcaceae	↑IL-6, CRP, TNF-α	[18, 20, 21]
Metabolites	↑ LPS-endotoxins	↓ Butyrate, SCFA	Systemic inflammation	[1, 3, 6, 10, 13, 16, 17]

↓lower; ↑higher; IL-6 -interleukin 6; CRP-C-reactive protein; TNF-α-Tumor necrosis factor α; LPS-endotoxins- Lipopolysaccharide endotoxins; SCFA-short-chain fatty acids;

1.2 Gut Microbiota and achieving extreme longevity by centenarians

Studies of centenarians and very long-lived individuals reveal distinct gut microbiota signatures which show higher stability, resilience, taxonomic anti-inflammatory richness (Coprococcus, Faecalibacterium, Bifidobacterium, Akkermansia, Lactobacillus) and higher SCFA production [22, 23]. Their gut microbiota is characterized by increased potential for butyric acid production and antioxidant effect. These traits are linked to reduced systemic inflammation and resistance to age-related diseases, which would suggest the part the microbiota plays in extending an individual's lifespan [22, 24]. Gut microbiota compositions could be used as a biomarker in geriatric medicine, enhancing quality of life in the elderly, healthy aging and record longevity [10, 25].

2.INFLAMMATION

Age-associated dysbiosis in gut microbiota composition and metabolites contribute to increased gut permeability, imbalanced immunity and chronic low-grade inflammation, marked by elevated inflammatory mediators and activation of inflammatory pathways [26, 27]. Low-grade, chronic inflammation is often seen in aging and known as "inflammaging." Inflammaging is linked to immunosenescence and an imbalanced gut (called dysbiosis) and contributes to age-related disorders. It is a syndrome of "homeostatic frailty" and appears as a trait of the elderly. It is causing uncontrolled development of chronic diseases linked to frailty [15, 16]. 'Immunosenescence' is a decline in adaptive immune function-caused by impairing T-cell differentiation and reducing regulatory T cells (Tregs). Dysbiosis increases intestinal permeability ("leaky gut"), allowing translocation of pathogen-associated molecular patterns (PAMPs), including LPS and pro-inflammatory cytokines (e.g., TNF-α, IL-6) into systemic circulation. This triggers chronic low-grade inflammation, endothelial dysfunction, and disruption of metabolic homeostasis [26, 27, 28, 29]. Declines in SCFA synthesis impairs anti-inflammatory signaling gut barrier integrity and anti-inflammatory signaling. The high degree of inflammatory mediators lowers the blood-brain barrier integrity, increases intestinal permeability and causes an inflammatory state, cognitive decline, and susceptibility to ARDs [30, 31].

3.IMPLICATIONS OF THE GUT MICROBIOTA IN AGING-RELATED DISEASES

Recent studies highlight, gut microbiota has an impact on many disorders in the elderly. There are several aging-associated diseases related to gut microbiota such as neurodegenerative diseases, gastrointestinal diseases, musculoskeletal diseases and cardiometabolic diseases, making it a key factor in both the onset and management of age-associated diseases [1, 2, 3, 8, 11]. The main characteristics and scope of the included studies are summarized in Table 2.

3.1. Neurodegenerative disorders

Alzheimer's disease (AD) is considered to be the most common neurodegenerative condition and type of dementia among elderly people. AD patients show reduced SCFA-producing genera (Bifidobacterium, Faecalibacterium, Roseburia, Clostridiaceae) and increased pro-inflammatory taxa (Escherichia/Shigella, Bacteroides, Alistipes) [2, 11, 21, 31]. Dysbiosis contributes to neuroinflammation via gut–brain axis signaling, blood–brain barrier (BBB) compromise, microglial activation, and amyloid-beta accumulation. Microbial metabolites such as trimethylamine N-oxide (TMAO), delta-valerobetaine and exacerbate systemic and cerebral inflammation and impaired short-term and spatial memory function [27, 31, 32, 33, 34].

Parkinson's Disease (PD) is the second most common neurodegenerative disorder. Recent research has identified that patients exhibit a distinctive gut microbiota pattern referred to as "pro-inflammatory dysbiosis", which may induce neuroinflammation by α-synuclein misfolding [2, 8, 11, 27]. The microbiota shows reduced diversity and change in the composition of dominant and common types. Especially, there is an increase in Proteobacteria and Bacteroidetes and decrease in Firmicutes and Actinobacteria, a pattern that resembles results in AD. PD microbiota is enriched with

mucin-degrading and pro-inflammatory taxa like Akkermansia, Escherichia/Shigella and Klebsiella. Consequently, there is a substantial depletion of beneficial SCFAs [31, 34, 35]. These changes correlate with both motor features impairment (e.g., postural instability, gait difficulty) and non-motor features (e.g., cognitive impairment, constipation) [36]. More importantly, the PD-associated microbiota also disrupts neurotransmitter homeostasis by affecting GABA, dopamine, and noradrenaline synthesis while inducing systemic inflammation through pro-inflammatory cytokines, TMAO, delta-valerobetaine, isovaleramide (IAA) [36, 37, 38, 39].

3.2. Gastrointestinal disorders

Aging gut microbiota contributes importantly to the etiology and pathogenesis of gastrointestinal diseases like Inflammatory Bowel Disease (IBD) and Irritable Bowel Syndrome (IBS). In IBD patients the changes in the microbiome exhibit decreased Firmicutes (especially Clostridium spp.) and SCFA-producing bacteria and overgrowth of pro-inflammatory taxa like Proteobacteria (such as Escherichia coli and Klebsiella pneumoniae), which further exacerbate mucosal barrier dysfunction, enhance inflammatory activity and impairs epithelial repair [2, 13, 30, 40]. Simultaneously, this leads to decreased levels of butyrate. impairs colonocyte function, disrupts intestinal barrier, and increases gut permeability, leading to translocation of LPS and PAMPs into the circulation. This mechanism is commonly referred to as "leaky gut." [4, 13, 30]. Similarly, in IBS, age-related microbial changes exacerbate typical symptoms including abdominal pain, bloating, and altered bowel habits, through dysregulated fermentation, gut motility, and immune responses [4, 28].

3.3. Musculoskeletal disorders

Gut microbiota dysbiosis is found to be an important etiological cause of age-associated musculoskeletal disruptions, such as osteoporosis and sarcopenia. Aging is associated with low microbial diversity, loss of Firmicutes and increased pro-inflammatory bacteria such as Proteobacteria and Bacteroidetes, promoting osteoclastogenesis and systemic inflammation [10, 33, 41, 42]. All these lead to faulty production of anti-inflammatory butyrate and other SCFAs whose constitutive role preserves the integrity of colonocytes, regulates calcitriol metabolism and enhances absorption of calcium [11, 42]. Dysbiosis within the gut microbiota disrupts the balance of bone modeling, initiates the osteoclast differentiation due to overexpression of the RANKL, with a bias towards bone loss rather than bone absorption, leading to reduced bone mass. Inflammatory cytokines lead to low bone mineral density and fracture risk [41, 42]. Beneficial taxa (Faecalibacterium, Bacteroides) correlate with better muscle mass, strength, and mitochondrial function, while overgrowth of pro-inflammatory taxa exacerbates muscle loss and oxidative stress [41, 43, 44].

3.4. Cardiometabolic disorders

The microbiota of the gut and its metabolites have been identified as a possible major driver of cardiometabolic disease (CMD) pathogenesis, including diabetes, hypertension, and atherosclerosis. Low-grade systemic inflammation, increased intestinal permeability, and metabolic endotoxemia are linked to imbalances and decreased diversity in microbiota and are major drivers of CMD development [2, 45, 46]. Gut microbiota dysbiosis in CMD disorders has also been shown to present reduced microbial diversity and higher Firmicutes/Bacteroidetes ratio [11, 28].

The dysbiosis in type 2 diabetes patients is characterized by lower abundance of of protective SCFA-producers (Faecalibacterium prausnitzii, Roseburia hominis, Eubacterium rectale) and higher abundance of pro-inflammatory taxa (Ruminococcus, Fusobacterium, Desulfovibrio, and Akkermansia muciniphila) [47, 48, 49]. The dysbiosis in type 1 diabetes patients is associated with higher abundance of Bacteroides, Bifidobacterium, Escherichia, Veillonella and Bacteroides, intestinal hyperpermeability, endotoxemia, β-cell dysfunction and insulin resistance induction [7, 47, 50]. Lipopolysaccharide evoked inflammation and lowered SCFAs levels (e.g. butyrate) impaired insulin signaling and β-cell function [47, 51]. Therapeutic glucose-lowering, improved insulin sensivity and anti-inflammatory effects are produced by strains like Bifidobacterium adolescentis, B. bifidum and Lactobacillus rhamnosus [52].

Hypertensive and atherosclerotic patients exhibit reduced microbial diversity, overgrowth of pro-inflammatory genera (Klebsiella, Escherichia, Streptococcus) and loss of the SCFA's producers (Roseburia, Faecalibacterium, Bacteroides, Ruminococcaceae, Akkermansia), involved in blood pressure regulation and suppress atherosclerotic plaque through inhibition of metabolic endotoxemia-induced inflammation [53, 54, 55, 56, 57]. Microbial metabolites like TMAO exacerbate vascular inflammation and contribute to arterial stiffness, thrombosis, cardiovascular events and are pro-hypertensive [54, 56, 57, 58]. Additionally, LPS and other bacterial compounds translocate from the intestine through increased gut permeability and cause inflammation provoking vascular damage, arterial stiffness and vascular aging [55, 57].

Table 2. The summary of Gut Microbiota Alterations and Aging-Related Diseases.

Aging-Related Disease	Key Microbiota Alterations	Main mechanisms	Clinical implications	Reference number
Frailty	↓ Diversity, ↓ Firmicutes, ↓ Fusobacteria ↑ Proteobacteria, ↑ Actinobacteria,	↓ SCFAs, ↑ IL-6, ↑TNF-α, leaky gut	Systemic inflammation	[1, 3, 6, 10, 13, 15, 16, 17]
Neurodegenerative	↓ Faecalibacterium, ↓ Roseburia. ↑ Escherichia/ Shigella, ↑Alistipes	↓SCFAs, ↑LPS, BBB destabilization,microglial activation	Neuroinflammation, amyloid deposition, α-synuclein aggregation	[2, 11, 21, 27, 31, 32, 33, 34, 35]
Gastrointestinal	↓ Clostridium, ↓ Lactobacillus, ↑ Proteobacteria	↓Butyrate, ↑Th1/Th17 inflammation	Mucosal injury, gut permeability ("leaky gut")	[2, 4, 13, 30, 40]
Musculoskeletal	↓ Faecalibacterium, ↓Roseburia, ↑ Proteobacteria	↓ Butyrate, ↑ RANKL, ↑ IL-6, ↑ TNF-α	Bone loss, muscle atrophy, oxidative stress	[10, 11, 33, 41, 42]
Cardiometabolic	↑Firmicutes/ Bacteroidetes ratio, ↑Enterobacteriaceae	↓ SCFAs ↑ TMAO, ↑ LPS endotoxemia,	Insulin resistance, hypertension, endothelial dysfunction	[47, 48, 49, 50, 51, 52, 53, 54, 55, 56, 57]

↓- lower; ↑- higher; RANKL-apoptosis regulator gene; IL-6- interleukin 6; TNF-α-tumor necrosis factor; SCFAs-short fatty acids; TMAO-Trimethylamine N-oxide; Th1/Th17-T helper cell 1/T helper cell 17; BBB destabilization-blood-brain barrier destabilization; LPS endotoxemia- Lipopolysaccharide endotoxemia;

4. INTERVENTIONS TO MODULATE GUT MICROBIOTA IN OLDER ADULTS

Gut microbiome therapies, such as probiotics, prebiotics, dietary interventions and fecal microbiota transplantation (FMT), show therapeutic potential in modulating gut microbiota, enhancing metabolic health and mitigating age-related diseases [1, 3, 11, 29].

Probiotic interventions such as Lactobacillus and Bifidobacterium species restore intestinal barrier function and boost production of SCFAs, reducing systemic inflammation, decreasing endotoxemia, pro-inflammatory cytokines and modulating immune responses and metabolic pathways [11, 13, 29, 34]. These effects also resulted in a reduction in the level of TMAO, a cardiovascular risk metabolite. Supplementation duration was correlated with improvement in markers of metabolic syndrome, reduction in blood pressure, lipid profile and and improving sensitivity to insulin and glucose metabolism [13, 14, 27, 34, 59].

Prebiotics like inulin, fructooligosaccharides (FOS), galactooligosaccharides (GOS), resistant starch selectively stimulate growth of beneficial microbes and boost production of SCFAs and restore intestinal barrier function. Prebiotics like polyphenols create neuroprotective effects, while combination of FOS and inulin may also boost cognitive function. Galactooligosaccharides promote growth of other beneficial microbiota like Bacteroides and Bifidobacteria [31, 59].

Another therapeutic application is a combination of synbiotics, probiotics and prebiotics simultaneously, which demonstrates synergy between these compounds. Supplementation by synbiotics reduces leaky intestine, neuroinflammation, frailty and improves healthy aging [44, 59].

Diet determines composition of beneficial and pathogenic microorganisms, regulates production and modulation of gut microbiome and directly influences inflammation, gut barrier integrity and age-related disease prevention and management. Fiber-rich diets (e.g., inulin, fructooligosaccharides), resistant starch-favor and Mediterranean diet enrichment with SCFA-producing species (e.g., Faecalibacterium prausnitzii, Roseburia species), with anti-inflammatory effect, enhanced glucose sensitivity and healthy intestinal microbiome [8, 10, 11, 14, 27].

Probiotic-like effect with enhanced bacterial diversity and metabolic parameters is also shown by polyphenol-dense foods (e.g., berries, cocoa and green tea) [60, 61]. The Mediterranean diet (high consumption of olive oil, legumes,

whole cereals, fruits, vegetables and healthy fats), enhances health and inhibits synthesis of TMAO and is associated with improved cardiovascular health, neurocognitive performance, reduced muscle wasting and reduced gut inflammation [11, 27, 61].

On the contrary, the Western diet, high in saturated fats and processed food with added sugars, favors dysbiosis through expansion of pro-inflammatory species (e.g., Escherichia coli and Clostridia), decreases SCFAs production, increases endotoxemia, raises TMAO, leads to insulin resistance and systemic inflammation. Reduced consumption of saturated fats and added sugars helps to prevent dysbiosis [8, 31, 61, 62].

By restoring a healthy and varied gut microbiota, fecal microbiota transplantation (FMT) has shown itself to be an effective intervention supporting healthy aging and improving metabolic health. It has been discovered that transferring gut microorganisms from healthy, typically younger donors into elderly or metabolically challenged receivers can restore the gut microbiota balance by increasing both beneficial bacteria and microbial diversity [63]. This increase in beneficial taxa results in lowering overall inflammation and modifies metabolic functions, increased SCFA production, reduced TMAO and potentially delay age-related cognitive and motor function [11, 27, 64]. Clinical application challenges include donor selection, standardization of transplant procedures, regulatory and safety problems such as transmission of infection and the need for more long-term data reviews.

DISCUSSION

The findings of this literature review collect and summarize the role of gut microbiota composition and functionality in modulating the pathophysiology of aging-related diseases (ARDs), including metabolic, cardiovascular, and neurodegenerative disorders. The present review highlights that age-associated dysbiosis, characterized by reduced microbial diversity, depletion of beneficial taxa and overrepresentation of pro-inflammatory species correlates with systemic inflammation, metabolic dysfunction and frailty [1, 2, 3, 11, 29, 31, 44].

In metabolic aging, evidence supports a bidirectional relationship between gut microbial alterations and insulin resistance, obesity and diabetes mellitus [3, 5, 11]. Dysbiotic microbiota can influence host glucose metabolism via modulation of short-chain fatty acid (SCFA) production, bile acid signaling, and endotoxin-induced inflammation. Similarly, gut-derived metabolites such as trimethylamine N-oxide (TMAO) have been implicated in atherosclerosis and vascular aging, suggesting that microbial metabolism directly contributes to cardiovascular disease progression [10, 13, 50, 53, 54].

Neurodegenerative aging presents another critical link between gut microbial imbalance and disease onset. Studies have associated alterations in gut microbiota with cognitive decline, neuroinflammation, and disrupted gut–brain axis signaling in conditions such as Alzheimer's and Parkinson's disease [6, 7, 31, 32, 36, 37]. The mechanisms proposed include increased intestinal permeability ("leaky gut"), systemic inflammation, and altered tryptophan-serotonin pathways [16, 17, 26]. Despite strong associative evidence, causality remains difficult to establish. Many reviewed studies are observational and rely on small or heterogeneous cohorts, making it challenging to differentiate cause from consequence. Furthermore, interindividual variability in diet, genetics, medication use, and environment confounds the generalizability of findings [9, 10, 11].

Another limitation concerns the inconsistent methodology for microbiota profiling, as varying sequencing technologies and bioinformatic pipelines yield divergent taxonomic results. Notably, intervention studies indicate that modulation of the gut microbiota holds potential in mitigating aging-related pathologies [11, 27, 59, 61, 64]. However, some studies combining approaches to explain the temporal relationship between microbiological changes and ARDs progression are warranted and show consistent study results. Overall, the current evidence positions the gut microbiome not merely as a biomarker of aging. Future research should also investigate whether maintaining microbiome diversity throughout life can prevent biological aging.

This review has several limitations. Most included studies were observational and based on small or heterogeneous cohorts, limiting causal inference. Differences in diet, medication use, genetics and comorbidities reduce comparability across studies. Microbiota profiling relied on variable sequencing platforms and bioinformatic pipelines, which may partly explain divergent taxonomic findings. As this was a narrative review, no formal quality assessment was performed. Future studies should include randomized controlled trials in older adults, long-term follow-up of microbiota-targeted interventions, and integrated profiling combining metagenomics, metabolomics and immune markers. Validation of microbiota-based biomarkers for frailty and multimorbidity is required.

CONCLUSIONS

Recent findings underscore the therapeutic promise of targeting specific gut microbial taxa to modulate pathways implicated in the onset and progression of aging associated disorders. In particular, an elevated Firmicutes/Bacteroidetes ratio and microbiota instability may serve as early biomarkers for the development of age related pathologies, highlighting the diagnostic potential of microbiome profiling. As a result of this shift the research shows a reduction in key beneficial SCFAs and increases susceptibility to many diseases in older adults. Notably, frail seniors

exhibit depletion of major butyrate producers, underscoring their role as biomarkers and potential modulators. Studies of centenarian microbiomes reveal distinct structures characterized by greater taxonomic richness, stability and enrichment of anti-inflammatory genera, which may contribute to exceptional longevity.

Interventions including probiotics and prebiotics, fecal microbiota transplantation and special diet have been shown to reestablish microbial homeostasis, dampen pro inflammatory signaling cascades, and improve cognitive, motor and metabolic functions. A high fiber-rich and plant-based Mediterranean diet supports beneficial microbial communities, improves microbiota diversity, reduces inflammation, diet is associated with lower TMAO levels and reduced inflammatory profiles in older adults. The expanding field of microbiota based therapeutics holds the potential to revolutionize preventive and restorative strategies for older adults, however it demands further refinement to ensure both effectiveness and safety. Integrative protocols combining precision dietary modulation, personalized treatment regimens, and standardized FMT procedures are required to deliver truly comprehensive care. Crucially, future research must focus on individualizing treatments according to each patient's microbial and metabolic profile, and on conducting long term, large scale interventional trials capable of establishing causal relationships and assessing the durability of clinical benefits. Only through such rigorous evaluation can microbiome targeted approaches be optimized for healthy aging and extended healthspan. It is important to emphasize the significant role of intestinal microbiota in human health, as it is a promising therapeutic target and is therefore currently the subject of interest of many research centers.

Although current evidence supports the therapeutic relevance of microbiota-targeted strategies, several challenges remain. Most available studies are observational, include small or heterogeneous cohorts and apply different sequencing and analytical methods, which limits comparability and causal interpretation. Standardization of microbiome profiling, donor selection procedures for FMT, and safety monitoring remains necessary before routine clinical use. Large randomized controlled trials in older adults with long-term follow-up are required to determine durability of clinical benefits and to confirm whether microbiota modulation can truly slow biological aging.

DISCLOSURE

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The authors declare no conflict of interest.

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The authors used artificial intelligence tools to assist with language editing and structural refinement. All AI-assisted

content was reviewed and revised by the authors to ensure that its use did not influence the scientific integrity or substantive content of the work. Artificial intelligence was not used to extract data from primary studies, all information was checked manually.

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