Osteoporosis International (2026) 37:281–304
https://doi.org/10.1007/s00198-025-07801-w

REVIEW

Impact of probiotic, prebiotic, and synbiotic supplementation on the gut microbiome in older adults with sarcopenia, obesity, and sarcopenic obesity

Anoohya Gandham[1,2] · Konstantinos Prokopidis[3] · Costas Glavas[2,4] · David Scott[2,4] · Mattias Lorentzon[5,6]

Received: 3 September 2025 / Accepted: 1 December 2025 / Published online: 8 January 2026

Abstract

Gut microbiome plays an important role in several metabolic, immune, and inflammatory pathways; however, there is limited evidence for its role in body composition and musculoskeletal health. Sarcopenia, defined as a loss of skeletal muscle mass and function, and obesity, can co-exist in a condition known as sarcopenic obesity. This condition is highly prevalent among older adults, hence increasing the risk of negative health implications such as metabolic dysfunction, chronic inflammation, reduced physical performance, and poor quality of life. These age-related conditions are closely associated with alterations to the gut microbiome, including microbial profiles and a reduction in beneficial metabolites such as short-chain fatty acids (SCFAs). Probiotic, prebiotic, and synbiotic interventions are therefore emerging as promising strategies to improve the gut microbiome by enhancing microbial diversity and restoring microbial communities. This review utilizes current evidence on the impact of these interventions on gut microbiota composition, inflammatory and metabolic biomarkers, body composition, and functional outcomes in older adults with sarcopenia, obesity, and sarcopenic obesity. Probiotics, containing live beneficial microorganisms, have shown potential in enhancing SCFA production, reducing inflammation, and improving insulin sensitivity. Prebiotics are non-digestible fibers that selectively activate the growth of beneficial gut bacteria, further supporting gut health by proliferating the growth of SCFA-producing bacteria. Synbiotics, a combination of probiotics and prebiotics, provide a synergistic approach to gut health, accounting for the microbial composition and functional capability. Recent studies have demonstrated that probiotics, prebiotics, and synbiotics may reduce inflammation and improve muscle mass and strength among older adults with sarcopenia, obesity, and sarcopenic obesity. These interventions have the potential in mitigating obesity-related metabolic dysfunction and inflammation, particularly in individuals with sarcopenic obesity. Although, preclinical studies in mice exhibit beneficial effects, clinical studies in older adults remain limited, with heterogeneity of study design, intervention types, and outcome measures. This review highlights the need for robust, well-designed clinical trials to understand the mechanistic and molecular pathways through which probiotic, prebiotic, and synbiotic supplementation may modulate the gut microbiome and improve musculoskeletal health among older adults. These interventions may provide innovative, non-invasive therapeutic strategies for managing sarcopenia, obesity, and sarcopenic obesity, ultimately contributing to healthier aging and improved quality of life of older adults. This review also underscores the potential of microbiome-targeted interventions for aging populations, highlighting the need for further research.

Keywords Gut health · Microbiome · Sarcopenia · Sarcopenic obesity

Introduction

Older adults are at increased risk of chronic health conditions including sarcopenia (defined as a loss of skeletal muscle mass and function), obesity, and sarcopenic obesity (defined as the comorbid presence of sarcopenia and obesity) [1–3]. Age-associated declines in muscle mass and increases in fat mass may contribute to an increased risk of metabolic dysfunction [4–6]. Sarcopenia affects approximately 10% of adults aged 50 years and older, with its prevalence increasing to over 50% in individuals aged 60 years and older [7–11]. Obesity is another condition that is highly prevalent in aging populations, affecting over 40% of older adults globally [12]. Sarcopenic obesity is associated with a two- to threefold increased

---

Extended author information available on the last page of the article risk of disability, reduced quality of life, and premature mortality [13–15]. These conditions are influenced by several multifactorial processes, such as increased inflammation, metabolic abnormalities, and age-related hormonal changes [16, 17].

Recent evidence suggests that the gut microbiome is a dynamic community comprised of approximately 100 trillion microorganisms residing in the gastrointestinal tract, playing a pivotal role in the human body [18–20]. In older adults, age-related shifts in gut microbiota are commonly observed and are characterized by reduced alpha diversity, decline in beneficial taxa, and an increased presence of pathogenic microbes. These changes are associated with increased risk of chronic low-grade inflammation, impaired insulin sensitivity, and anabolic resistance [21, 22] contributing to muscle loss and fat accumulation, increasing the risk of sarcopenic obesity. Emerging studies in older adults with sarcopenic obesity have demonstrated distinct differences in gut microbiota diversity and composition compared to healthy counterparts [23–25].

Moreover, aging is associated with a decline in gut microbiota abundance and diversity, particularly an observed reduction in short-chain fatty acid (SCFA)-producing bacterial strains such as butyrate, acetate, and propionate, which are crucial for the maintenance of muscle function and overall metabolic health [18, 26]. This reduction in these SCFAs which is likely driven by a loss of beneficial microbial species, negatively impacts gut barrier integrity, inflammation regulation, and energy metabolism, thereby contributing to increased risk of developing sarcopenia, obesity, sarcopenic obesity, and metabolic dysfunction in older adults [27].

Given the gut microbiome's critical role in overall health, research and clinical interventions aimed at its modulation are increasing [28, 29]. Probiotics, prebiotics, and synbiotics are among the most commonly investigated approaches shown to regulate gut microbiota composition and function, improving metabolic health, such as insulin sensitivity and lipid metabolism, and musculoskeletal health outcomes, including bone mineral density and muscle mass and strength [30]. Probiotics are live microorganisms that are associated with several health benefits including enhanced gut barrier function, reduced inflammation, and increased production of SCFAs that promote muscle health [31]. Prebiotics are indigestible dietary fibers that selectively promote the growth of beneficial gut bacteria, improving microbial diversity and metabolic function [32]. Synbiotics are a combination of probiotics and prebiotics, creating a synergistic effect as they are simultaneously providing live microorganisms and the substrates needed to thrive [33]. Additionally, emerging evidence supports postbiotics, which are non-viable microbial cells conferring several health benefits to the host. However, since there is currently limited availability of studies specifically targeting postbiotics among older adults with sarcopenia and sarcopenic obesity, this review does not discuss postbiotics in detail.

Despite encouraging results from pre-clinical studies, the specific effects of these interventions on the gut microbiome and their roles on muscle health, body composition, and metabolic outcomes in older adults remain poorly established [30]. With an aging population, there is a greater need to have an understanding of these relationships, particularly in the context of sarcopenia, obesity, and sarcopenic obesity [34]. A recent meta-analysis provided an important foundation on the effects of probiotic supplementation on overall muscle outcomes including muscle mass, strength and lean body mass across randomized controlled trials [35]. Interactions between the gut microbiota and skeletal muscle, along with gut-mediated metabolic regulation, may therefore have therapeutic potential for mitigating these conditions. However, this meta-analysis was primarily focused on overall probiotic effects without distinguishing underlying health conditions, microbiota profiles, or the combined metabolic and muscular impairments [35].

This narrative review critically evaluates existing evidence on the effects of probiotic, prebiotic, and synbiotic supplementation on the gut microbiome in older adults with sarcopenia, obesity, and sarcopenic obesity. Through the synthesis of existing literature, this review aims to provide a foundation for future research and inform targeted therapeutic strategies to address these important health challenges in aging populations.

Methodology

This narrative review was based on a literature search of PubMed and Scopus databases up to October 2025. The search included combinations of the terms "probiotic," "prebiotic," "synbiotic," "gut microbiome," "sarcopenia," "obesity," "sarcopenic obesity," and "older adults." Only human studies published in English were considered, with a focus on trials or observational studies evaluating the effects of these interventions on the gut microbiota in older adults with sarcopenia, obesity, or sarcopenic obesity. In addition to database searches, we examined the reference lists of relevant published articles to identify further studies. Relevant articles were selected based on their relevance to the topic, and the findings were summarized narratively to identify key mechanisms and trends.

Sarcopenia, obesity, and sarcopenic obesity in older adults

The global prevalence of obesity, sarcopenia, and sarcopenic obesity among older adults (aged ≥ 65 years) is rising significantly, challenging healthcare systems worldwide [36, 37]. In 2021, the global prevalence of sarcopenic obesity was 11% and is expected to increase with the aging of the population [37]. The individual and combined impacts of obesity and sarcopenia on health outcomes are particularly important in relation to musculoskeletal health as they have a significant economic burden [38].

Obesity

Obesity is a chronic condition characterized by excessive adiposity increasing the risk of compromised physical health, metabolic function, and overall quality of life [39]. Obesity is commonly defined as a body mass index (BMI $\geq$ 30 kg/m^2) according to the World Health Organization classification [40]. Additionally, obesity can also be defined in terms of body fat percentage, assessed through dual-energy X-ray absorptiometry (DXA) or bioimpedance [41, 42].

Obesity is a major public health concern, with projected steady increase in prevalence, particularly among older adults [43–45]. Older adults with obesity are at an increased risk of developing severe musculoskeletal and metabolic health issues which may lead to an increased risk of falls and subsequent fractures, osteoarthritis, sarcopenia, functional impairment, cardiovascular disease, cancer, and type 2 diabetes mellitus [12, 46]. This underscores the increased healthcare needs of older adults with obesity, therefore emphasizing the importance of implementing targeted interventions to improve health outcomes and reduce overall healthcare costs.

Sarcopenia

Sarcopenia is defined as an age-associated progressive loss of skeletal muscle mass, strength, and function [47]. Recently, a global consensus conceptual definition has been established through the Global Leadership Initiative on Sarcopenia (GLIS), but there is currently no universally-accepted operational definition [4]. The three most widely accepted operational definitions of sarcopenia are the revised European Working Group on Sarcopenia in Older People (EWGSOP2), the Sarcopenia Definition and Outcomes Consortium (SDOC), and the Asian Working Group for Sarcopenia (AWGS) [48–50]. These definitions of sarcopenia reflect differing priorities on the diagnosis and characterization of sarcopenia. The lack of consensus for an operational definition of sarcopenia heightens the complexity of its diagnosis. Regardless of the definition, sarcopenia remains highly prevalent (up to 10–30%) in community-dwelling older adults contributing significantly to physical disability, falls and subsequent fractures, and diminished quality of life [4, 51, 52]. Several studies have shown that different sarcopenia definitions lead to varying prevalence estimates and have different abilities to predict adverse outcomes [53–55]. Recent studies in older women have demonstrated that sarcopenia definitions vary in their ability to predict fractures and injurious falls [56, 57]. These findings suggest that definitions incorporating physical function are more clinically relevant for identifying individuals at greater risk and guiding targeted interventions [56, 57].

Sarcopenic obesity

Sarcopenic obesity is projected to increase in prevalence as both obesity and sarcopenia rates are growing substantially in aging populations [2]. However, due to the lack of a universally accepted operational definition for sarcopenia, there is conflicting evidence surrounding the prevalence and etiology of sarcopenic obesity [58]. The European Society for Clinical Nutrition and Metabolism (ESPEN) and the European Association for the Study of Obesity (EASO) definition of sarcopenic obesity combines low muscle mass and muscle strength with high fat mass, but its application remains inconsistent across studies [59, 60]. The pathophysiology of sarcopenic obesity is thought to involve a combination of poor lifestyle behaviors, chronic inflammation, hormonal dysregulation, and metabolic factors, which creates a "vicious cycle" of deteriorating body composition and function [47]. Targeted interventions are therefore required to address both muscle loss and increased adiposity in older adults with sarcopenic obesity. Recent evidence supports lifestyle interventions focussing on resistance training and diet for the management and reduction of sarcopenic obesity [61, 62].

Gut microbiome and its role in health

The gut microbiome, a dynamic and complex ecosystem of trillions of microorganisms, plays a vital role in maintaining overall health [63]. This microbial community regulates various vital physiological processes, including nutrient absorption, energy metabolism, immune function, and the synthesis of bioactive compounds [64]. However, aging is also accompanied by series of changes in the gut microbiome, that can have crucial negative impacts on health outcomes [64]. Within the scope of this study, unhealthy aging, defined as chronic low-grade inflammation, anabolic resistance, reduced mobility, and functional decline is associated with gut microbiota alterations, including an expansion of potentially pathogenic taxa (Table 1) [77, 78]. Conversely, healthy aging is associated with the maintenance of healthy bacteria (Table 1) [63, 72, 79]. These changes are a result of age-associated dietary shifts, reduced physical activity, increased medication use, and a weakened immune system [63]. These gut microbiota alterations may contribute to several age-related conditions, including chronic low-grade inflammation, also known as inflammaging, which is a key contributor to sarcopenia, obesity, and metabolic dysfunction [63].

In older adults, the gut microbiome exerts its effects predominantly through the production of microbial metabolites [63]. SCFAs, such as butyrate, acetate, and propionate have a key role in supporting gut barrier integrity, maintaining immune balance, and providing energy to host tissues [63, 64]. Aging is nevertheless associated with a significant reduction in SCFA production that increases intestinal permeability and systemic inflammation and impairs muscle anabolism [31, 64]. Additionally, alterations in the gut microbiota can disrupt bile acid metabolism by impairing the conversion of primary to secondary bile acids, affecting digestion and metabolic regulation [80]. This imbalance further reduces the production of neurotransmitters such as serotonin, which plays a key role in metabolic function and musculoskeletal health [80]. These disruptions contribute to overall metabolic dysfunction and compromised bone and muscle health [80].

The gut microbiome also plays a key role in regulating muscle health through the gut-muscle interaction [64]. It is a bidirectional communication system involving multiple signaling pathways mediated by bacteria, SCFAs, cytokines, and other microbial metabolites, which could affect muscle protein synthesis, mitochondrial function, and muscle repair [64, 81]. These pathways are disrupted by alterations in the gut microbiota, contributing to anabolic resistance, muscle atrophy, and physical decline, which is commonly observed in individuals with sarcopenia [81].

In obesity, alterations in the gut microbiota exacerbate energy imbalance and adiposity through increased energy extraction from food, promotion of lipogenesis, and activation of pro-inflammatory pathways [82]. Some of the bacterial taxa that have been implicated in obesity include *Firmicutes* (e.g., *Lactobacillus*, *Clostridium coccoides*,

Table 1 Gut microbiota changes associated with unhealthy and healthy aging and their roles in gut health

Bacterial taxa	Association with aging	Role in gut health
Unhealthy aging		
Proteobacteria [65]	Increased	Potentially harmful, associated with inflammation and gut dysbiosis
Eggerthella [66]	Increased	Involved in metabolism of amino acids, but overgrowth may contribute to harmful effects
Bacteroides fragilis [67]	Increased	Associated with gut inflammation and disruption of intestinal barrier function
Clostridium hathewayi [68]	Increased	Linked with gut dysbiosis and inflammation
Clostridium bolteae [68]	Increased	May promote inflammatory responses in the gut
Clostridium clostridioforme [68]	Increased	Potential to induce inflammation and disrupt gut microbiome balance
Clostridium cindens [68]	Increased	Implicated in gut dysbiosis and gut-related diseases
Ruminococcus torques [69]	Increased	Associated with gut inflammation
Ruminococcus gnavus [69]	Increased	Potentially harmful in high amounts, may contribute to intestinal inflammation
Coprobacillus [66]	Increased	Linked to dysbiosis and gut-related diseases
Streptococcus [70]	Increased	Associated with an imbalance in the gut microbiome and inflammation
Clostridioides difficile [71]	Increased	Harmful pathogen causing gut infections and inflammation
Bilophila [72]	Increased	Associated with inflammatory diseases and metabolic disturbances
Actinomyces [73]	Increased	Associated with gum disease and may be involved in gut inflammation
Desulfovibrio	Increased	Contributes to gut inflammation and may affect gut integrity
Campylobacter [74]	Increased	Known to cause gastrointestinal infections and disrupt gut homeostasis
Atopobiaceae [75]	Increased	May influence immune responses and gut dysbiosis
Veillonella [76]	Increased	Linked with gut inflammation and metabolic disorders
Enterococcus [24]	Increased	Contributes to gut inflammation and dysbiosis
Enterobacteriaceae [24]	Increased	Associated with gut dysbiosis and inflammation
Healthy aging		
Bifidobacterium [68]	Preserved	Contributes to gut homeostasis and digestion, beneficial for immune function
Faecalibacterium prausnitzii [18]	Preserved	Anti-inflammatory, produces butyrate and promotes gut health and metabolic balance
Roseburia [72]	Preserved	Beneficial for gut health, produces butyrate and propionate (an anti-inflammatory SCFA)
Coprococcus [72]	Preserved	Produces SCFAs (butyrate) and acetate, important for maintaining gut health and homeostasis
Prevotella [66]	Preserved	Beneficial for gut health, involved in fiber metabolism and anti-inflammatory effects
Akkermansia [66]	Preserved	Supports gut barrier function, promotes gut health, and prevents inflammation

SCFA, short-chain fatty acids

Eubacterium rectale), *Proteobacteria* (e.g., *Enterobacteriaceae*), and *Bacteroides* spp., which have been associated with increased caloric uptake and metabolic inflammation [68, 83]. Conversely, decreased abundance of beneficial bacteria such as *Akkermansia muciniphila* and *Bifidobacterium* have been associated with dysregulated gut barrier function and increased systemic inflammation, further contributing to obesity-related metabolic dysfunction [84, 85]. These effects, coupled with the decline in microbial diversity and beneficial taxa, increase the risk of developing sarcopenic obesity in older adults [86]. Targeted interventions that modulate the gut microbiota therefore hold significant promise in reversing the adverse effects of aging on the gut–muscle interactions and metabolic health.

Microbiome alterations in sarcopenia, obesity, and sarcopenia obesity

The gut microbiome plays a critical role in regulating several metabolic processes, and the associated age-related microbiome alterations are significant determinants of the pathophysiology of sarcopenia, obesity, and sarcopenic obesity in older adults (Table 4) [25, 88, 90–93]. Recent systematic reviews have demonstrated evidence linking gut microbiota with sarcopenia and sarcopenic obesity, highlighting microbial alterations, inflammation, and altered metabolite profiles [91, 92]. This review contributes novel insights by integrating mechanistic relationships, focusing on SCFAs, and host-microbe relationships from both preclinical and human studies. Additionally, this review highlights underexplored areas such as the implications for precision-based interventions in older adults.

In disrupted gut environments, pathobionts, such as those belonging to the *Enterobacteriaceae* family, proliferate at the expense of beneficial bacteria [94]. This overgrowth increases gut permeability, allowing endotoxins, including lipopolysaccharides (LPS), to enter the circulation [94]. The resultant elevated LPS levels contribute to a state of chronic low-grade inflammation, or metabolic endotoxemia, a hallmark of obesity, sarcopenic obesity, and age-related muscle decline [18, 95]. This inflammatory state is characterized by the release of pro-inflammatory cytokines, including tumor necrosis factor-alpha (TNF-alpha), interleukin-6 (IL-6), and C-reactive protein (CRP), which aggravate muscle catabolism, impair anabolic signaling, and augment the progression of sarcopenia and metabolic dysfunction [96, 97].

An altered *Firmicutes*-to-*Bacteroidetes* ratio may be a prominent feature of gut microbiota compositional shifts associated with obesity and related metabolic disorders. However, its use as a biomarker of microbial imbalance remains controversial due to inconsistent findings across recent studies [98]. The *Firmicutes* species, particularly those with enhanced energy-harvesting capacity, contribute to the overproduction of SCFAs, such as acetate and butyrate [99]. Although SCFAs are critical for gut health well-being and energy metabolism, their excessive production in an obesogenic environment can lead to increased caloric absorption and fat deposition [100]. This "calorie harvesting" effect exacerbates the positive energy balance, contributing to weight gain and further exacerbating metabolic challenges in sarcopenic obesity [100]. Besides energy harvesting, SCFAs also regulate satiety through the modulation of gut hormones such as peptide YY (PYY) and glucagon-like peptide-1 (GLP-1) [101]. Additionally, insufficient butyrate production can impair PYY and GLP-1 signaling, leading to appetite dysregulation, overeating, and further energy imbalance [102]. This disruption is particularly concerning among older adults with obesity and sarcopenic obesity, potentially exacerbating both adiposity and muscle decline [102]. Furthermore, the gut microbiome also influences adipogenesis by regulating lipogenesis and fat storage pathways [102]. Microbial-derived metabolites, including SCFAs, secondary bile acids, and LPS, also have direct impacts on adipocyte differentiation and function [102]. This metabolic dysregulation is further affected by chronic inflammation that creates a vicious cycle supporting both adipogenesis and metabolic dysfunction in sarcopenic obesity.

Taxonomic alterations in the gut microbiome of older adults with sarcopenia, obesity, and sarcopenic obesity are characterized by a reduction in beneficial microbes, such as *Bifidobacterium* spp., *Faecalibacterium prausnitzii*, and *Akkermansia muciniphila* [18]. These bacteria are vital for maintaining gut barrier integrity, producing anti-inflammatory metabolites, and regulating metabolic homeostasis [18]. Conversely, there is an increased abundance of opportunistic pathogens and pro-inflammatory taxa, such as *Enterobacteriaceae*, *Clostridium* spp., and *Proteobacteria* [69, 103, 104]. This shift towards a pro-inflammatory microbial profile exacerbates gut permeability, metabolic endotoxemia, and systemic inflammation, further promoting sarcopenia and metabolic dysfunction.

In addition to taxonomic alterations, there are also functional changes in the gut microbiome playing a significant role in these conditions. Disruptions to the gut microbiota can lead to a reduction in SCFA production, depriving the host of essential metabolites such as butyrate, propionate, and acetate [92, 105]. These metabolites are crucial for gut health, energy metabolism, and inflammation regulation [105]. Altered microbial communities may also exhibit upregulation of genes involved in LPS biosynthesis, leading to increased systemic endotoxemia and inflammation [106].

Metabolomic profile in sarcopenia, obesity, and sarcopenic obesity

In sarcopenia, metabolomic studies have highlighted reductions in metabolites that are favorable for muscle health, including amino acids like branched-chain amino acids (BCAAs) and glutamine, as well as lipids involved in energy metabolism [51]. Similarly, obesity and sarcopenic obesity both are linked to elevated levels of lipid metabolites indicative of dysregulated fat metabolism, such as ceramides and long-chain fatty acids, alongside increased pro-inflammatory mediators and decreased SCFAs [51]. The metabolomic profiles of sarcopenic obesity are particularly complex, reflecting the compounded effects of adiposity-induced inflammation and muscle catabolism.

The gut microbiome plays a pivotal role in determining circulating metabolomic profiles through the production of metabolites such as SCFAs, secondary bile acids, and microbial-derived amino acids [107]. In sarcopenia, obesity, and sarcopenic obesity, alterations in the gut microbiota disrupt the production of these beneficial metabolites, while promoting the generation of harmful metabolites like LPS, which trigger systemic inflammation [107]. This interplay illustrates the potential of targeting the gut microbiome to modulate metabolomic pathways and mitigate the progression of these conditions.

In sarcopenia, plasma metabolomic profiles often show reduced levels of SCFAs, particularly butyrate and propionate, which are crucial for anti-inflammatory effects, energy metabolism, and muscle protein synthesis [27]. Reduced gut microbiota diversity and the depletion of beneficial SCFA-producing microbes such as *Faecalibacterium prausnitzii* and *Roseburia* are associated with lower SCFA levels [108]. Additionally, reduced circulating levels of essential amino acids, particularly BCAAs, may impair muscle protein synthesis, further exacerbating muscle loss in sarcopenia [109].

Obesity and sarcopenic obesity are also characterized by elevated levels of circulating LPS, secondary bile acids, and metabolites associated with insulin resistance, such as BCAAs and aromatic amino acids (e.g., phenylalanine and tyrosine) [18, 110]. Elevated LPS, derived from the outer membrane of gram-negative bacteria, is a crucial driver of metabolic endotoxemia and systemic inflammation [105, 110]. Secondary bile acids, produced by altered gut microbiota, can enhance inflammation and disrupt glucose metabolism, resulting in adiposity and insulin resistance [111].

Moreover, the accumulation of lipid-derived metabolites, including ceramides and acylcarnitines, are commonly observed in obesity and sarcopenic obesity [112]. These metabolites are associated with mitochondrial dysfunction, impaired fatty acid oxidation, and inflammation, which have negative impacts on both muscle and overall metabolic health. Reduced levels of plasma antioxidants and increased oxidative stress markers further indicate heightened metabolic stress in these populations.

Impact of probiotics, prebiotics, and synbiotics in sarcopenia, obesity, and sarcopenic obesity

Evidence from animal studies

The association between the gut microbiota and components of sarcopenia is an emerging field of research, in which, causative results have been derived primarily from preliminary studies in mice, supporting a gut–muscle axis [30, 113, 114]. For instance, faecal samples from older adults with high and low physical function transferred into germ-free mice to assess the microbiome's causative role demonstrated increased grip strength (by 6.4%) in high functioning-colonized mice compared to low functioning-colonized mice at 1-month follow-up [113]. However, lean mass and endurance capacity did not differ between the two groups of mice, despite variations in microbiota composition from different human donors [113]. Regarding gut bacteria, *Prevotellaceae*, *Prevotella*, *Barnesiella*, and *Barnesiella intestinihominis* were consistently higher in high functioning adults and mice compared to low functioning counterparts [113]. These taxa may influence muscle function through their role in fermenting dietary fiber into SCFAs, such as propionate and butyrate, which have been associated with anti-inflammatory effects, improving energy metabolism, and muscle protein synthesis.

Furthermore, faecal sample transplantation from individuals with sarcopenia undergoing hemodialysis into antibiotic-treated mice for 3 weeks exhibited reduced muscle function, muscle mass, and intestinal *Akkermansia* abundance compared to colonized mice with microbiota from individuals without sarcopenia with haemodialysis [113]. Additionally, metabolomic analysis further revealed that *Lachnoclostridium* was inversely correlated with muscle endurance, while increased *Coprobacillus* was negatively correlated with physical performance [113]. More recently, another study investigated whether the phenotype of reduced bone and lean mass in old mice (21 months old) could be transferred to young (5 months old), healthy mice via gut microbiota transplantation [115]. Germ-free mice were colonized with gut microbiota from the older or young adult donors and although microbial species from old mice did not significantly affect bone mass or muscle strength, a reduction of lean mass was observed after 5 weeks of colonization [115]. These findings demonstrate that not all studies consistently report improvements in muscle function, therefore suggesting that specific microbial strains may have differing effects. Reduced cecal propionate production and the absence of *Bacteroides ovatus*, a bacterium enriched in young donor germ-free mice that is associated with higher lean mass in mice and humans, were observed [115]. Moreover, an 8-week faecal microbiota transplantation (FMT) from young donor mice (12 weeks old) to aged mice (88 weeks old) significantly mitigated age-related muscle mass loss, reduced grip strength, and functional decline [116]. These improvements were assessed using magnetic resonance imaging (MRI) for muscle mass, grip strength tests, rotarod performance, and exhaustive running tests [115, 116]. Specifically, after 8 weeks, regeneration in both fast- and slow-twitch muscles, increased myofiber cross-sectional area, and satellite cells were also observed in conjunction with increases (e.g., *Akkermansia*, *Lactobacillus*) and reductions (e.g., *Collinsella*, indoxyl sulfate) in bacteria or metabolites [116]. In addition, transplanting microbiota from young mice (5 weeks old) into 12- and 25-month-old mice improved muscle fiber thickness and grip strength after 8 weeks [117]. Gut bacteria enriched in the recipient older mice from the young counterparts correlated with improved fitness, for which, *Muribaculaceae*, *Bacteroidaceae*, and *Prevotellaceae* were the most distinct species that changed over time [117]. These findings highlight the potential of young-derived microbiota to rejuvenate sarcopenia-related decline in older rodent models by modulating microbial composition [117].

Although, animal studies provide important insights into the relationship between the gut microbiome and sarcopenia- and obesity-related outcomes, these findings should be interpreted with caution when extrapolating to humans. Differences in microbiota composition, physiology, environmental exposures, and complexities of human lifestyle may limit direct translation from animal models to human settings. Therefore, further evidence in humans is essential to validate these underlying pathways and their therapeutic potential.

Evidence from human studies

In humans, several observational studies have demonstrated a strong association between gut microbial composition and reductions in physical function and inflammation in clinical populations [118, 119]. In a recent study involving 1,821 older adults aged between 62 and 96 years, 18 microbial species and 17 circulating metabolites were identified which varied along a frailty index continuum [120]. Notably, species such as *Faecalibacterium prausnitzii* and various *Clostridium* species were inversely associated with frailty severity, and a composite microbial signature significantly predicted 2-year mortality (adjusted Hazard Ratio (HR) = 2.86; 95% CI 1.38–5.93) [120]. These findings underscore the potential of gut microbiota as a biomarker and modifiable target in aging and frailty risk stratification. Additionally, ongoing research is exploring whether microbial communities may play a role in mediating the effects of exercise in frail populations; however, most interventional studies include the incorporation of dietary modification, particularly via probiotics, prebiotics, and/or synbiotics [121].

A previous meta-analysis, including ten studies, demonstrated significant improvements in muscle mass (standardized mean difference (SMD) 0.42, $p < 0.01$) and overall muscle strength (six studies: SMD 0.69, $p < 0.01$) following probiotics compared to placebo [35]. However, caution is warranted when interpreting these findings due to variability in results, particularly in subgroup analyses. For example in a subgroup analysis of adults with a mean age of 50 years, the effect on muscle mass did not reach statistical significance ($p = 0.09$), despite demonstrating a modest clinically meaningful improvement (SMD 0.41), which may be explained by the high degree of variability [35]. In contrast, handgrip strength showed a significant improvement (SMD 0.96, $p < 0.01$) based on data from only three studies [35].

Prebiotic supplementation, specifically 13 weeks of inulin (3,375 mg) and fructooligosaccharides (FOS) (3,488 mg), in adults over 65 years of age with frailty, resulted in significant improvements in grip strength (right hand only) ($p = 0.04$) and reduced feelings of exhaustion [32]. Although this finding may indicate potential clinical relevance, it may be explained due to statistical variation, and therefore should be interpreted with caution. However, gait speed and the Barthel Index, a surrogate measure of activities of daily living, remained unchanged [32]. In a 12-week study involving community-dwelling Japanese individuals over 65 years, inulin supplementation (5 g/day) resulted in no significant changes in the upper- or lower-limb muscle strength or body composition compared to placebo [122].

Moreover, synbiotic supplementation (probiotics and 6-g fructooligosaccharides) in older adults with dynapenia (defined as an age-associated loss of muscle strength) did not exhibit any effects on grip strength compared to placebo after 12 weeks of intervention, although there was an observed reduction in body fat and fat-free mass [123]. These changes in body composition were reflective of a significant change between groups pertinent to body weight, suggesting that synbiotics may reduce body weight alongside fat-free mass, without affecting upper body strength [123]. In older adults with dynapenia, these results may lead to unfavorable outcomes, as reductions in body weight and fat-free mass are often associated with decreased physical function, increased dependence, and higher mortality [123]. This suggests that such interventions may be more suitable for individuals with obesity or metabolic syndrome [123]. Therefore, there is a need for caution when considering synbiotic supplementation in this population, as it underscores the importance of preserving lean mass in interventions aimed at improving musculoskeletal health in older adults**.** In addition, synbiotic supplementation with calorie restriction has also led to weight and body fat losses in older adults with metabolic syndrome compared to placebo after

12 weeks of intervention, without affecting lean mass [124]. These studies support the hypothesis of synbiotic supplementation as a nutritional therapy that may be effective in older adults with sarcopenic obesity rather than those with sarcopenia or dynapenia alone.

Recent trials investigating these nutritional interventions have demonstrated benefits for muscle health. Among older individuals with osteoarthritis, 12 weeks of probiotics supplementation (strains included *Streptococcus thermophilus, Bifidobacterium longum, Bifidobacterium breve, Lactobacillus strains, and Lactobacillus delbrueckii*) compared to placebo improved balance, grip strength, and gait speed [125, 126]. In older adults, 16 weeks of supplementation resulted in positive changes in grip strength, skeletal muscle index, and sarcopenia-related quality of life (SarQoL), while also decreasing plasma zonulin, a marker of intestinal permeability [126].

In addition, adults with knee osteoarthritis and obesity on 6 months of prebiotic supplementation (oligofructose-enriched inulin; 16 g/day) compared to placebo have resulted in several positive changes, including a significant difference in serum metabolites between groups [127]. Specifically, there was an upregulation of phenylalanine and tyrosine metabolism [127]. Additionally, reductions in trunk fat mass, improvements in grip strength, 6-minute walking distance (6MWD), and the timed-up-and-go test were also observed [127]. Moreover, *Bifidobacterium* abundance was positively correlated with both 6MWD and grip strength [127]. Similar benefits in gait speed and grip strength were observed in frail older adults supplemented with inulin and oligofructose (15 g/day) compared to placebo for 12 weeks [128]. In the frail group, *Bifidobacterium* and *Blautia* were negatively associated with exhaustion and frailty scores, while *Prevotella* showed positive correlations with both frailty and exhaustion, suggesting a possible link to poorer physical function [128]. In contrast, when a lower dosage of prebiotics (inulin plus fructooligosaccharides; 7.5g/day) was supplemented in older community-dwelling twins for 12 weeks, no effects were observed on chair rising time, grip strength, or the short physical performance battery (SPPB) test, despite an increase in relative *Bifidobacterium* abundance [129]. These findings suggest that a higher tolerated dosage may have favorable effects on sarcopenia outcomes, while protecting against fat-free mass and lean mass losses in individuals with obesity. However, there is limited research on prebiotic supplements in individuals with sarcopenic obesity. To effectively investigate the gut-muscle axis, studies should include assessments of microbial taxa at baseline and mid/post-intervention. Most studies lack these methods and fail to control dietary intake, limiting the strength of the current evidence in literature. A summary of a literature search of studies on probiotic, prebiotic, and synbiotic supplementation versus placebo and their effects on measures of sarcopenia in adults aged 50 and above is presented in Table 2, and the results from these studies are presented in Table 3.

Clinical implications and potential therapeutic approaches

The interplay between the gut microbiome, metabolic function, and musculoskeletal health in older adults presents a novel therapeutic potential targeting sarcopenia, obesity, and sarcopenic obesity. Probiotics, prebiotics, and synbiotics are proposed to be promising interventions to modulate the gut microbiota, reduce inflammation, and support muscle and metabolic health (Figure 1).

Dietary and lifestyle considerations

The efficacy of probiotics, prebiotics, and synbiotics can be significantly enhanced when combined with dietary and lifestyle interventions. Diets rich in fiber, polyphenols, and anti-inflammatory nutrients create a favorable environment for modulating gut microbiota [130]. These nutrients have been shown to promote the growth of beneficial gut bacteria, enhancing microbial diversity. In particular, fiber-rich diets support the production of SCFAs, which promote gut barrier integrity [130].

In addition to dietary factors, regular physical activity plays a pivotal role in overall gut health [131]. Exercise has been linked to improvements in microbial diversity, with evidence showing that physically active individuals tend to have a more balanced gut microbiome compared to sedentary individuals [131]. Moreover, physical activity enhances the communication pathway between the gut and skeletal muscle, which can amplify the benefits of microbiome-targeted therapies. Exercise has been shown to positively influence muscle mass, strength, and metabolic health, and when combined with probiotics, prebiotics, or synbiotics, the effects on muscle health and glucose metabolism can be further enhanced [131]. A study involving 48 healthy sedentary young men found that 12 weeks of circuit training combined with multi-strain probiotic supplementation led to significant improvements in muscular performance and strength [132]. These findings highlight the potential synergistic effects of exercise and gut microbiota-targeted interventions in promoting musculoskeletal and metabolic health.

Future directions and challenges

Although evidence supports the therapeutic effectiveness of probiotics, prebiotics, and synbiotics, challenges remain in translating these findings into clinical practice.

Table 2 Studies including probiotics, prebiotics, and synbiotic supplementation compared to placebo on measures of sarcopenia

Study	Health status	Intervention sample size *n* (men/women)	Control sample size (m/f)	Intervention age (years)	Control age (years)	Intervention	Duration (weeks)	Body composition tool	Dietary intake control (yes/no)	Baseline microbiome status assessed (yes/no)	Outcomes/primary endpoints
Probiotics											
Bartos 2023	Community-dwelling	37 (-/-)	30 (-/-)	69 (7)	71.5 (6)	10^6 *Streptococcus thermophilus* GH, *Streptococcus salivarius* GH NEXARS, *Lactobacillus plantarum* GH, and *Pediococcus pentosaceus* GH	12	BIA	No	No	Muscle mass, gait speed, 30-s CST; sarcopenia
Chaiyasut 2022	Community-dwelling	24 (3/21)	24 (7/17)	61.6 (0.8)	58.8 (1.2)	2×10^{10} CFU of *Lactobacillus paracasei* HII01; 2×10^{10} CFU of *Bifidobacterium breve*; 1×10^{10} CFU of *Bifidobacterium longum*)	12	BIA	No	No	Muscle mass; sarcopenia
Hevilla 2023	Hemodialysis	10 (7/3)	10 (8/2)	66 (18.5)	65.1 (18.4)	Protein supplement and probiotic (*Bifidobacterium breve* CNCMI-4035 [1.00 E+09 colony formingunits (CFU)], *Bifidobacterium animalis lactis* BPL1CECT 8145 (3.50 E+09 CFU), and *Lactobacillus paracasei* CNCMI-4034 (5.00 E+08 CFU)	12/24	BIA	Yes	No	Fat-free mass, handgrip strength, SPPB; sarcopenia
Higashikawa 2016	Overweight	21 (8/13)	20 (7/13)	52.5 (11.8)	52.8 (11.6)	10^{11} CFU *Pediococcus pentosaceus* LP28	12	BIA	No	No	Lean mass; obesity

Table 2 (continued)

Study	Health status	Intervention sample size *n* (men/women)	Control sample size (m/f)	Intervention age (years)	Control age (years)	Intervention	Duration (weeks)	Body composition tool	Dietary intake control (yes/no)	Baseline microbiome status assessed (yes/no)	Outcomes/primary endpoints
Inoue 2018	Community-dwelling	20 (7/13)	18 (7/11)	69.9 (3.0)	70.9 (3.2)	*B. longum* BB536, *B. infantis* M-63, *B. breve* M-16 V, and *B. breve* B-3 (approximately 1.25×10^{10} CFU each	12	BIA	No	No	Lean mass; sarcopenia
Jaff 2024	Type 2 diabetes	34 (11/23)	34 (8/26)	55.6 (4.7)	56.5 (4.8)	*Bacillus coagulans*, *Lactobacillus plantarum*, *Lactobacillus acidophilus*, and *Bifidobacterium bifidum*, 3 billions/capsules (3×10^9 CFU)	12	BIA	Yes	No	Fat-free mass; obesity
Karim 2022	Heart failure	44 (44/0)	48 (48/0)	67.6 (4.9)	65.2 (5.6)	112 billion live bacteria, including bifidobacteria (*B. longum* DSM 24736, *B. breve* DSM 24732, DSM 24737), lactobacilli (DSM 24735, DSM 24730, DSM 24733, *L. delbrueckii* subsp. *bulgaricus* DSM24734), and *Streptococcus thermophilus* (DSM 24731)	12	BIA	No	No	Handgrip strength, gait speed, skeletal muscle index, SPPB, 5-time CST; sarcopenia

Table 2 (continued)

Study	Health status	Intervention sample size *n* (men/women)	Control sample size (m/f)	Intervention age (years)	Control age (years)	Intervention	Duration (weeks)	Body composition tool	Dietary intake control (yes/no)	Baseline microbiome status assessed (yes/no)	Outcomes/primary endpoints
Karim 2022a	Chronic obstructive pulmonary disease	13 (8/5)	17 (9/8)	66.9 (3.4)	68.3 (4.2)	112 billion live bacteria, including bifidobacteria (*B. longum* DSM 24736, *B. breve* DSM 24732, DSM 24737), lactobacilli (DSM 24735, DSM 24730, DSM 24733, *L. delbrueckii* subsp. *bulgaricus* DSM24734), and *Streptococcus thermophilus* (DSM 24731)	12	BIA	No	No	Appendicular lean mass, handgrip strength, gait speed, skeletal muscle index, SPPB, 5-time CST; sarcopenia
Karim 2024	Osteoarthritis	64 (19/45)	71 (21/50)	69.3 (5.5)	69.1 (4.6)	112 billion live bacteria, including bifidobacteria (*B. longum* DSM 24736, *B. breve* DSM 24732, DSM 24737), lactobacilli (DSM 24735, DSM 24730, DSM 24733, *L. delbrueckii* subsp. *bulgaricus* DSM24734), and *Streptococcus thermophilus* (DSM 24731)	12	-	No	No	Gait speed, handgrip strength; sarcopenia

Table 2 (continued)

Study	Health status	Intervention sample size *n* (men/women)	Control sample size (m/f)	Intervention age (years)	Control age (years)	Intervention	Duration (weeks)	Body composition tool	Dietary intake control (yes/no)	Baseline microbiome status assessed (yes/no)	Outcomes/primary endpoints
Lee 2021	Frailty	18 (10/8)	13 (8/5)	80.5 (9.4)	75.2 (7.2)	TWK10 high-dose group (6×10^{10} CFU/day) (TWK10-H)	18	DXA	No	No	Muscle mass, gait speed, TUG, 30-s CST, handgrip strength; sarcopenia
Lei 2016	Distal radius fracture	189 (93/96)	192 (94/98)	64.3 (4.1)	65.1 (3.7)	Skimmed milk and 6×10^{9} *Lactobacillus casei* Shirota	24	-	No	No	Handgrip strength; sarcopenia
Minami 2015	Obesity	19 (6/13)	25 (11/14)	58.9 (2.0)	61.9 (1.9)	*Bifidobacterium breve* B-3 5×10^{10} CFU	12	BIA	No	No	Muscle mass; sarcopenia obesity
Moludi 2019	Coronary artery disease	22 (20/2)	22 (21/1)	51.2 (11.1)	54.0 (8.6)	1.6×10^{9} CFU *Lactobacillus rhamnosus* GG	12	BIA	Yes	No	Fat-free mass; sarcopenia
Nilsson 2018	Low bone mineral density	34 (0/34)	36 (0/36)	76.4 (1.0)	76.3 (1.1)	10^{10} CFU *L. reuteri* 6475	52	DXA	No	No	Lean mass; sarcopenia
Qaisar 2024	Geriatric population	60 (60/0)	63 (63/0)	73 (4)	71.4 (3.9)	Bifidobacteria (*B. longum* DSM 24736, *B. breve* DSM 24732, DSM 24737), *Streptococcus thermophilus* DSM 24731, and lactobacilli (DSM 24735, DSM 24730, DSM 24733, *L. delbrueckii* subsp. *bulgaricus* DSM 24734)	16	BIA	No	No	Handgrip strength, skeletal muscle index, gait speed; sarcopenia

Table 2 (continued)

Study	Health status	Intervention sample size *n* (men/women)	Control sample size (m/f)	Intervention age (years)	Control age (years)	Intervention	Duration (weeks)	Body composition tool	Dietary intake control (yes/no)	Baseline microbiome status assessed (yes/no)	Outcomes/primary endpoints
Roman 2019	Cirrhosis	18 (-/-)	18 (8/10)	65.8 (3.1)	64.0 (2.6)	*Streptococcus thermophilus* DSM 24731, *Bifidobacterium breve* (*B. breve*) DSM 24732, *B. longum* DSM 24736, *B. infantis* DSM 24737, *Lactobacillus paracasei* (*L. paracasei*) DSM 24733, *L. acidophilus* DSM 24735, *L. delbrueckii subspbulgaricus* DSM 24734, and *L. plantarum* DSM 24730, at a total dose of 450 billion live bacteria	12	-	No	No	TUG, gait speed, handgrip strength; sarcopenia
Skrypnik 2019	Obesity	23 (0/23)	24 (0/24)	56.0 (6.6)	60.5 (6.9)	10^{10} CFU (*Bifidobacterium bifidum* W23, *B. lactis* W51, *B. lactis* W52, *Lactobacillus acidophilus* W37, *L. brevis* W63, *L. casei* W56, *L. salivarius* W24, *Lactococcus lactis* W19, and *Lc. lactis* W58)	12	BIA	No	No	Fat-free mass; sarcopenic obesity
Tay 2020	Prediabetes	15 (6/9)	11 (2/9)	52.9 (8.7)	54.1 (6.4)	6×10^9 CFU *Lacticaseibacillus rhamnosus* HN001	12	DXA	Yes	No	Fat-free mass; sarcopenia

Table 2 (continued)

Study	Health status	Intervention sample size *n* (men/women)	Control sample size (m/f)	Intervention age (years)	Control age (years)	Intervention	Duration (weeks)	Body composition tool	Dietary intake control (yes/no)	Baseline microbiome status assessed (yes/no)	Outcomes/primary endpoints
Prebiotics											
Buigues 2016	Frailty	28 (9/19)	22 (6/16)	74.2 (1.6)	73.4 (1.8)	Prebiotic—inulin (3375 mg) plus fructooligosaccharides (3488 mg)	13	-	Yes	No	Gait speed, handgrip strength; sarcopenia
Fortuna 2024	Knee osteoarthritis and obesity	17 (-/-)/14 (-/-)/8 (-/-)	18 (-/-)/14 (-/-)/8 (-/-)	59.2 (9.7)	59.1 (11.5)	Prebiotic—oligofructose-enriched inulin (16 g)	12/24/36	DXA	Yes	Yes	Gait speed, TUG, handgrip strength, 6MWD, 30-s CST, lean mass; sarcopenic obesity
Murata 2023	Community-dwelling	14 (0/14)	14 (0/14)	73.1 (3.2)	72.7 (3.6)	Prebiotic—inulin (5 g)	12	DXA	Yes	No	Lean mass, handgrip strength, knee extension strength, gait speed; sarcopenia
Ni Lochlainn 2024	Community-dwelling	32 (-/-)	34 (-/-)	73.1 (4.9)	73.1 (4.9)	Prebiotic—inulin plus fructooligosaccharides (7.5 g)	12	-	Yes	Yes	Chair rising time, handgrip strength, SPPB
Pan 2020	Metabolic syndrome	16 (8/8)	15 (7/8)	53.6 (6.8)	57.6 (6.1)	Fermented barley—wheat flour compound noodle	10	BIA	Yes	No	Muscle mass; sarcopenic obesity
Yang 2024	Frailty	44 (-/-)	48 (-/-)	74 (8.8)	73.7 (4.6)	Prebiotic—inulin and oligofructose (15 g)	12	BIA	Yes	Yes	Muscle mass, handgrip strength, gait speed; Sarcopenia
Synbiotics											

Table 2 (continued)

Study	Health status	Intervention sample size *n* (men/women)	Control sample size (m/f)	Intervention age (years)	Control age (years)	Intervention	Duration (weeks)	Body composition tool	Dietary intake control (yes/no)	Baseline microbiome status assessed (yes/no)	Outcomes/primary endpoints
Neto 2013	Dynapenia	9 (1/8)	8 (3/5)	All: 67.9 (4.5)	All: 67.9 (4.5)	Synbiotic (6 g fructooligosaccharide, 10^8 to 10^9 CFU *Lactobacillus paracasei*, 10^8 to 10^9 CFU *Lactobacillus rhamnosus*, 10^8 to 10^9 CFU *Lactobacillus acidophilus* and 10^8 to 10^9 CFU *Bifidobacterium lactis*)	12	BIA	No	No	Fat-free mass, handgrip strength; sarcopenia

Table 2 (continued)

Study	Health status	Intervention sample size *n* (men/women)	Control sample size (m/f)	Intervention age (years)	Control age (years)	Intervention	Duration (weeks)	Body composition tool	Dietary intake control (yes/no)	Baseline microbiome status assessed (yes/no)	Outcomes/primary endpoints
Rabiei 2015	Metabolic syndrome	20 (-/-)	20 (-/-)	57.1 (7.2)	60.8 (7.7)	Synbiotic—2 capsules (*Lactobacillus casei*, *Lactobacillus rhamnosus*, *Streptococcus thermophilus*, *Bifidobacterium breve, Lactobacillus acidophilus, Bifidobacterium longum, Lactobacillus bulgaricus,* FOS (Fructooligosaccharide- prebiotic), magnesium stearate (source: mineral and vegetable), vegetable capsule (hydroxypropyl methyl cellulose) TVC: 200 million CFU TVC: 2×10^8 CFU	12	BIA	Yes	No	Lean mass; sarcopenic obesity

Data is presented as mean (standard deviation/error). *6MWD*, 6-min walking distance; *BIA*, bioelectrical impedance; *CFU*, colony forming units; *CST*, chair stand test; *DXA*, dual X-ray absorptiometry; *SPPB*, short physical performance battery; *TUG*, timed up and go

Table 3 Studies including probiotic, prebiotic, and synbiotic supplementation compared to placebo on gut microbial changes

Study	Baseline microbiome status assessed (yes/no)	Post-interventional gut bacterial changes
Probiotics		
Bartos 2023	No	-
Chaiyasut 2022	No	-
Hevilla 2023	No	-
Higashikawa 2016	No	-
Inoue 2018	No	-
Jaff 2024	No	-
Karim 2022	No	-
Karim 2022a	No	-
Karim 2024	No	-
Lee 2021	No	-
Lei 2016	No	-
Minami 2015	No	-
Moludi 2019	No	-
Nilsson 2018	No	-
Qaisar 2024	No	-
Roman 2019	No	Authors declare that they did not observe significant changes at the end of the treatment in comparison with baseline data in either group at a phylum, genus, or species level
Skrypnik 2019	No	-
Tay 2020	No	-
Prebiotics		
Buigues 2016	No	-
Fortuna 2024	Yes	No overall structural difference found between placebo and prebiotic regarding alpha and beta diversity There were significantly different between placebo and prebiotic at 3 months, including 16 ASVs that were decreased (6 ASVs classified as *Bacteroides*, 4 ASVs classified as *Blautia*, and ASVs classified as *Oscillospiraceae*, *Catenibacterium*, *Holdemanella*, and *Akkermansia*) and 21 ASVs that were increased (3 *Faecalibacterium*, 3 *Bacteroides*, 2 *Ruminococcus*, and ASVs classified as *Roseburia*, *Lachnospiraceae*, *Bifidobacterium*) by prebiotic At 6 months, significant differences were found for 15 ASVs, chiefly *Ruminococcaceae*, *Bacteroides*, *Blautia,* and *Roseburia*
Murata 2023	No	-
Ni Lochlainn 2024	Yes	Sixty microbiome features changed between baseline and end of the study for the prebiotic group, while only three were changed in placebo. These features included alpha diversity and relative abundance of bacterial taxa. A reduction in alpha diversity was observed in the prebiotic arm The prebiotic group had a significant increase in the relative abundance of *Actinobacteria*, particularly *Bifidobacterium*, compared to placebo. Notably, no increase in *Lactobacillus* or *Faecalibacterium* was seen
Pan 2020	No	-
Yang 2024	Yes	There was no significant change in α-diversity among the groups after the intervention. There was significant β-diversity between the subgroup in the prefrailty group that received maltodextrin placebo after placebo and the subgroup in the prefrailty group that received prebiotic intervention In the prefrail group, *B. adolescentis* was predominant in the prebiotic mixture group, and *Faecalibacterium* was predominant in the placebo group. In the prebiotic group, *Bifidobacterium pseudocatenulatum* and *E. coli*, *Veillonellaceae*, *Enterobacteriales*, and *Negativicutes* were the dominant bacteria
Synbiotics		
Neto 2013	No	-
Rabiei 2015	No	-

ASVs, amplicon sequence variants

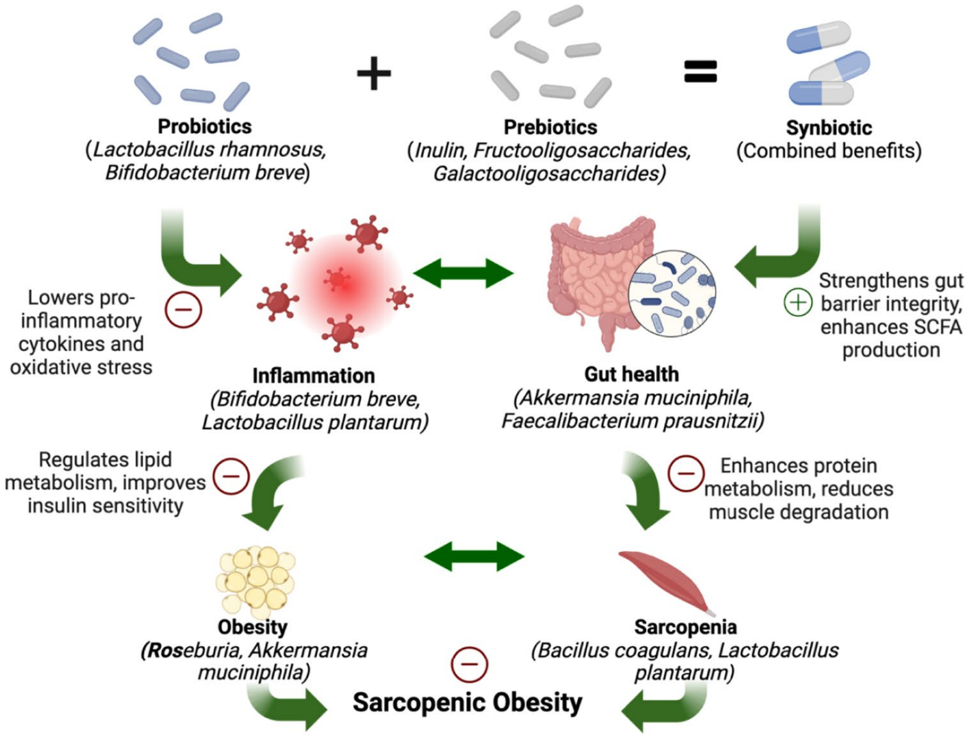

Fig. 1 Associations between prebiotics, probiotics, and synbiotic supplementation on sarcopenic obesity. This figure illustrates the potential mechanisms by which prebiotic, probiotic, and synbiotic supplementation may influence sarcopenic obesity. These interventions are shown to reduce inflammation and improve gut health, which may, in turn, lower the risk of obesity, sarcopenia, and sarcopenic obesity. The pathways involve modulation of the gut microbiome, leading to improved metabolic outcomes and muscle preservation. SCFA, short-chain fatty acids

Current studies lack control for dietary intake, which can significantly influence gut microbiota composition and outcomes. Additionally, individual variability, dosage for optimal effect, and strain-specific effects require further investigation. Also, long-term studies are needed to confirm the sustained benefits of these interventions and their safety in older populations. Evidence in the context of sarcopenic obesity remains sparse, as there are currently only limited studies available in this population [3, 59, 60, 133]. Among these studies, there is often inconsistent diagnostic criteria limiting the ability to draw any definitive conclusions about the efficacy of such nutritional interventions [59, 133]. Future studies should therefore prioritize clearly defining sarcopenic obesity and evaluating such interventions with appropriately stratified musculoskeletal and metabolic outcomes including muscle mass, strength, physical performance, adiposity, and inflammatory and microbiome markers.

Probiotic, prebiotic, and synbiotic interventions offer promising strategies to counteract these microbiome-driven alterations. Probiotics, particularly *Lactobacillus* and *Bifidobacterium* species, can suppress the growth of pathobionts, reduce gut permeability, and mitigate systemic inflammation by enhancing intestinal barrier function [134]. Prebiotics, such as inulin and GOS, selectively promote the growth of beneficial microbes, helping to restore a healthy *Firmicutes*-to-*Bacteroidetes* ratio and optimize SCFA production [135]. Synbiotics offer combined benefits by supporting microbial diversity and promoting beneficial metabolite production, such as SCFAs, which may contribute to improved gut and systemic health among older adults [136] (Table 4).

In older adults with sarcopenia, obesity, and sarcopenic obesity, such interventions have the potential to reduce inflammation, improve energy metabolism, and support muscle health. Through the restoration of the gut microbiome and regulating key metabolic pathways, probiotics, prebiotics, and synbiotics could serve as adjunctive therapies to improve clinical outcomes in these populations [136]. Future research must address how to tailor these interventions to target individual-specific microbial and metabolic alterations associated with these conditions to maximize their efficacy and long-term impact. Furthermore, personalized supplementation strategies maybe equipped, and future research should identify microbial strains or profiles that predict responses to specific probiotic, prebiotic, or synbiotic supplementation. These methods may support precision nutritional intake allowing for interventions to be tailored individually for benefits.

Probiotic, prebiotic, and synbiotic interventions can help reverse these taxonomic and functional shifts by modulating microbial composition and restoring metabolic functions. Probiotics such as *Bifidobacterium* and *Lactobacillus* species can repopulate depleted beneficial taxa, reduce gut permeability, and suppress pro-inflammatory microbes [136]. Prebiotics, such as FOS and inulin, selectively promote the growth of SCFA-producing bacteria, enhancing their functional capacity [136]. Synbiotics combine these effects by offering live beneficial microbes along with substrates for

Table 4 Reported gut microbiota alterations in sarcopenia and sarcopenic obesity, and their possible functional implications

Condition	Reported microbiota alterations	Possible functional implications
Sarcopenia	• ↓ α-diversity (richness and evenness) [87] • ↑ phylum *Proteobacteria,* ↑ genus *Escherichia-Shigella* [88] • ↓ phylum *Firmicutes*, ↓ genera *Faecalibacterium, Prevotella, Blautia* [88] • Higher *Prevotella*/*Bacteroides* (P/B) ratio, ↓ *Coprococcus* and *Lachnospiraceae* in one study [89]	• ↓ Beneficial SCFA-producers → reduced anti-inflammatory and energy-yielding capacity • ↑ Pathobionts → enhanced intestinal and systemic inflammation • → Anabolic resistance, muscle mass and strength decline via gut–muscle axis
Sarcopenic obesity	• Distinct signature compared with sarcopenia alone: e.g., ↑ family *Enterobacteriaceae* identified as biomarker for SO [25] • Although fewer studies, evidence of combined effect of obesity + low muscle on gut dysbiosis (e.g., worse diversity, altered taxa) [25]	• Combined metabolic and muscle impairments • Worsened gut barrier/permeability → endotoxaemia → inflammation → muscle catabolism and fat accumulation

their colonization and activity [136]. Synbiotic combinations may offer synergistic effects, simultaneously replenishing beneficial microbes and providing substrates to enhance their metabolic activity [33]. Synbiotic supplementation has been linked to reduced levels of pro-inflammatory metabolites and increased SCFA concentrations, with subsequent improvements in insulin sensitivity and muscle function [136]. Additionally, synbiotics may help modulate bile acid profiles, reducing the harmful effects of secondary bile acids while promoting the production of primary bile acids, which support lipid metabolism and energy balance [33]. These interventions have shown promise in reducing inflammation, improving gut barrier function, and restoring SCFA production in older adults with sarcopenia and obesity [33]. They also hold potential to enhance muscle health and metabolic outcomes by modulating pathways related to amino acid metabolism, vitamin synthesis, and bile acid regulation [123, 137]. Nevertheless, further research is required to determine if synbiotics offer greater benefits compared to probiotics or prebiotics alone, or whether these combinations need to be tailored on an individual basis for best outcomes.

Future studies should focus on well-designed randomized controlled trials (RCTs) with larger sample sizes, longer follow-up periods, and diverse populations. These RCTs should incorporate objective measures of sarcopenia such as muscle mass (MRI or computed tomography), muscle strength (e.g., grip strength), physical performance (e.g., gait speed, sit-to-stand test), and metabolic assessments (e.g., insulin sensitivity). Additionally, studies should include gut microbiota assessments via stool samples to characterize microbial diversity and identify specific compositional changes. To optimize interventions, RCTs should explore different bacterial strains and their specific effects on microbial pathways related to muscle health and metabolism, guiding personalized supplementation strategies. Additionally, future studies among older adults with sarcopenic obesity should include subgroup analyses to identify whether individuals with the comorbid impairment of muscle and fat have a varied response to the different probiotic, prebiotic, or synbiotic combinations. Furthermore, these studies should also investigate if microbial shifts correlate with changes in strength, body composition, and metabolic markers. In addition, international multi-center studies would further enhance the generalizability and real-world applicability of these findings by allowing for the understanding of region-specific associations between gut microbiome and sarcopenia, obesity, and sarcopenic obesity. Given the differences in dietary patterns and cultural variations influencing these associations, conducting studies across diverse population would provide a more comprehensive evaluation of the underlying mechanisms contributing to gut microbiota variations and their impact on these age-associated conditions.

Conclusion

In summary, the supplementation of probiotics, prebiotics, and synbiotics has shown potential in modulating the gut microbiome and improving muscle health in older adults, particularly those with sarcopenia, obesity, and sarcopenic obesity. Although, evidence from recent studies demonstrates beneficial effects on muscle strength, function, and body composition, the overall findings remain inconsistent, largely due to variations in study design, sample size, and intervention duration. Despite these discrepancies, specific bacterial species, such as *Bifidobacterium* and *Blautia*, have emerged as potential biomarkers for improvements in physical function, suggesting a link between the gut microbiota and musculoskeletal health.

Future research should focus on more robust RCTs that include comprehensive dietary assessments and longitudinal interventions to better understand the mechanisms through which gut microbiota influence sarcopenia and related conditions. Additionally, more investigation into optimal dosing, prebiotic and probiotic combinations, and their long-term impact on older adults is needed to establish clear guidelines for clinical use. Ultimately, gut-targeted therapies may provide a novel and non-invasive approach to managing sarcopenia, obesity, and sarcopenic obesity and improving the overall health and quality of life of older adults.

Funding Open access funding provided by University of Gothenburg.

Declarations

Conflicts of interest None.

References

1. Barazzoni R, Bischoff S, Boirie Y, Busetto L, Cederholm T, Dicker D et al (2018) Sarcopenic obesity: time to meet the challenge. Obes Facts 11(4):294–305
2. Batsis JA, Villareal DT (2018) Sarcopenic obesity in older adults: aetiology, epidemiology and treatment strategies. Nat Rev Endocrinol 14(9):513–537
3. Cauley JA (2015) An overview of sarcopenic obesity. J Clin Densitom 18(4):499–505
4. Castillo EM, Goodman-Gruen D, Kritz-Silverstein D, Morton DJ, Wingard DL, Barrett-Connor E (2003). Sarcopenia in elderly men and women: the Rancho Bernardo study. Am J Prev Med 25(3):226-31
5. Bijlsma AY, Meskers CG, Ling CH, Narici M, Kurrle SE, Cameron ID et al (2013) Defining sarcopenia: the impact of different diagnostic criteria on the prevalence of sarcopenia in a large middle aged cohort. AGE 35(3):871–81
6. Buckinx F, Aubertin-Leheudre M (2022) Sarcopenia in menopausal women: current perspectives. Int J Womens Health 14:805–819
7. Aibar-Almazan A, Martinez-Amat A, Cruz-Diaz D, Jimenez-Garcia JD, Achalandabaso A, Sanchez-Montesinos I et al (2018) Sarcopenia and sarcopenic obesity in Spanish community-dwelling middle-aged and older women: association with balance confidence, fear of falling and fall risk. Maturitas 107:26–32
8. Hong W, Cheng Q, Zhu X, Zhu H, Li H, Zhang X et al (2015) Prevalence of sarcopenia and its relationship with sites of fragility fractures in elderly Chinese men and women. PLoS One 10(9):e0138102
9. Domiciano DS, Figueiredo CP, Lopes JB, Caparbo VF, Takayama L, Menezes PR et al (2013) Discriminating sarcopenia in community-dwelling older women with high frequency of overweight/obesity: the Sao Paulo Ageing & health study (SPAH). Osteoporos Int 24(2):595–603
10. Hwang B, Lim JY, Lee J, Choi NK, Ahn YO, Park BJ (2012) Prevalence rate and associated factors of sarcopenic obesity in Korean elderly population. J Korean Med Sci 27(7):748–755
11. Gandham A, Mesinovic J, Jansons P, Zengin A, Bonham MP, Ebeling PR et al (2021) Falls, fractures, and areal bone mineral density in older adults with sarcopenic obesity: a systematic review and meta-analysis. Obes Rev 22(5):e13187
12. Abdelaal M, le Roux CW, Docherty NG (2017) Morbidity and mortality associated with obesity. Ann Transl Med 5(7):161
13. Compston JE, Watts NB, Chapurlat R, Cooper C, Boonen S, Greenspan S et al (2011) Obesity is not protective against fracture in postmenopausal women: GLOW. Am J Med 124(11):1043–50
14. Cao JJ (2011) Effects of obesity on bone metabolism. J Orthop Surg Res 6:30
15. Hruby A, Hu FB (2015) The epidemiology of obesity: a big picture. Pharmacoeconomics 33(7):673–689
16. Granic A, Sayer AA, Robinson SM (2019) Dietary patterns, skeletal muscle health, and sarcopenia in older adults. Nutrients. https://doi.org/10.3390/nu11040745
17. Scott D, Blizzard L, Fell J, Jones G (2011) The epidemiology of sarcopenia in community living older adults: what role does lifestyle play? J Cachexia Sarcopenia Muscle 2(3):125–134
18. Prokopidis K, Cervo MM, Gandham A, Scott D (2020) Impact of protein intake in older adults with sarcopenia and obesity: a gut microbiota perspective. Nutrients. https://doi.org/10.3390/nu12082285
19. Guinane CM, Cotter PD (2013) Role of the gut microbiota in health and chronic gastrointestinal disease: understanding a hidden metabolic organ. Therap Adv Gastroenterol 6(4):295–308
20. Hou K, Wu ZX, Chen XY, Wang JQ, Zhang D, Xiao C et al (2022) Microbiota in health and diseases. Signal Transduct Target Ther 7(1):135
21. Alagiakrishnan K, Morgadinho J, Halverson T (2024) Approach to the diagnosis and management of dysbiosis. Front Nutr 11:1330903
22. Zou S, Fang L, Lee MH (2018) Dysbiosis of gut microbiota in promoting the development of colorectal cancer. Gastroenterol Rep 6(1):1–12
23. Gizard F, Fernandez A, De Vadder F (2020) Interactions between gut microbiota and skeletal muscle. Nutr Metab Insights 13:1178638820980490
24. Mahnic A, Breskvar M, Dzeroski S, Skok P, Pintar S, Rupnik M (2020) Distinct types of gut microbiota dysbiosis in hospitalized gastroenterological patients are disease non-related and characterized with the predominance of either Enterobacteriaceae or Enterococcus. Front Microbiol 11:120
25. Wang L, He X, Zhang Z, Chen N (2025) Distinct gut microbiota signatures in older people with sarcopenic obesity and sarcopenia without obesity. Clin Nutr 49:77–89
26. Bulut O, Temba GS, Koeken V, Moorlag S, de Bree LCJ, Mourits VP et al (2024) Common and distinct metabolomic markers related to immune aging in Western European and East African populations. Mech Ageing Dev 218:111916
27. Du Y, He C, An Y, Huang Y, Zhang H, Fu W et al (2024) The role of short chain fatty acids in inflammation and body health. Int J Mol Sci. https://doi.org/10.3390/ijms25137379
28. Jandhyala SM, Talukdar R, Subramanyam C, Vuyyuru H, Sasikala M, Nageshwar Reddy D (2015) Role of the normal gut microbiota. World J Gastroenterol 21(29):8787–8803
29. de Vos WM, Tilg H, Van Hul M, Cani PD (2022) Gut microbiome and health: mechanistic insights. Gut 71(5):1020–1032
30. Liu C, Cheung WH, Li J, Chow SK, Yu J, Wong SH et al (2021) Understanding the gut microbiota and sarcopenia: a systematic review. J Cachexia Sarcopenia Muscle 12(6):1393–407

31. de Marco Castro E, Murphy CH, Roche HM (2021) Targeting the gut microbiota to improve dietary protein efficacy to mitigate sarcopenia. Front Nutr 8:656730
32. Buigues C, Fernandez-Garrido J, Pruimboom L, Hoogland AJ, Navarro-Martinez R, Martinez-Martinez M et al (2016) Effect of a prebiotic formulation on frailty syndrome: a randomized, double-blind clinical trial. Int J Mol Sci. https://doi.org/10.3390/ijms17060932
33. Macfarlane S, Cleary S, Bahrami B, Reynolds N, Macfarlane GT (2013) Synbiotic consumption changes the metabolism and composition of the gut microbiota in older people and modifies inflammatory processes: a randomised, double-blind, placebo-controlled crossover study. Aliment Pharmacol Ther 38(7):804–816
34. Handajani YS, Turana Y, Hengky A, Hamid G, Schroeder-Butterfill E, Kristian K (2024) Probiotics supplementation or probiotic-fortified products on sarcopenic indices in older adults: systematic review and meta-analysis from recent randomized controlled trials. Front Aging 5:1307762
35. Prokopidis K, Giannos P, Kirwan R, Ispoglou T, Galli F, Witard OC et al (2023) Impact of probiotics on muscle mass, muscle strength and lean mass: a systematic review and meta-analysis of randomized controlled trials. J Cachexia Sarcopenia Muscle 14(1):30–44
36. Jaacks LM, Vandevijvere S, Pan A, McGowan CJ, Wallace C, Imamura F et al (2019) The obesity transition: stages of the global epidemic. Lancet Diabetes Endocrinol 7(3):231–40
37. Petermann-Rocha F, Balntzi V, Gray SR, Lara J, Ho FK, Pell JP et al (2022) Global prevalence of sarcopenia and severe sarcopenia: a systematic review and meta-analysis. J Cachexia Sarcopenia Muscle 13(1):86–99
38. Bruyere O, Beaudart C, Ethgen O, Reginster JY, Locquet M (2019) The health economics burden of sarcopenia: a systematic review. Maturitas 119:61–69
39. Cm A (2016) Obesity: definition, comorbidities, causes, and burden. Am J Manag Care 22:s176-85
40. Gandham A, Zengin A, Bonham MP, Winzenberg T, Balogun S, Wu F et al (2020) Incidence and predictors of fractures in older adults with and without obesity defined by body mass index versus body fat percentage. Bone. https://doi.org/10.1016/j.bone.2020.115546
41. Kindler JM, Lappe JM, Gilsanz V, Oberfield S, Shepherd JA, Kelly A et al (2019) Lumbar spine bone mineral apparent density in children: results from the bone mineral density in childhood study. J Clin Endocrinol Metab 104(4):1283–92
42. Byambasukh O, Eisenga MF, Gansevoort RT, Bakker SJ, Corpeleijn E (2019) Body fat estimates from bioelectrical impedance equations in cardiovascular risk assessment: the PREVEND cohort study. Eur J Prev Cardiol 26(9):905–916
43. Batsis JA, Zagaria AB (2018) Addressing obesity in aging patients. Med Clin North Am 102(1):65–85
44. Walls HL, Magliano DJ, Stevenson CE, Backholer K, Mannan HR, Shaw JE et al (2012) Projected progression of the prevalence of obesity in Australia. Obesity 20(4):872–878
45. Dobbs R, Sawers C, Thompson F, Manyika J, Woetzel JR, Child P, et al. (2014) Overcoming obesity: an initial economic analysis: McKinsey global institute
46. Guh DP, Zhang W, Bansback N, Amarsi Z, Birmingham CL, Anis AH (2009) The incidence of co-morbidities related to obesity and overweight: a systematic review and meta-analysis. BMC Public Health 9:88
47. Baumgartner RN, Koehler KM, Gallagher D, Romero L, Heymsfield SB, Ross RR et al (1998) Epidemiology of sarcopenia among the elderly in New Mexico. Am J Epidemiol. https://doi.org/10.1093/oxfordjournals.aje.a009520
48. Cruz-Jentoft AJ, Bahat G, Bauer J, Boirie Y, Bruyere O, Cederholm T et al (2019) Sarcopenia: revised European consensus on definition and diagnosis. Age Ageing 48(1):16–31
49. Cawthon PM, Manini T, Patel SM, Newman A, Travison T, Kiel DP et al (2020) <article-title update="added"> Putative cut-points in sarcopenia components and incident adverse health outcomes: an <scp>SDOC</scp> analysis. J Am Geriatr Soc 68(7):1429–37
50. Chen LK, Woo J, Assantachai P, Auyeung TW, Chou MY, Iijima K, et al. (2020) Asian Working Group for Sarcopenia: 2019 consensus update on sarcopenia diagnosis and treatment. J Am Med Dir Assoc. 21(3):300–7 e2.
51. Ali SR, Nkembo AT, Tipparaju SM, Ashraf M, Xuan W (2024) Sarcopenia: recent advances for detection, progression, and metabolic alterations along with therapeutic targets. Can J Physiol Pharmacol 102(12):697–708
52. Auyeung TW, Lee SW, Leung J, Kwok T, Woo J (2014) Age-associated decline of muscle mass, grip strength and gait speed: a 4-year longitudinal study of 3018 community-dwelling older Chinese. Geriatr Gerontol Int 14(Suppl 1):76–84
53. Bischoff-Ferrari HA, Orav JE, Kanis JA, Rizzoli R, Schlogl M, Staehelin HB et al (2015) Comparative performance of current definitions of sarcopenia against the prospective incidence of falls among community-dwelling seniors age 65 and older. Osteoporos Int 26(12):2793–802
54. Harvey NC, Orwoll E, Kwok T, Karlsson MK, Rosengren BE, Ribom E et al (2021) Sarcopenia definitions as predictors of fracture risk independent of FRAX((R)), falls, and BMD in the osteoporotic fractures in men (MrOS) study: a meta-analysis. J Bone Miner Res 36(7):1235–44
55. Scott D, Hayes A, Sanders KM, Aitken D, Ebeling PR, Jones G (2014) Operational definitions of sarcopenia and their associations with 5-year changes in falls risk in community-dwelling middle-aged and older adults. Osteoporos Int 25(1):187–193
56. Gandham A, Gregori G, Johansson L, Larsson BAM, Johansson H, Harvey NC et al (2024) Sarcopenia definitions and their association with injurious falls in older Swedish women from the Sahlgrenska University Hospital Prospective Evaluation of Risk of Bone fractures (SUPERB) study. Osteoporos Int 35(11):1963–1972
57. Gandham A, Gregori G, Johansson L, Johansson H, Harvey NC, Vandenput L et al (2024) Sarcopenia definitions and their association with fracture risk in older Swedish women. J Bone Miner Res 39(4):453–61
58. Roh E, Choi KM (2020) Health consequences of sarcopenic obesity: a narrative review. Front Endocrinol (Lausanne) 11:332
59. Schluessel S, Huemer MT, Peters A, Drey M, Thorand B (2023) Sarcopenic obesity using the ESPEN and EASO consensus statement criteria of 2022 - results from the German KORA-Age study. Obes Res Clin Pract 17(4):349–352
60. Donini LM, Busetto L, Bischoff SC, Cederholm T, Ballesteros-Pomar MD, Batsis JA (2022) Definition and diagnostic criteria for sarcopenic obesity: ESPEN and EASO consensus statement. Obes Facts 15:1
61. Park JE, Lee S, Kim K (2023) The effect of combining nutrient intake and physical activity levels on central obesity, sarcopenia, and sarcopenic obesity: a population-based cross-sectional study in South Korea. BMC Geriatr 23(1):119
62. Hsu KJ, Liao CD, Tsai MW, Chen CN (2019) Effects of exercise and nutritional intervention on body composition, metabolic health, and physical performance in adults with sarcopenic obesity: a meta-analysis. Nutrients. https://doi.org/10.3390/nu11092163
63. Sanchez YSdlBB, Martinez Carrillo BE, Aguirre Garrido JF, Martinez Mendez R, Benitez Arciniega AD, Valdes Ramos R, et al. (2022) Emerging evidence on the use of probiotics

and prebiotics to improve the gut microbiota of older adults with frailty syndrome: a narrative review. J Nutr Health Aging. 26(10):926–35.

64. Li W, Sheng RW, Cao MM, Rui YF (2024) Exploring the relationship between gut microbiota and sarcopenia based on gut-muscle axis. Food Sci Nutr 12(11):8779–8792
65. Kelso AL (2024) The effects of aging on gut microbiome composition and association with age-related disease states: a literature review. Undergraduate Research in Natural and Clinical Science and Technology (URNCST) Journal 8(5):1–10
66. Salazar J, Duran P, Diaz MP, Chacin M, Santeliz R, Mengual E et al (2023) Exploring the relationship between the gut microbiota and ageing: a possible age modulator. Int J Environ Res Public Health. https://doi.org/10.3390/ijerph20105845
67. Shen Y, Fan N, Ma SX, Cheng X, Yang X, Wang G (2025) Gut microbiota dysbiosis: pathogenesis, diseases, prevention, and therapy. MedComm (2020) 6(5):e70168
68. Abenavoli L, Scarpellini E, Colica C, Boccuto L, Salehi B, Sharifi-Rad J et al (2019) Gut microbiota and obesity: a role for probiotics. Nutrients. https://doi.org/10.3390/nu11112690
69. Xu Z, Jiang W, Huang W, Lin Y, Chan FKL, Ng SC (2022) Gut microbiota in patients with obesity and metabolic disorders - a systematic review. Genes Nutr 17(1):2
70. Santoro A, Zhao J, Wu L, Carru C, Biagi E, Franceschi C (2020) Microbiomes other than the gut: inflammaging and age-related diseases. Semin Immunopathol 42(5):589–605
71. Fischer N, Relman DA (2018) *Clostridium difficile*, aging, and the gut: can microbiome rejuvenation keep us young and healthy? J Infect Dis 217(2):174–6
72. Lim MY, Nam YD (2023) Gut microbiome in healthy aging versus those associated with frailty. Gut Microbes 15(2):2278225
73. Kazarina A, Kuzmicka J, Bortkevica S, Zayakin P, Kimsis J, Igumnova V et al (2023) Oral microbiome variations related to ageing: possible implications beyond oral health. Arch Microbiol 205(4):116
74. White AE, Ciampa N, Chen Y, Kirk M, Nesbitt A, Bruce BB et al (2019) Characteristics of *Campylobacter* and *Salmonella* infections and acute gastroenteritis in older adults in Australia, Canada, and the United States. Clin Infect Dis 69(9):1545–52
75. Ghosh TS, Shanahan F, O'Toole PW (2022) The gut microbiome as a modulator of healthy ageing. Nat Rev Gastroenterol Hepatol 19(9):565–84
76. Ogawa T, Hirose Y, Honda-Ogawa M, Sugimoto M, Sasaki S, Kibi M et al (2018) Composition of salivary microbiota in elderly subjects. Sci Rep 8(1):414
77. Lordan C, Thapa D, Ross RP, Cotter PD (2020) Potential for enriching next-generation health-promoting gut bacteria through prebiotics and other dietary components. Gut Microbes 11(1):1–20
78. DeJong EN, Surette MG, Bowdish DME (2020) The gut microbiota and unhealthy aging: disentangling cause from consequence. Cell Host Microbe 28(2):180–189
79. Zhang T, Cheng JK, Hu YM (2022) Gut microbiota as a promising therapeutic target for age-related sarcopenia. Ageing Res Rev 81:101739
80. Sah DK, Arjunan A, Park SY, Jung YD (2022) Bile acids and microbes in metabolic disease. World J Gastroenterol 28(48):6846–6866
81. Wang Y, Li Y, Bo L, Zhou E, Chen Y, Naranmandakh S et al (2023) Progress of linking gut microbiota and musculoskeletal health: casualty, mechanisms, and translational values. Gut Microbes 15(2):2263207
82. Liu BN, Liu XT, Liang ZH, Wang JH (2021) Gut microbiota in obesity. World J Gastroenterol 27(25):3837–3850
83. Geng J, Ni Q, Sun W, Li L, Feng X (2022) The links between gut microbiota and obesity and obesity related diseases. Biomed Pharmacother 147:112678
84. Pellegrino A, Coppola G, Santopaolo F, Gasbarrini A, Ponziani FR (2023) Role of *Akkermansia* in human diseases: from causation to therapeutic properties. Nutrients. https://doi.org/10.3390/nu15081815
85. Yarahmadi A, Afkhami H, Javadi A, Kashfi M (2024) Understanding the complex function of gut microbiota: its impact on the pathogenesis of obesity and beyond: a comprehensive review. Diabetol Metab Syndr 16(1):308
86. Breton J, Galmiche M, Dechelotte P (2022) Dysbiotic gut bacteria in obesity: an overview of the metabolic mechanisms and therapeutic perspectives of next-generation probiotics. Microorganisms. https://doi.org/10.3390/microorganisms10020452
87. Mai X, Yang S, Chen Q, Chen K (2024) Gut microbial composition is altered in sarcopenia: a systematic review and meta-analysis of clinical studies. PLoS One 19(8):e0308360
88. Wang G, Li Y, Liu H, Yu X (2025) Gut microbiota in patients with sarcopenia: a systematic review and meta-analysis. Front Microbiol 16:1513253
89. Wu Y, Xia Y, Huang S, Liu S, Yang J, Yang Y et al (2022) The composition of the gut microbiome in patients with sarcopenia. Turk J Biochem 47(3):325–32
90. Bilski J, Pierzchalski P, Szczepanik M, Bonior J, Zoladz JA (2022) Multifactorial mechanism of sarcopenia and sarcopenic obesity. Role of physical exercise, microbiota and myokines. Cells. https://doi.org/10.3390/cells11010160
91. Hoseini Tavassol Z, Ejtahed HS, Atlasi R, Saghafian F, Khalagi K, Hasani-Ranjbar S et al (2023) Alteration in gut microbiota composition of older adults is associated with obesity and its indices: a systematic review. J Nutr Health Aging 27(10):817–23
92. Lapauw L, Rutten A, Dupont J, Amini N, Vercauteren L, Derrien M et al (2024) Associations between gut microbiota and sarcopenia or its defining parameters in older adults: a systematic review. J Cachexia Sarcopenia Muscle 15(6):2190–2207
93. Hosseini FS, Behrouzi A, Shafaie E, Sharifi F, Ejtahed HS (2025) Assessment of gut microbiota in the elderly with sarcopenic obesity: a case-control study. J Diabetes Metab Disord 24(1):83
94. Baldelli V, Scaldaferri F, Putignani L, Del Chierico F (2021) The role of *Enterobacteriaceae* in gut microbiota dysbiosis in inflammatory bowel diseases. Microorganisms. https://doi.org/10.3390/microorganisms9040697
95. Sun L, Yu Z, Ye X, Zou S, Li H, Yu D et al (2010) A marker of endotoxemia is associated with obesity and related metabolic disorders in apparently healthy Chinese. Diabetes Care 33(9):1925–1932
96. Antuna E, Cachan-Vega C, Bermejo-Millo JC, Potes Y, Caballero B, Vega-Naredo I et al (2022) Inflammaging: implications in sarcopenia. Int J Mol Sci. https://doi.org/10.3390/ijms232315039
97. Schaap LA, Pluijm SM, Deeg DJ, Visser M. (2006) Inflammatory markers and loss of muscle mass (sarcopenia) and strength. Am J Med. 119(6):526 e9–17.
98. Stojanov S, Berlec A, Strukelj B. (2020) The influence of probiotics on the Firmicutes/Bacteroidetes ratio in the treatment of obesity and inflammatory bowel disease. Microorganisms. 8(11):1715
99. Fusco W, Lorenzo MB, Cintoni M, Porcari S, Rinninella E, Kaitsas F et al (2023) Short-chain fatty-acid-producing bacteria: key components of the human gut microbiota. Nutrients. https://doi.org/10.3390/nu15092211

100. Ecklu-Mensah G, Choo-Kang C, Maseng MG, Donato S, Bovet P, Viswanathan B et al (2023) Gut microbiota and fecal short chain fatty acids differ with adiposity and country of origin: the METS-microbiome study. Nat Commun 14(1):5160
101. Mazhar M, Zhu Y, Qin L (2023) The interplay of dietary fibers and intestinal microbiota affects type 2 diabetes by generating short-chain fatty acids. Foods. https://doi.org/10.3390/foods12051023
102. van Deuren T, Blaak EE, Canfora EE (2022) Butyrate to combat obesity and obesity-associated metabolic disorders: current status and future implications for therapeutic use. Obes Rev 23(10):e13498
103. Zhou J, Liu J, Lin Q, Shi L, Zeng Z, Guan L et al (2023) Characteristics of the gut microbiome and metabolic profile in elderly patients with sarcopenia. Front Pharmacol 14:1279448
104. Zhang Y, Zhu Y, Guo Q, Wang W, Zhang L (2023) High-throughput sequencing analysis of the characteristics of the gut microbiota in aged patients with sarcopenia. Exp Gerontol 182:112287
105. Parada Venegas D, De la Fuente MK, Landskron G, Gonzalez MJ, Quera R, Dijkstra G et al (2019) Short chain fatty acids (SCFAs)-mediated gut epithelial and immune regulation and its relevance for inflammatory bowel diseases. Front Immunol 10:277
106. Tan J, Taitz J, Nanan R, Grau G, Macia L (2023) Dysbiotic gut microbiota-derived metabolites and their role in non-communicable diseases. Int J Mol Sci. https://doi.org/10.3390/ijms242015256
107. Liu J, Tan Y, Cheng H, Zhang D, Feng W, Peng C (2022) Functions of gut microbiota metabolites, current status and future perspectives. Aging Dis 13(4):1106–1126
108. Zhang D, Jian YP, Zhang YN, Li Y, Gu LT, Sun HH et al (2023) Short-chain fatty acids in diseases. Cell Commun Signal 21(1):212
109. Shin HE, Won CW, Kim M (2022) Metabolomic profiles to explore biomarkers of severe sarcopenia in older men: a pilot study. Exp Gerontol 167:111924
110. Ciesielska A, Matyjek M, Kwiatkowska K (2021) TLR4 and CD14 trafficking and its influence on LPS-induced pro-inflammatory signaling. Cell Mol Life Sci 78(4):1233–1261
111. Biagioli M, Di Giorgio C, Massa C, Marchiano S, Bellini R, Bordoni M et al (2024) Microbial-derived bile acid reverses inflammation in IBD via GPBAR1 agonism and RORgammat inverse agonism. Biomed Pharmacother 181:117731
112. Hong SH, Choi KM (2020) Sarcopenic obesity, insulin resistance, and their implications in cardiovascular and metabolic consequences. Int J Mol Sci. https://doi.org/10.3390/ijms21020494
113. Fielding RA, Reeves AR, Jasuja R, Liu C, Barrett BB, Lustgarten MS (2019) Muscle strength is increased in mice that are colonized with microbiota from high-functioning older adults. Exp Gerontol 127:110722
114. Yang W, Si SC, Wang WH, Li J, Ma YX, Zhao H et al (2025) Gut dysbiosis in primary sarcopenia: potential mechanisms and implications for novel microbiome-based therapeutic strategies. Front Microbiol 16:1526764
115. Lawenius L, Cowardin C, Grahnemo L, Scheffler JM, Horkeby K, Engdahl C et al (2023) Transplantation of gut microbiota from old mice into young healthy mice reduces lean mass but not bone mass. Gut Microbes 15(1):2236755
116. Mo X, Shen L, Cheng R, Wang P, Wen L, Sun Y et al (2023) Faecal microbiota transplantation from young rats attenuates age-related sarcopenia revealed by multiomics analysis. J Cachexia Sarcopenia Muscle 14(5):2168–83
117. Kim KH, Chung Y, Huh JW, Park DJ, Cho Y, Oh Y et al (2022) Gut microbiota of the young ameliorates physical fitness of the aged in mice. Microbiome 10(1):238
118. Yuzefpolskaya M, Bohn B, Ladanyi A, Pinsino A, Braghieri L, Carey MR et al (2024) Alterations in the sarcopenia index are associated with inflammation, gut, and oral microbiota among heart failure, left ventricular assist device, and heart transplant patients. J Heart Lung Transplant 43(9):1395–408
119. Wang B, Hu W, Zhang X, Cao Y, Shao L, Xu X et al (2023) Sarcopenia and gut microbiota alterations in patients with hematological diseases before and after hematopoietic stem cell transplantation. Chin J Cancer Res 35(4):386–98
120. Pu Y, Sun Z, Zhang H, Huang Q, Wang Z, Mei Z et al (2024) Gut microbial features and circulating metabolomic signatures of frailty in older adults. Nat Aging 4(9):1249–1262
121. Wijma AG, Bongers BC, Annema C, Dekker R, Blokzijl H, van der Palen JA et al (2024) 'Effects of a home-based bimodal lifestyle intervention in frail patients with end-stage liver disease awaiting orthotopic liver transplantation': study protocol of a non-randomised clinical trial. BMJ Open 14(1):e080430
122. Murata H, Zhang Q, Ito T, Kawakami R, Sakamoto S, Torii S et al (2023) The combined effect of rowing exercise and the intake of functional foods containing inulin on muscle mass and bone mineral density in older Japanese women. Geriatr Gerontol Int 23(11):779–87
123. Neto JV, de Melo CM, Ribeiro SM (2013) Effects of three-month intake of synbiotic on inflammation and body composition in the elderly: a pilot study. Nutrients 5(4):1276–1286
124. S R, R S, N S. (2015) The effects of symbiotic therapy on anthropometric measures, body composition and blood pressure in patient with metabolic syndrome: a triple blind RCT. Medical Journal of the Islamic Republic of Iran 29(213).
125. Davani-Davari D, Negahdaripour M, Karimzadeh I, Seifan M, Mohkam M, Masoumi SJ et al (2019) Prebiotics: definition, types, sources, mechanisms, and clinical applications. Foods. https://doi.org/10.3390/foods8030092
126. Karim A, Khan HA, Iqbal MS, Ahmad F, Qaisar R (2024) Probiotics' supplementation alleviates disease severity and improves postural balance by repairing intestinal leak in patients suffering from osteoarthritis: a double-blinded clinical trial. Br J Nutr. https://doi.org/10.1017/S0007114524002824
127. Fortuna R, Wang W, Mayengbam S, Tuplin EWN, Sampsell K, Sharkey KA et al (2024) Effect of prebiotic fiber on physical function and gut microbiota in adults, mostly women, with knee osteoarthritis and obesity: a randomized controlled trial. Eur J Nutr 63(6):2149–2161
128. Yang J, Hou L, Wang A, Shang L, Jia X, Xu R et al (2024) Prebiotics improve frailty status in community-dwelling older individuals in a double-blind, randomized, controlled trial. J Clin Invest. https://doi.org/10.1172/JCI176507
129. Ni Lochlainn M, Bowyer RCE, Moll JM, Garcia MP, Wadge S, Baleanu AF et al (2024) Effect of gut microbiome modulation on muscle function and cognition: the PROMOTe randomised controlled trial. Nat Commun 15(1):1859
130. Wang X, Qi Y, Zheng H (2022) Dietary polyphenol, gut microbiota, and health benefits. Antioxidants. https://doi.org/10.3390/antiox11061212
131. Monda V, Villano I, Messina A, Valenzano A, Esposito T, Moscatelli F et al (2017) Exercise modifies the gut microbiota with positive health effects. Oxid Med Cell Longev 2017:3831972
132. Ibrahim NS, Muhamad AS, Ooi FK, Meor-Osman J, Chen CK (2018) The effects of combined probiotic ingestion and circuit training on muscular strength and power and cytokine responses in young males. Appl Physiol Nutr Metab 43(2):180–186

133. Goisser S, Kemmler W, Porzel S, Volkert D, Sieber CC, Bollheimer LC et al (2015) Sarcopenic obesity and complex interventions with nutrition and exercise in community-dwelling older persons–a narrative review. Clin Interv Aging 10:1267–1282
134. Zheng Y, Zhang Z, Tang P, Wu Y, Zhang A, Li D et al (2023) Probiotics fortify intestinal barrier function: a systematic review and meta-analysis of randomized trials. Front Immunol 14:1143548
135. Victoria Obayomi O, Folakemi Olaniran A, Olugbemiga Owa S (2024) Unveiling the role of functional foods with emphasis on prebiotics and probiotics in human health: a review. J Funct Foods. https://doi.org/10.1016/j.jff.2024.106337
136. Gomez Quintero DF, Kok CR, Hutkins R (2022) The future of synbiotics: rational formulation and design. Front Microbiol 13:919725
137. Wang Y, Lei P (2025) Efficacy of probiotic supplements in the treatment of sarcopenia: a systematic review and meta-analysis. PLoS One 20(2):e0317699

Authors and Affiliations

Anoohya Gandham[1,2] · Konstantinos Prokopidis[3] · Costas Glavas[2,4] · David Scott[2,4] · Mattias Lorentzon[5,6]

✉ Mattias Lorentzon
mattias.lorentzon@medic.gu.se

Anoohya Gandham
anoohya.gandham@acu.edu.au

[1] Mary MacKillop Institute for Health Research, Australian Catholic University, Victoria, Australia

[2] Department of Medicine, School of Clinical Sciences at Monash Health, Monash University, Clayton, VIC, Australia

[3] Department of Musculoskeletal and Ageing Science, Institute of Life Course and Medical Sciences, University of Liverpool, Liverpool, UK

[4] School of Exercise and Nutrition Sciences, Institute for Physical Activity and Nutrition (IPAN), Deakin University, Geelong, VIC, Australia

[5] Sahlgrenska Osteoporosis Centre, Institute of Medicine, University of Gothenburg, Göteborg, Sweden

[6] Geriatric Medicine, Region Västra Götaland, Sahlgrenska University Hospital, Mölndal, Sweden