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The Gut–Muscle Axis in Age-Related Sarcopenia: Cause, Consequence, or Modifier?

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Abstract: Sarcopenia is an age-related, progressive loss of skeletal muscle mass, strength, and physical performance, increasing the risk of falls, hospitalization, loss of independence, and mortality among older adults. Recent research highlights the gut–muscle axis, through which gut microbiota communicate with skeletal muscle via metabolites and signalling pathways. This review examines animal mechanistic studies, intervention trials, and human observational research investigating the relationship between gut microbiota and sarcopenia in older adults. Key areas include short-chain fatty acids, bile acids, urolithin A, and tryptophan-derived metabolites as potential signalling molecules, alongside gut barrier integrity and low-grade inflammation as possible mediating pathways. Nutritional strategies, exercise, probiotics, prebiotics, synbiotics, postbiotics, and faecal microbiota transplantation are explored as potential interventions. Human observational studies generally indicate that individuals with sarcopenia exhibit reduced gut microbial diversity and fewer butyrate-producing taxa, although specific microbial profiles vary across studies. However, most observational evidence cannot establish causal relationships. Several intervention trials have reported measurable improvements in muscle strength or physical performance following supplementation with specific probiotic strains, Akkermansia-based postbiotics, prebiotics, and purified urolithin A. Nevertheless, the observed benefits are generally modest, follow-up periods are limited, and large-scale clinical trials remain lacking. Current evidence suggests that the gut microbiome may be a modifiable contributor to sarcopenia within the broader biological processes of ageing rather than an established independent cause. Future research should prioritize longitudinal studies, standardized microbiome assessment, mechanistic investigations, and adequately powered randomized controlled trials to establish causal relationships, identify effective interventions, and determine their long-term clinical relevance in preventing and managing sarcopenia among older adults.

Keywords: Sarcopenia; Gut–muscle axis; Gut microbiome; Older adults; Skeletal muscle health.

INTRODUCTION

Sarcopenia is now established as a separate clinical entity and not just a side effect of ageing. The European Working Group on Sarcopenia in Older People (EWGSOP2) set a new consensus on sarcopenia the following year, which is very similar to the one defined by the Asian Working Group on Sarcopenia (AWGS) (Cruz-Jentoft et al., 2019; Chen et al., 2020). The two frameworks employ slightly different cut-points and assessment methods, so measurement heterogeneity is one of the more persistent challenges in the field that makes it difficult to compare studies and ultimately, to compare the very cohorts that this review relies on (Dent et al., 2021). A systematic review and meta-analysis reported that the prevalence of sarcopenia was estimated to be between 10 and 27% in community-dwelling adults aged over 60 years before and significantly higher in hospitalised and institutionalised older adults (Petermann-Rocha et al., 2022). The aging process of the microbiome goes hand-in-hand with the aging process of its host. The microbiome's aging process is coupled with its host's aging process.

The human gut microbiome is not a static background, but rather undergoes significant changes throughout life. In one of the earliest studies in the field, the faecal microbiota of 178 Irish adults over 65 years of age was described, and was found to be broadly similar to that of younger adults in community-dwelling seniors, but less diverse in residents of long-term facilities that were associated with clinical indicators of frailty (Claesson et al., 2012). This followed the previous widely discussed discovery that the human gut microbiome undergoes a relatively stable evolution from infancy to old age, over distinct populations and geographical regions (Yatsunenko et al., 2012). On the opposite side of the age continuum, a group of Italian studies on centenarians identified a special remodelling of the Firmicutes population and

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an increase of facultative anaerobes that was accompanied by a decrease in Faecalibacterium prausnitzii and its relatives, typically linked to anti-inflammatory activity (Biagi et al., 2010).

This trajectory was mapped out in greater taxonomic detail in a large cross-sectional Japanese study that ranged from newborns to centenarians (Odamaki et al., 2016), and more recent research comparing the very old with the young and the old with the very old found that centenarians who were healthy had some youth-like microbial characteristics that were absent in less healthy ageing peers (Santoro et al., 2018). Wilmanski and colleagues carried out an even more dramatic analysis, finding that the microbiome actually diverges and individualises throughout old age and not towards a general 'elderly' profile - and that this has a very direct association with survival - an unusual direct connection between microbiome trajectory and a hard clinical outcome (Wilmanski et al., 2021). Considering this body of literature, Ghosh, Shanahan and O'Toole have suggested that there are no universal definitions for a 'healthy' ageing microbiome either, only definitions that maintain sufficient functional capacity and resilience (Ghosh, Shanahan, & O'Toole, 2022).

The concept of an old microbiome in the gut directly communicating with skeletal muscle is a relatively new notion. They proposed that, as the gut ages, tight junctions become damaged, allowing increased amounts of LPS and other microbial products to enter the circulation, and this endotoxin burden is “Well suited to contribute to the inflammatory and metabolic changes associated with ageing muscle” (Grosicki, Fielding, & Lustgarten, 2018). A mouse study around the same time reported by the Bowdish lab virtually a proof of principle for this mechanism: germ-free mice do not develop the typical rise in circulating inflammatory cytokines with age, but co-housing them with old (not young) conventional mice restores this age-related increase, and blocking it with anti-TNF therapy does so as well – which was a pretty compelling demonstration that it is not age per se but rather the aged microbiota that drives part of that inflammatory phenotype (Thevaranjan et al., 2017).

Ticinesi and colleagues was one of the first to explicitly state the specific hypothesis for sarcopenia, that dysbiosis may be a driver of muscle loss due to nutrient malabsorption, chronic inflammation and disruption of anabolic signalling (Ticinesi et al., 2017) and later the same Parma group reviewed the growing body of evidence for a bi-directional relationship between dysbiosis and frailty (Ticinesi et al., 2019). With higher resolution than the 16S rRNA sequencing that is the common denominator of the literature, they then characterised taxonomic and functional differences between the two groups of older adults, and proceeded to perform a proof-of-concept human study (Ticinesi et al., 2020). Liu and colleagues carried out a systematic review of 26 pre-clinical and 10 clinical studies, and concluded that the causal evidence is reasonably strong from animal studies, especially the faecal transplants, and largely associative from the human studies (Liu et al., 2021). Cross-ing of germ-free mice with donor microbiota from high- versus low-functioning older adults revealed a measurable difference in grip strength between high- and low-functioning groups in recipient mice (Fielding et al., 2019), and transplant of young rat microbiota into aged rats led to improvements in muscle mass, strength, exercise capacity and mitochondrial markers across a multi-omics approach (Mo et al., 2023).

2 HUMAN EVIDENCE: WHAT DO CROSS-SECTIONAL AND CASE-CONTROL STUDIES ACTUALLY REVEAL?

The vast majority of human literature is cross-sectional or case-control, and it is important to remember that the relationship between gut microbiota and sarcopenia is both valid and consistent, but the specific bacteria that are involved are extremely variable, depending on the study. However, in an older adult population from Korea, the prevalence of sarcopenia correlated with decreased Prevotella and Prevotella copri and increased Parabacteroides (Lee et al., 2022). The Xiangya Sarcopenia Study, a population-based shotgun metagenomic study in rural China, identified its own unique taxonomic and functional signature (Wang et al., 2022).

A Taiwanese case-control study identified something more mechanistically specific – lower levels of faecal butyrate and changes in its composition in those with low skeletal muscle mass index, implicating a specific deficiency in short-chain fatty acid production, but not any one named organism (Han et al., 2022). In older women, a diet nutrient intake association was reported with the microbiota profile in the same individuals regardless of their sarcopenia status (Yan et al., 2023), and another high-throughput sequencing-based study in elderly sarcopenic patients found a different microbiota pattern in the same age group (Zhang et al., 2023). One of the earlier and still commonly cited case-control studies (Kang et al., 2021) sequenced stool from 60 healthy controls and 27 older adults who were sarcopenic or possibly sarcopenic, and found a systematic depletion of known butyrate-producing genera - Lachnospira, Fusicatenibacter, Roseburia, Eubacterium and Lachnoclostridium - along with a functional shift toward lipopolysaccharide biosynthesis pathways in the sarcopenic group, which the authors interpreted as evidence of a pro-inflammatory metagenome.

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The most useful single reference point here is probably the systematic review by Lapauw et al. that synthesised 32 different studies and over 10,000 participants, finding that associations between the gut microbiota and the defining parameters of sarcopenia are reported repeatedly, but that the specific taxa identified were not reproducible across studies for a single 'sarcopenia signature' (Lapauw et al., 2024). Sex seems to be part of the reason for this heterogeneity. A large Japanese outpatient cohort revealed clear differences in microbiota-sarcopenia associations between men and women, suggesting that sex should be considered as a nuisance variable if not routinely (Asaoka et al., 2025).

Most work on Frailty is related to but not synonymous with Sarcopenia, and a 2026 study in Swedish women aged 75-80 (n=2,081) and independently replicated in a Chinese cohort (n=2,088) showed that the composite Frailty Mortality Index was associated with decreased microbial diversity and gene richness, and predicted mortality, hip fracture and injurious falls better than the Charlson Comorbidity Index (Geraldi et al., 2026). It is one of the few studies that has true hard outcome power and is replicated and size, should not be taken lightly.

A study in the US, using a Dietary Index for Gut Microbiota to assess diet in over 12,900 middle-aged and older adults of diverse ethnic backgrounds, showed that diet predicted to have a positive impact on the gut microbiome was associated with reduced odds of frailty, bringing a large dataset of ethnically diverse adults in the Western world to the table, which is otherwise dominated by East Asian and European cohorts (Li et al., 2025).

The Italian study BIOSPHERE, which specifically studied the relation between physical frailty and sarcopenia (PF&S), instead, revealed a different microbial fingerprint: increased abundance of Oscillospira and Ruminococcus together with decreased abundances of Barnesiellaceae and Christensenellaceae in the participants, alongside metabolic and inflammatory changes typical of the whole inflammaging syndrome (Picca et al., 2019). In a Chinese study, composition of intestinal flora was also shown to change, and an inflammatory index reflecting skeletal muscle mass was reported to correlate with the loss of muscle mass in elderly sarcopenic patients (Lou et al., 2024), and, in a 2023 study, the relative abundance of Prevotella copri was specifically linked to a lower rate of muscle mass loss and improved functional preservation (Liu et al., 2023), which was then tested experimentally in mice. An independently conducted line of research identified that large daily fluctuations in temperature were correlated with sarcopenia prevalence in humans and experimentally induced sarcopenia in mice through an environment-microbiota-dependent pathway not typically linked to sarcopenia (Liu et al., 2023).

A study of the ethnic minority population of the Uyghur ethnic group in China (Zhang et al., 2024) also revealed their own diversity, compositional and functional changes associated with sarcopenia, and a study of adults in Turkey (Wu et al., 2022) observed a similar collection of compositional changes in their sarcopenic population. Another study from China with a wider metabolic profile scope identified differences in gut microbiome and circulating metabolites in elderly sarcopenic patients (Zhou et al., 2023). Finally, genetic epidemiology has come into play: a Mendelian randomisation analysis found associations between specific gut microbial taxa and traits, which in principle are less susceptible to reverse causation than a simple cross-sectional comparison (Zhao et al., 2023). Taking all of this together, the systematic review and meta-analysis done by Lapauw's group the year before, and now the group of Wang et al., 2025, arrived at the conclusion that there exists a real association, but the relationship is not taxonomically consistent, between gut microbiota and sarcopenia in available human studies.

3 MECHANISTIC PATHWAYS: HOW CAN THE GUT REALLY COMMUNICATE WITH THE MUSCLE?

3.1 Short-chain fatty acids

Production of short-chain fatty acids (SCFAs) acetate, propionate and butyrate, occurs when gut bacteria ferment resistant starch and dietary fibre which is not metabolized by the human gut, and are the most characterized class of gut-derived signal in this context (Louis & Flint, 2017). Butyrate in particular has anti-inflammatory and pro-anabolic activity, as well as an inhibitory effect on histone deacetylases, and may also have an effect on bone formation via its influence on regulatory T-cell signalling, a pathway with clear relevance to the broader musculoskeletal system in ageing, given the shared biology of bone and muscle (Tyagi et al., 2018). As noted above, the ubiquitous association of lower faecal butyrate and the reduced prevalence of butyrate-producing taxa in sarcopenic cohorts (Han et al., 2022; Kang et al., 2021) is consistent with this mechanism – although it should be noted that nearly all of the human SCFA data are correlational rather than interventional.

3.2 Bile acids

In the gut, chemically modified by microbes, bile acids can become secondary bile acids, which can function as true signalling molecules, working through the nuclear receptor FXR and membrane receptor TGR5 expressed in skeletal

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muscle (Jia, Liu, & Liu, 2025). In summary, these pathways are generally regarded as being pro- or anti-metabolic to muscle at physiological and pathological concentrations, respectively, bile acids like deoxycholic and cholic acid cause mitochondrial dysfunction in cultured myotubes, which leads to lower mitochondrial mass and higher oxidative stress (Abrigo et al., 2022) – gut metabolites are not "good" or "bad" they are dose-dependent and context-dependent. This finding cannot be directly extrapolated to sarcopenia in humans, except for those who have liver cirrhosis, in which liver bile acid handling is already disrupted (Aliwa et al., 2023).

3.3 Urolithin A and mitochondrial health.

Urolithin A is generated from ellagitannins and ellagic acid found in pomegranate, walnuts and berries, and while there is high variability in bacterial capability to make urolithin A from ellagic acid, direct oral supplementation with purified urolithin A is mechanistically different than the natural production of urolithin A via the gut bacteria, and should not be confused. In a randomised, placebo controlled trial of middle-aged and older adults, 4-months of urolithin A supplementation resulted in an approximate 12% increase in muscle strength as well as improved aerobic endurance and mitochondrial health markers (Singh et al., 2022); a separate trial in older adults found improvements in muscle aerobic endurance and mitochondrial health, but not all pre-specified endpoints were significant (Liu et al., 2022). These are some of the cleaner, downstream, interventional pieces of evidence in the entire field, but they're acting on a purified downstream metabolite, not on some manipulation of the gut microbiome directly, and so this is evidence that the metabolite is important, but not of the gut microbiome being the limiting factor for most people.

3.4 Tryptophan-derived metabolites

Food derived tryptophan is metabolized by gut bacteria into indole derivatives, and host tissues produce kynurenine-pathway metabolites from tryptophan; both categories are ligands for the aryl hydrocarbon receptor which is expressed in skeletal muscle. Levels of circulating kynurenine increase with age, and experimentally increasing kynurenine, leads to muscle atrophy and lipid peroxidation in animal models by stimulating aryl hydrocarbon receptor (Kaiser et al., 2019). In the extreme case of this pathway, indoxyl sulfate (a bacterially produced indole metabolite that builds up in the blood in cases of renal insufficiency) enhances skeletal muscle atrophy by upregulating the expression of myostatin and atrogin-1 through oxidative stress (Enoki et al., 2016). The question whether similarly-sized effects play a role in a normal geriatric sarcopenia, when the kidneys do not induce the accumulation of indole to pathological levels, is really a matter of speculation.

3.5 The gut barrier integrity, endotoxin and inflammation.

The proposed pathway that is repeated in this literature is: dysbiosis, breakdown of the intestinal barrier, translocation of LPS and other microbial products to blood circulation, systemic low grade inflammation, catabolism of muscles. The mucin degrading Akkermansia muciniphila, which has been extensively studied in this role, has been shown to have broadly barrier-protective, anti-inflammatory effects in IBD (Zheng et al., 2022), but not in all disease contexts studied (and in keeping with the general theme of this literature, abundance does not uniformly mean barrier protection). As further discussed below, the Mediterranean-diet NU-AGE trial demonstrated that a year of increased adherence to a Mediterranean diet in more than 600 older Europeans non-frail and pre-frail, achieved through dietary counseling, resulted in an observable shift of the gut microbiome toward a profile with greater predicted production of short-chain fatty acids (SCFAs) and decreased production of putrefactive, pro-inflammatory metabolites, as well as measurable decreases of frailty markers. A diet's overall inflammatory potential, without the microbiome measures per se, was directly related to sarcopenia risk in a Chinese cohort (Bian et al., 2023), so it is important to keep this in mind: diet can influence muscle through the microbiome, but also in parallel to the microbiome.

4. GUT-MUSCLE AXIS AND EXERCISE

In this literature, exercise is in an interesting position, because it can have a direct effect on the muscle as well as go through the microbiome. A 6-week resistance training intervention in older adult males was shown to increase lean mass and strength as expected, with no significant differences in overall microbiome composition, but rather an increased predicted metabolic capacity of the microbiome to produce mucin and a decrease in serum zonulin (a marker of gut barrier integrity), although this did not reach statistical significance, indicating that resistance training may enhance gut barrier integrity, even over a relatively short intervention (Moore et al., 2022). In healthy elderly women, aerobic exercise has been shown to raise abundance of the gut microbiome species, Bacteroides (Morita et al., 2019); in a pilot study, three-arm randomised aerobic, resistance and combined training in healthy older adults was directly compared for its effects on the gut microbiome, although this study has yet to be completed (Merelim et al., 2025).

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Protein supplementation, in combination with resistance exercise, is known to reliably produce changes in body composition and physical function in older people, regardless of any microbiome effect (Liao et al., 2017), while animal studies have shown that resistance training (RT) itself, rather than just aerobic training, has measurable effects on gut microbiota composition in rats (Castro et al., 2021). A targeted metabolite supplement together with exercise is also effective: in a randomized controlled exercise and beta-hydroxy-beta-methylbutyrate (HMB, a metabolite of leucine) trial in older women with low muscle mass, targeted omega-3 supplementation was found to be effective for muscle mass, muscle strength and physical performance (Osuka et al., 2021) indicating that the gut-muscle axis is not an alternative to, but exists alongside, the known anabolic action of exercise and protein.

5. INTERVENTIONS AFFECTING THE GUT-MUSCLE AXIS

5.1 Probiotics

What's been most well-tested is probiotic supplementation, which is promising for certain strains and mixed on the whole. In the meta-analysis, Prokopidis and colleagues (2023) combined the results of 24 studies, revealing that probiotics had a beneficial effect on muscle mass and muscle strength, but did not have a significant effect on total lean mass, and there was moderate to large heterogeneity between trials. It is that heterogeneity that is demonstrated through individual trial levels. Lee et al. (2025) evaluated the impact of a 12-week supplementation with either live Lacticaseibacillus paracasei PS23 or heat-treated L. paracasei PS23, on lower-limb muscle strength and endurance, inflammatory markers, and muscle mass in healthy adults. The authors found that supplementation with L. paracasei PS23, both live and heat-treated, enhanced lower-limb muscle strength and endurance and decreased inflammatory markers, without a significant increase in muscle mass.

In a mouse-accelerated ageing model, the same strain was demonstrated to slow age-related muscle loss by maintaining mitochondrial function, providing a potentially plausible mechanism (Chen et al., 2019). A trial of frail older adults (over 18 years) over 18 weeks with a separate strain, Lactobacillus plantarum TWK10, showed an increase in muscle mass and functional performance (Lee et al., 2021). Following the classic probiotics, an interesting recent study in this area was with a non-viable (prior to pulsing) preparation of Akkermansia muciniphila (HB05P) that increased follistatin, a myostatin-antagonising hormone, and improved the peak torque of leg extensors in a placebo-controlled study of adults > 60yo over 12-weeks (Kang et al., 2024), one of the more mechanistically interesting recent studies in this area because it linked a specific gut organism to a well characterised muscle growth-inhibitory pathway.

5.2 Prebiotics, synbiotics

Prebiotics do not add new bacteria into the body but selectively nourish the beneficial resident bacteria. In a small study but with a well-designed, mechanistically clean design, a 12-week trial of the prebiotic 1-kestose in super-elderly patients with sarcopenia showed a significant increase in skeletal muscle mass index and an increase in the level of Bifidobacterium longum in the intestine in all six patients treated with 1-kestose (Tominaga et al., 2021). A synbiotic trial protocol is currently underway in older Australians that will assess the effects of a combined prebiotic-probiotic formulation on handgrip strength, gait speed, and physical performance, while also having a planned secondary analysis conducted of the microbiome-function associations (Barry et al., 2024). Results from previous studies have demonstrated the benefits of using a different prebiotic formulation on the outcomes of the frailty syndrome in a randomised, double-blind trial (Buigues et al., 2016), of a fortified dairy product on sarcopenia measures in a double-blind, randomised trial (Nasimi et al., 2021), and of three months of synbiotic intake on inflammation and body composition in a small pilot study of elderly participants (Neto et al., 2013).

The most methodologically advanced trial in this particular location is the PROMOTe study from King's College London, which tested a prebiotic (in addition to resistance exercise and branched-chain amino acid supplementation) in 36 pairs of older twins that were randomized to receive either the prebiotic or a placebo (Ni Lochlainn et al., 2021). The published data showed a measurable impact on these muscle and cognitive outcomes, with some of the most direct causal evidence to date that a targeted microbiome intervention, on top of all else that they do, can move a clinically relevant needle (Ni Lochlainn et al., 2024). The field itself has also been trying to formalise its own terminology; the International Scientific Association of Probiotics and Prebiotics (ISAPP) published a consensus definition that separated probiotics from prebiotics, synbiotics and postbiotics as biologically and regulatorily distinct entities, and hence, trials of live vs dead or fragmented organisms vs organism-specific substrates are not easily interchangeable evidence of one another (Salminen et al., 2021).

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5.3 Targeted metabolite and multi-ingredient supplements

A line of work, parallel to the microbiome-modulation one, is supported by the delivery of the metabolites that are relevant to muscles, that are produced in the gut, or that are relevant to gut barrier, directly. In a randomized controlled trial with specifically a focus on the restoration of gut permeability, a patented supplement that included HMB, carnosine, magnesium, butyrate, and lactoferrin, reduced levels of CRP, zonulin, and TNF-alpha levels, while enhancing measures of muscle mass and function, after two months of supplementation (Rondanelli et al., 2024). A previous trial from the same Italian group showed that a supplement containing leucine, omega-3 fatty acids and the probiotic Lactobacillus paracasei PS23, improved appendicular lean mass after two months of supplementation in sarcopenic elderly patients (Rondanelli et al., 2022). These combination treatments will make it more difficult to determine which component is responsible for the effect, but they do have a practical appeal as sarcopenia is not likely to be fixable with one single nutrient.

5.4 Evidence of genetics, causal inference and faecal microbiota transplantation

In addition to observational cohorts, Mendelian randomisation can be used to investigate causal effects which are less prone to reverse causation and dietary confounding, as genetic variants are used as instruments for the exposure and outcome. The relationship between the gut microbiome and inflammatory bowel disease (IBD) and between the gut microbiome and sarcopenia (Liang et al., 2025), as described above in the mechanisms of the inflammation picture, further supports this association; however, this association is more relevant to the IBD population than primary ageing-related sarcopenia. At this time, faecal microbiota transplantation is not suited to clinical use for sarcopenia, as it has been seen to provide real muscle benefits in aged rodents (Mo et al., 2023), but no human FMT study for the primary geriatric sarcopenia is reported to date that has been sufficiently powered, safety-tested and vetted, and the infection risk is a serious factor to consider and not just a obstacle for bureaucratic purposes.

6. Synthesis: Where Does the Evidence Actually Point?

The following tables summarize the current evidence on gut microbiome alterations in sarcopenia, highlighting key findings, the direction of effects, and the level of confidence across different evidence domains.

Table 1

Summary of Evidence on Gut Microbiome Alterations and Sarcopenia in Older Adults

Domain	Representative evidence	Direction of effect	Confidence
Diversity/composition in sarcopenia	Lapauw et al., 2024 (32 studies); Wang et al., 2025 (meta-analysis)	Lower diversity, altered composition associated with sarcopenia	Consistent association; taxa inconsistent
Butyrate/SCFA producers	Han et al., 2022; Kang et al., 2021	Depleted in sarcopenia/low muscle mass	Moderate - mechanistically coherent
Germ-free/FMT causal models	Fielding et al., 2019; Mo et al., 2023	Donor microbiota transfers a measurable muscle phenotype	Animal evidence only; not directly generalisable
Longitudinal, hard-outcome human data	Geraldi et al., 2026 (SUPERB cohort, n=2,081, replicated)	Microbial diversity/richness predicts mortality, falls, fracture	Strongest human evidence to date; still observational
Probiotic strains (PS23, TWK10)	Lee et al., 2025; Lee et al., 2021	Improved strength/performance, inconsistent effect on mass	Real but strain-specific; short trials
Postbiotic Akkermansia (HB05P)	Kang et al., 2024	Improved leg extensor strength, raised follistatin	Single trial; mechanistically interesting
Urolithin A (purified postbiotic)	Singh et al., 2022; Liu et al., 2022	Improved strength and mitochondrial biomarkers	Among the more rigorous human RCT evidence
Prebiotic + anabolic stack (PROMOTe)	Ni Lochlainn et al., 2021, 2024	Improved muscle/cognitive outcomes vs. placebo	Well-designed twin RCT; single trial
Faecal microbiota transplantation	Mo et al., 2023 (animal); no human sarcopenia RCT	Benefit in aged rodents only	Experimental; not translatable yet

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7. GUT MICROBIOME AS A CAUSE, A CONSEQUENCE OR A MODIFIER OF SARCOPENIA?

This is the one point that the entire review has been swinging around and it does need a straightforward response instead of a fence that's coated in nuance. This is supported by the muscle relevant metabolites, such as butyrate, bile acids, urolithin A, and kynurenine, by faecal transplants and germ-free colonisation experiments (Fielding et al., 2019; Mo et al., 2023), and several targeted interventions – PROMOTe prebiotic trial, Akkermansia postbiotic trial, urolithin A – have been shown to shift muscle outcomes in RCTs (Ni Lochlainn et al., 2024; Kang et al., 2024; Singh et al., 2022). The case for caution is at least as strong: even though most cross-sectional associations cannot be explained by reverse causation, because the taxa reported as 'sarcopenia-associated' vary so much between cohorts (though similar patterns suggest they may indicate a similar underlying process, such as inflammation, which is shared by sarcopenia, diabetes, and frailty), and even within the interventional literature, effect sizes are generally small and lengths of trial are brief.

We think the most defensible interpretation is that the gut microbiome is a genuine modifier and partial mediator within a larger system of diet, physical activity, inflammation and comorbidity – and that it has specific and demonstrated pathways of metabolites to nudge the way of muscle ageing, but not on present evidence can make sarcopenia reversible by microbiome manipulation alone.

8. CONTROVERSIES AND METHODOLOGICAL LIMITATIONS

There are some common issues that constrain the extent to which the current literature can bear this load. Although the studies reviewed here (Cruz-Jentoft et al., 2019; Chen et al., 2020; Dent et al., 2021) may be directly compared, there are some caveats to that. Sarcopenia case definitions have changed over time and vary by region (EWGSOP versus AWGS). Methodology is inconsistent: the majority of published studies still use 16S rRNA amplicon sequencing, which provides much lower taxonomic and functional resolution than the shotgun metagenomics used by only a few studies (Wang et al., 2022; Ticinesi et al., 2020) and even sharing the raw sequencing data can lead to different conclusions in terms of composition, as described in detail in methodologic reviews of microbiome data processing (Zhou et al., 2023).

All these factors independently influence the microbiome and muscle outcomes, and are not measured or adjusted for consistently across studies. Very few of the human studies are longitudinal and even the best interventional studies are of only weeks to a few months in duration, too brief a period to show lasting change in what is a slow process as sarcopenia. Some of the conceptual frameworks this more recent sarcopenia-specific literature builds on are decades old relative to the speed of the field; and foundational descriptive work on how the relative abundance of the major bacterial phyla changes over age (Mariat et al., 2009) and how richness of the microbiome in the gut correlates with metabolic health markers more generally (Le Chatelier et al., 2013) pre-date the newer and higher-resolution sequencing technologies now available.

Research Gaps and What Should Be Measured Next

The below table 2 highlights the major research gaps in understanding the gut–muscle axis in sarcopenia and outlines key priorities for future investigation. It emphasizes the need for longitudinal studies, mechanistic validation, standardized methodologies, and large-scale clinical trials to establish causality and evaluate the long-term effectiveness of microbiome-targeted interventions.

Table 2

Research Gaps and Future Perspectives in the Gut–Muscle Axis in Sarcopenia

Gap	Why it matters	What future studies should measure
Few large longitudinal cohorts with hard outcomes	The SUPERB study (Geraldi et al., 2026) is close to unique in linking microbiome data to mortality/falls/fracture	Repeated microbiome sampling (at least 3 timepoints) alongside DXA, handgrip strength, gait speed, SPPB, and multi-year mortality/falls follow-up
Inconsistent sarcopenia definitions across studies	Findings using EWGSOP vs. AWGS criteria are not directly comparable (Cruz-Jentoft et al., 2019; Chen et al., 2020)	Uniform application of one current consensus definition, with mass/strength/performance sub-components reported separately

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Underuse of shotgun metagenomics and multi-omics	16S data (the majority of studies cited here) cannot resolve strain-level or functional differences	Paired shotgun metagenomics, metabolomics and, where feasible, muscle biopsy transcriptomics in the same participants
Short intervention durations	Even the stronger RCTs (Kang et al., 2024; Singh et al., 2022; Tominaga et al., 2021) ran 8-16 weeks	Trials of 6-12 months with pre-registered muscle-mass and strength endpoints, not just biomarkers
Underrepresented populations	Existing cohorts cluster in East Asia and Europe (Wang et al., 2022; Ghosh et al., 2020), with newer additions from the US (Li et al., 2025) and Uyghur China (Zhang et al., 2024) still rare	Deliberate recruitment across South Asian, African, Latin American and oldest-old (80+) populations using harmonised protocols
Sex as an under-examined effect modifier	Associations differ meaningfully by sex in at least one large cohort (Asaoka et al., 2025)	Pre-specified sex-stratified analysis as standard practice, not an afterthought
Confounding by diet, medication and comorbidity	Antibiotics, proton-pump inhibitors, metformin and diet independently reshape both microbiome and muscle	Systematic capture and adjustment for medication use and validated dietary intake instruments in every study

10. CONCLUSION

In the last few years, the gut-muscle axis has shifted from an interesting hypothesis to an area with a lot of evidence, but not enough to move on with confidence. Animal studies demonstrate that the link can go in either direction: from gut to muscle, as older animals with sarcopenia become stronger and more active when given specific probiotic strains, a postbiotic based on Akkermansia muciniphila, a prebiotic that's added to existing anabolic treatment, and purified urolithin A. And there are a growing number of carefully designed trials that show that older animals with sarcopenia can be strengthened, in a measurable way, with these treatments. What the field is missing at this moment is a large, long, well powered trial that advances hard sarcopenia outcomes (falls, fracture, hospitalisation, independence) as opposed to surrogate strength outcomes over a few weeks. While waiting for that trial to arrive, modulating the gut-muscle axis, with diet, exercise, and selected probiotic or postbiotics compounds, is reasonable, low risk, and is not a replacement, but rather an addition, to existing care for sarcopenia; extending it beyond that is taking a step that the data, read carefully across over eighty studies, do not support.

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