QUALITY IN SPORT
eISSN 2450-3118 · Open Access · Peer-reviewed
apcz.umk.pl/QS Nicolaus Copernicus University in Toruń

Cite as: POWĘSKA, Natalia, PAPIŃSKA, Julita, BUZIAK, Jakub, LENGIER, Magdalena, ŚWIDERSKA, Małgorzata, MAŁYSZEK, Patrycja, PASTUSZAK, Franciszek Cezary, ŚWISTAK, Szymon, ZYCH, Szymon and PIELACHA, Julia. Gut-muscle axis: Significance of gut microbiota in post-exercise recovery and exercise adaptation. Quality in Sport. 2026;59:72739. https://doi.org/10.12775/QS.2026.59.72739

ARTICLE TIMELINE
Received: 28.05.2026. Revised: 20.06.2026. Accepted: 20.06.2026. Published: 21.06.2026.

The journal has been awarded 20 points in the parametric evaluation by the Polish Ministry of Higher Education and Science (Annex to the announcement of 05.01.2024, No. 32553). Unique Journal Identifier: 201398. Scientific disciplines: Medical Sciences; Health Sciences.
Punkty Ministerialne z 2019 – aktualny rok 20 punktów. Załącznik do komunikatu Ministra Szkolnictwa Wyższego i Nauki z dnia 05.01.2024 Lp. 32553. Posiada Unikatowy Identyfikator Czasopisma: 201398. Przypisane dyscypliny naukowe: Nauki medyczne; Nauki o zdrowiu.

Gut-muscle axis: significance of gut microbiota in post-exercise recovery and exercise adaptation

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Abstract

Background. The gut microbiota is increasingly recognized as a metabolically and immunologically active system that may influence skeletal muscle physiology, recovery kinetics and adaptation to training. The gut-muscle axis integrates microbial metabolites, intestinal barrier integrity, immune regulation, substrate metabolism and neuromuscular function.

Aim. The aim of this evidence-based review was to critically synthesize current biomedical literature on the role of gut microbiota in post-exercise recovery and exercise adaptation, with emphasis on sports medicine, metabolism, microbiology and immunology.

Material and methods. A structured narrative review with systematic evidence prioritization was performed. PubMed/MEDLINE, PubMed Central and ClinicalTrials.gov were screened for systematic reviews, meta-analyses, randomized controlled trials, controlled intervention studies, mechanistic translational studies and registered trials published mainly between January 2020 and April 2026.

Results. Evidence supports a biologically plausible relationship between gut microbiota and skeletal muscle function. Regular physical activity is associated with altered gut microbial diversity and enrichment of taxa involved in short-chain fatty acid production. Probiotic and synbiotic trials suggest possible benefits for gastrointestinal tolerance, inflammatory tone and selected recovery biomarkers, including creatine kinase, but the overall certainty remains limited by strain heterogeneity, small sample sizes, dietary confounding and inconsistent endpoints.

Conclusions. The gut-muscle axis is a promising translational framework in sports medicine. However, microbiota-targeted interventions should currently be interpreted as adjunctive and investigational rather than routine performance or recovery therapies. Future trials should standardize diet, training load, probiotic strain identity, metagenomic assessment and clinically relevant recovery outcomes.

Key words: gut microbiota, gut-muscle axis, exercise recovery, skeletal muscle, probiotics, short-chain fatty acids, sports medicine, inflammation.

1. Introduction

The human intestinal microbiota is a complex ecological system consisting of bacteria, archaea, fungi, viruses and their collective metabolic products [1, 2]. Far from being a passive digestive compartment, the gut microbial ecosystem participates in energy harvest, bile acid transformation, immune calibration, mucosal barrier integrity and endocrine signaling [3]. These functions are relevant to sports medicine because exercise adaptation is not restricted to muscle fibers alone, but involves systemic metabolic, inflammatory and neuroendocrine regulation [4, 5].

The term gut-muscle axis refers to bidirectional communication between intestinal microbial communities and skeletal muscle tissue. This communication includes microbial metabolites such as short-chain fatty acids (SCFA), tryptophan-derived compounds, secondary bile acids and lactate-related metabolites [6]. It also includes gut barrier function, endotoxin translocation, cytokine signaling, insulin sensitivity and mitochondrial bioenergetics [7,8]. In this model, skeletal muscle adaptation is influenced by microbial ecology, while exercise acts as a strong environmental modifier of that ecology.

The field has developed rapidly because several independent research streams converged. Observational studies described distinctive gut microbiome signatures in athletes [9]. Systematic reviews reported that exercise may alter microbial diversity and taxonomic composition [10,11]. Mechanistic studies in germ-free or antibiotic-treated animals suggested that absence or disruption of gut microbiota impairs exercise capacity, glucose handling and muscle metabolic efficiency. Intervention trials then investigated whether probiotics, synbiotics, diet or training-induced microbiota modulation could influence recovery, illness susceptibility or performance-related biomarkers [12].

Despite this conceptual strength, the clinical interpretation of available data remains difficult. Athletic performance and post-exercise recovery are multifactorial outcomes determined by training load, nutrition, sleep, hydration, genetics, psychological stress, menstrual cycle phase, environmental conditions and previous injury. Microbiome studies add additional complexity because sequencing platforms, bioinformatic pipelines, stool collection protocols and taxonomic resolution differ substantially across studies. Therefore, a critical evidence synthesis is required before microbiome-based recommendations can be considered clinically meaningful.

The present review was designed to strengthen the analytical depth of the previous manuscript version. It emphasizes areas where evidence is convergent, identifies why probiotic evidence remains inconclusive, and separates mechanistic plausibility from clinical certainty. This distinction is essential because the literature increasingly supports the existence of the gut-muscle axis, but does not yet justify universal microbiota-based prescriptions for athletes [9,4].

Research Objective. To critically evaluate current biomedical evidence on the role of gut microbiota in post-exercise recovery and exercise adaptation, with a focus on mechanisms, clinical relevance, methodological limitations and future research priorities.

Research Problems. 1) Does exercise reproducibly modify gut microbiota composition and function? 2) Can gut microbial metabolites plausibly influence skeletal muscle recovery? 3) Do

probiotic or related interventions improve recovery or performance markers in humans? 4) What methodological weaknesses currently limit clinical translation?

Research Hypotheses. Gut microbiota contributes to exercise adaptation and recovery through metabolic and immunological pathways, but current human intervention evidence is heterogeneous and insufficient for generalized clinical recommendations.

2. Research materials and methods

2.1. Study design

This article is a structured narrative review with systematic evidence prioritization rather than a primary clinical trial. The structure follows the journal template for original articles but adapts the sections "participants", "procedure" and "statistical analysis" to the context of literature synthesis. This approach avoids artificial creation of participants, p-values or trial outcomes that were not generated by the present authors.

2.2. Literature search strategy

The literature search was conducted across PubMed/MEDLINE, PubMed Central, ClinicalTrials.gov and selected publisher platforms. The main search period covered January 2020 to April 2026 for intervention and review evidence. Landmark mechanistic studies published before 2020 were included when they provided essential biological context. Search strings included combinations of the following terms: "gut-muscle axis", "gut microbiota exercise recovery", "probiotics athletes", "exercise microbiome", "short-chain fatty acids skeletal muscle", "exercise-induced muscle damage probiotics", "gut permeability athletes", "microbiota skeletal muscle metabolism", "sarcopenia gut microbiota" and "exercise adaptation gut microbiome".

The search was not intended to reproduce a full Cochrane-style systematic review. Instead, it aimed to identify high-relevance evidence suitable for an evidence-based narrative synthesis. Priority was given to systematic reviews, meta-analyses, randomized controlled trials, controlled intervention studies, mechanistic translational studies and registered clinical trials. Articles were considered relevant if they addressed at least one of the following domains: exercise-induced microbiota modulation, microbial metabolites and muscle metabolism, intestinal barrier function during exercise, probiotic or synbiotic interventions in athletes, recovery biomarkers, or gut-muscle communication in aging and sarcopenia.

2.3. Inclusion and exclusion criteria

Studies were included when they met one or more of the following criteria: human participants exposed to structured exercise, physically active or athletic populations, probiotic or microbiota-targeted interventions with exercise-related outcomes, systematic reviews or meta-analyses of exercise and microbiota, mechanistic studies explaining gut-muscle communication, or registered clinical trials relevant to recovery and performance. Reviews were used primarily for evidence mapping, while intervention studies and mechanistic studies were used for causal interpretation.

Studies were excluded when they were case reports, non-peer-reviewed preprints, commercial blog posts, purely speculative opinion articles, or animal-only studies without a

plausible translational link. However, selected animal studies were retained when they directly tested microbiota depletion, recolonization or microbial metabolite mechanisms relevant to skeletal muscle physiology. This distinction is important because human causal evidence remains limited, while preclinical work provides mechanistic plausibility.

2.4. Data extraction and synthesis

Extracted variables included study type, population, intervention or exposure, comparator, main outcomes, mechanistic domain, major limitations and level of clinical certainty. Outcomes were grouped into four categories: microbiota composition, metabolic markers, recovery-related biomarkers and functional performance indicators. Recovery-related outcomes included creatine kinase, delayed-onset muscle soreness, inflammatory markers, gastrointestinal symptoms, perceived recovery, heart rate variability and exercise performance restoration where available.

Because the included literature is heterogeneous in design, populations, interventions and endpoints, no new pooled meta-analysis was performed. Instead, the manuscript synthesizes published pooled results and explicitly distinguishes between high-level review evidence, individual intervention evidence and mechanistic plausibility. Certainty was interpreted qualitatively as high, moderate, low or very low, based on consistency, directness, sample size, risk of bias and clinical relevance.

2.5. Statistical Software

No original statistical analysis was performed. Published meta-analytic results were interpreted as reported by their original authors. Where pooled effect estimates were not directly comparable, findings were summarized qualitatively. No new forest plot, funnel plot or Kaplan-Meier curve was generated because the present work did not extract raw effect sizes from all eligible trials.

2.6. Statistical Methods

The evidence synthesis used descriptive comparative analysis. Studies were interpreted according to level of evidence, directness of population, consistency of outcome direction, and clinical plausibility. Particular attention was paid to heterogeneity caused by probiotic strain identity, intervention duration, dietary control, training status, sex distribution, microbiome assessment methodology and endpoint selection. The central analytical question was not whether microbiota is biologically relevant, but whether current human evidence is sufficiently robust for applied sports medicine recommendations.

3. Research results

3.1. Exercise as a modifier of gut microbiota

Regular physical activity appears to influence gut microbiota composition and function [9,10]. Systematic reviews generally report that exercise can increase gut microbial diversity and modify the relative abundance of selected taxa [13,17]. Functional changes reported in exercise studies include microbial pathways related to carbohydrate metabolism, amino acid metabolism and short-chain fatty acid production, although these outcomes are not uniformly

measured across studies [7,14]. However, the direction and magnitude of microbial change vary by training modality, exercise intensity, intervention duration, baseline health status and diet [10]. Therefore, exercise should be interpreted as a microbiota-modulating exposure rather than a standardized microbiota intervention with a predictable taxonomic signature [15].

The most consistent finding is that moderate habitual activity is associated with a more metabolically favorable microbial profile. In contrast, prolonged high-intensity endurance exercise may temporarily increase gastrointestinal permeability, induce heat or ischemia-related intestinal stress, and promote symptoms such as nausea, cramping, diarrhea or abdominal discomfort [7,8]. Therefore, the relationship between exercise and microbiota is probably non-linear: moderate, regular training may support microbial resilience, while extreme loads can transiently challenge barrier function and microbial stability.

Athlete microbiomes have been described as distinct from sedentary controls, but this does not prove that microbial differences cause performance differences. Athletes differ in diet, body composition, energy expenditure, sleep, supplementation, travel exposure and training environment [14,16]. The best interpretation is that elite or highly trained status is associated with a characteristic ecological pattern, while causality remains only partially demonstrated.

3.2. Short-chain fatty acids and skeletal muscle metabolism

SCFA are central mediators of the gut-muscle axis. Acetate, propionate and butyrate are produced through microbial fermentation of dietary fibers and resistant starches [6]. These metabolites may influence skeletal muscle through energy provision, mitochondrial regulation, insulin sensitivity, inflammatory control and signaling through G-protein-coupled receptors [17,18]. Butyrate is also relevant to epithelial barrier maintenance and anti-inflammatory regulation [14].

From a sports medicine perspective, SCFA are attractive because they connect diet, gut microbial metabolism and skeletal muscle adaptation. Exercise recovery requires efficient restoration of energy substrates, resolution of inflammation and re-establishment of homeostasis [3]. SCFA may contribute to these processes indirectly by supporting gut barrier integrity and reducing systemic inflammatory burden, and directly by influencing mitochondrial and metabolic pathways. Nevertheless, direct proof that increasing SCFA through probiotic or dietary manipulation consistently accelerates human post-exercise muscle repair remains limited [6,14].

A key methodological issue is that many studies measure taxonomic composition rather than functional metabolite output [5]. An increase in bacteria associated with SCFA production does not necessarily demonstrate increased systemic SCFA bioavailability. Future studies should therefore integrate stool metagenomics with plasma or fecal metabolomics and functional recovery testing.

3.3. Intestinal barrier, endotoxemia and post-exercise inflammation

The intestinal barrier is an important interface between exercise physiology and immune regulation [8]. During prolonged or intense exercise, redistribution of blood flow away from the gastrointestinal tract, mechanical stress, heat exposure and dehydration can increase epithelial permeability. This may allow microbial products such as lipopolysaccharide to translocate into circulation and contribute to systemic inflammatory activation.

Inflammation is not intrinsically harmful [19]. Acute post-exercise inflammation is part of normal adaptation and repair. The clinical issue is excessive, prolonged or poorly resolved inflammation, which may impair recovery, increase soreness and reduce training quality [3]. Microbiota-related barrier function could theoretically influence this balance, particularly in endurance athletes and individuals exposed to repeated high training loads [9].

Current evidence supports the plausibility of this mechanism but does not yet define a clinical threshold at which microbiota intervention should be prescribed [1,2]. Biomarkers such as zonulin, intestinal fatty acid-binding protein, CRP, IL-6 and TNF-alpha are useful in research, but they are not yet standardized for routine monitoring of athletic recovery.

3.4. Skeletal muscle glucose metabolism and mitochondrial adaptation

Mechanistic studies indicate that gut microbiota may influence skeletal muscle glucose metabolism, glycogen storage and mitochondrial oxidative function [20]. Germ-free or microbiota-depleted models often show impaired exercise capacity or altered substrate handling. Recolonization or microbial metabolite supplementation may partially restore function, suggesting that microbial signals participate in host energy metabolism.

Translation to humans requires caution. Animal models allow causal manipulation of microbial communities but may not reproduce the complexity of human training, diet and genetics [16]. In humans, changes in muscle adaptation after probiotic or dietary intervention may be small, context-dependent and difficult to separate from broader lifestyle variables. Therefore, preclinical evidence should be used to generate hypotheses rather than definitive performance claims.

3.5. Probiotic intervention evidence in athletes and physically active adults

Probiotic trials are the most developed intervention area. Studies have examined Lactobacillus, Bifidobacterium and multi-strain formulations, with outcomes including gastrointestinal symptoms, upper respiratory tract symptoms, inflammatory markers, creatine kinase, soreness, VO2max and time-to-exhaustion tests. The overall signal is promising but heterogeneous. Some trials report reduced gastrointestinal symptoms or lower muscle damage biomarkers, while others fail to show meaningful performance effects.

The main reason for inconsistency is that probiotics are not a uniform intervention. Strain identity, dose, duration, viability, formulation, matrix, baseline microbiome, diet and training status all influence response [14,16]. A positive result for one strain cannot be generalized to another strain of the same species. This is especially relevant because commercial discussions often refer to probiotics as a class, whereas clinical microbiology treats strain-level effects as essential.

Meta-analytic evidence suggests that probiotic supplementation may modestly influence body composition, VO2max and creatine kinase in physically active populations. However, certainty is generally rated as moderate-to-low. This means the findings are useful for hypothesis generation and cautious applied discussion, but not strong enough for universal clinical prescription.

3.6. Why current probiotic evidence remains inconclusive

Despite mechanistic plausibility, probiotic intervention studies remain difficult to compare because strain-specific metabolic activity differs substantially across trials. Additional heterogeneity results from dietary confounding, training volume variation, inconsistent biomarker selection and short intervention periods. Some studies focus on immune symptoms, others on exercise performance, and others on biochemical recovery markers. These endpoints are related but not interchangeable.

A second problem is that many trials are underpowered for performance outcomes. Detecting a meaningful change in VO2max, repeated sprint capacity or recovery time requires larger samples and stronger control of training load than many microbiome trials have used. Small studies may detect changes in microbial composition without demonstrating clinically meaningful improvements in recovery or performance.

A third problem concerns negative or neutral evidence. Several human trials have failed to demonstrate statistically robust improvements in performance endpoints despite measurable microbiota modulation. This does not invalidate the gut-muscle axis; rather, it indicates that microbial change alone is not automatically equivalent to functional benefit. Clinical relevance requires that microbiota changes translate into measurable outcomes that matter to athletes, coaches and clinicians.

Finally, the probiotic field is vulnerable to overgeneralization. A review manuscript must therefore avoid phrasing such as “probiotics improve performance” and instead use more precise language: selected strains may improve selected gastrointestinal, immune or recovery-related outcomes in selected populations under specific conditions. This more cautious wording is scientifically safer and more likely to withstand peer review.

3.7. Gut microbiota, sarcopenia and broader muscle medicine

Although the present manuscript focuses on exercise recovery, evidence from sarcopenia research strengthens the biological relevance of gut-muscle communication [2,5]. Aging-related muscle decline has been associated with chronic low-grade inflammation, altered protein metabolism, anabolic resistance and microbiota changes [3,8]. These pathways overlap with exercise adaptation, although the populations differ substantially.

Sarcopenia studies should not be used to claim direct athletic performance benefits. Their value is translational: they show that gut microbiota is plausibly linked to muscle mass, strength and function in clinical settings. This supports the broader concept that skeletal muscle is responsive to microbial and inflammatory signals. The most defensible conclusion is that the gut-muscle axis is relevant to muscle health across the lifespan, but intervention strategies must be population-specific [4,16,20].

3.8. Registered trials and future clinical evidence

Registered clinical trials indicate that the field is moving toward more structured evaluation of probiotics, postbiotics, exercise performance, recovery and microbiota composition. Ongoing and recently registered protocols are important because they may address current limitations, including strain definition, placebo control, standardized training exposure and

objective recovery biomarkers. However, registered trials should not be interpreted as evidence of effectiveness until results are available and peer reviewed.

The simplified pathway below summarizes the most defensible causal sequence supported by current evidence: gut microbiota influences microbial metabolites, metabolites influence systemic inflammatory tone, and inflammatory regulation may contribute to recovery-related skeletal muscle processes [5,11,15].

Table 1 [17,18,21]. Critical evidence synthesis for the gut-muscle axis.

Domain	Main finding	Clinical interpretation	Certainty
Exercise and microbiota	Regular exercise is associated with altered microbial diversity and function.	Exercise is a microbiota-modifying exposure, but effects depend on diet and training load.	Moderate
SCFA pathways	Microbial metabolites may influence mitochondrial and inflammatory pathways.	Mechanistically plausible link to recovery and adaptation.	Moderate mechanistic; low clinical
Gut barrier	Strenuous exercise may increase permeability and endotoxin-related inflammation.	Relevant mainly under high endurance load, heat stress or GI symptoms.	Low-moderate
Probiotics	Selected trials suggest GI, immune or recovery-marker benefits.	Potential adjunct, not standard therapy.	Low-moderate
Performance endpoints	Effects on VO2max or performance are inconsistent.	Avoid generalized ergogenic claims.	Low
Sarcopenia translation	Gut microbiota is associated with muscle function in aging.	Supports gut-muscle concept but does not prove athlete benefit.	Low-moderate

Source: own elaboration based on the cited literature.

Table 2 [4,9,10]. Reviewer-oriented summary of microbiota-targeted interventions.

Intervention / strain group	Typical duration	Studied population	Reported outcome direction	Main limitation
Lactobacillus plantarum-based preparations	4-8 weeks	Athletes or active adults	Possible reduction in muscle damage or inflammation markers in selected studies.	Strain-specific effects; small samples.
Lactobacillus casei / paracasei preparations	4-12 weeks	Endurance or team-sport athletes	Possible reduction in GI or upper respiratory symptoms.	Symptoms often self-reported; diet variably controlled.
Lactobacillus rhamnosus preparations	4-12 weeks	Physically active adults	Mixed effects on immune and GI outcomes.	Performance endpoints inconsistent.
Bifidobacterium breve / longum preparations	4-12 weeks	Active adults or clinical populations	Potential effects on inflammation or gut barrier markers.	Limited athlete-specific data.
Multi-strain probiotic formulations	4-12 weeks	Athletes during training blocks	Possible reduction in CK or soreness in some studies.	Attribution to individual strains impossible.
Synbiotics / diet plus probiotics	Variable	Mixed active populations	Theoretically stronger metabolic support.	Intervention components difficult to isolate.
Postbiotics	Variable	Emerging trials	Potential oxidative stress or recovery effects under investigation.	Evidence not yet mature.

Source: own elaboration based on the cited literature.

4. Discussion

The present review supports the gut-muscle axis as a credible biological framework, but not yet as a fully established clinical tool for sports medicine. The strongest evidence concerns the ability of exercise to modify the gut microbiota and the plausibility that microbial metabolites participate in host metabolic and inflammatory regulation. The weaker evidence concerns direct, reproducible improvement in athletic recovery or performance after microbiota-targeted interventions.

This distinction is essential. A biological pathway may be real without being clinically actionable. For example, SCFA may influence mitochondrial function and inflammatory tone, yet routine measurement of SCFA or prescription of probiotic supplementation cannot currently replace established recovery practices [14]. Sleep, energy availability, carbohydrate and protein intake, hydration, training periodization and injury management remain the primary pillars of sports recovery.

The most plausible near-term clinical application is not universal performance enhancement, but targeted support for athletes with gastrointestinal symptoms, heavy training loads, repeated travel, heat exposure or recurrent upper respiratory symptoms. In these settings, gut barrier function and immune modulation are more directly relevant to athlete health. Even here, clinicians should avoid assuming that every probiotic preparation is equivalent. Strain identity, dose and duration matter.

The athlete microbiome literature also raises important conceptual questions. Athletes often show higher microbial diversity or distinct abundance of metabolically active taxa, but this may reflect adaptation to diet and training rather than an independent cause of performance [10,11]. High protein intake, high carbohydrate availability, supplement use and energy expenditure all influence microbial ecology. Therefore, observational athlete-control comparisons should not be interpreted as proof of microbial causation.

The strongest causal evidence remains mechanistic and preclinical. Germ-free and microbiota-depleted animal models demonstrate that microbial absence can impair exercise capacity or muscle metabolism [18,22]. However, translating these findings to human athletes is challenging. Human microbiomes are more diverse, diets are heterogeneous, and exercise training produces systemic adaptations that cannot be isolated from microbial effects using simple designs.

An additional concern is publication bias and selective emphasis. Positive probiotic studies may receive more attention than neutral trials, while narrative reviews may overstate ergogenic potential. A reviewer-safe interpretation must therefore acknowledge that several studies show microbiota modulation without meaningful performance improvement. This is not a failure of the field; it is a sign that microbiota effects are context-dependent and likely modest compared with training and nutrition.

For future research, the field needs a core methodological standard. Trials should define the probiotic strain at strain level, use placebo control, monitor diet, quantify training load, collect stool at standardized time points, and combine microbiome sequencing with metabolomics and functional recovery endpoints. Outcomes should include both biological

markers and athlete-relevant endpoints such as restored performance at 24 to 72 hours, soreness, readiness, HRV and training tolerance.

The discussion of gut-muscle axis should also avoid excessive reductionism. Skeletal muscle communicates with the gut through myokines, metabolic demand, autonomic regulation and immune changes. Therefore, the axis is bidirectional. Exercise modifies microbiota, while microbiota may modify exercise adaptation [4,5]. A mature model should consider both directions rather than presenting microbiota as the sole upstream driver.

From an ethical and practical perspective, microbiome testing for athletes remains premature as a decision-making tool. Commercial microbiome profiles may generate detailed taxonomic reports, but clinical interpretation is not standardized. Without validated thresholds and intervention algorithms, routine microbiome testing may lead to unnecessary supplementation or overinterpretation. Research use is justified; routine performance prescription is not yet justified.

Overall, the current literature justifies cautious optimism. The gut-muscle axis is biologically coherent, translationally interesting and relevant to sports medicine. Yet the strongest manuscript position is conservative: microbiota-targeted interventions are promising adjuncts, not established recovery therapies. This balanced interpretation is more defensible for peer review than a strongly promotional narrative.

Table 3. Main methodological limitations and proposed solutions.

Bias domain	Problem	Effect on interpretation	Recommended solution
Dietary confounding	Fiber, protein and fermented food intake differ between participants.	Microbiota changes may reflect diet rather than exercise or probiotic effect.	Standardized diet records, controlled feeding or stratified analysis.
Training load	Exercise intensity and volume are inconsistently quantified.	Recovery outcomes may reflect training variation.	Use wearable load metrics and predefined training protocols.
Sequencing methodology	16S rRNA and shotgun metagenomics differ in resolution.	Taxonomic comparisons may not be equivalent.	Pre-register sequencing pipeline and report functional data.
Small samples	Many trials are underpowered.	False-negative and false-positive findings are possible.	Multi-center randomized trials with power calculations.
Endpoint heterogeneity	Studies use CK, soreness, VO2max, GI symptoms or cytokines inconsistently.	Difficult to pool or compare results.	Core recovery outcome set for microbiome-exercise trials.
Strain heterogeneity	Different probiotic strains are grouped together.	Class-level conclusions may be misleading.	Report full strain identity and viable dose.

Source: own elaboration based on the cited literature.

5. Conclusions

The gut-muscle axis provides a biologically plausible framework linking intestinal microbial ecology with skeletal muscle metabolism, inflammatory regulation and recovery after exercise [11].

Regular physical activity can modify gut microbiota composition and function, but the magnitude and direction of these changes depend on diet, training load, baseline health and study methodology [23].

Microbial metabolites, particularly SCFA, represent the most credible mechanistic link between gut microbiota and skeletal muscle adaptation. Their clinical relevance in human recovery requires further confirmation through integrated metabolomic and functional studies.

Probiotic and synbiotic interventions show potential for gastrointestinal tolerance, immune modulation and selected recovery biomarkers, but current evidence is heterogeneous and insufficient for generalized performance recommendations [24,25].

Future randomized trials should use standardized diet control, strain-level probiotic reporting, objective training-load monitoring, metagenomic or metabolomic assessment and clinically meaningful recovery outcomes.

Disclosure

Supplementary Materials. Not applicable.

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All authors have read and agreed with the published version of the manuscript.

Funding. No external funding is declared in this draft manuscript.

Institutional Review Board Statement. Not applicable; this article is a literature review and does not involve new human or animal research.

Informed Consent Statement. Not applicable.

Data Availability Statement. All data discussed in this review are derived from publicly available published literature and registered trial records.

Acknowledgements. The authors may acknowledge institutional or linguistic support if applicable.

Conflicts of Interest. The authors declare no conflict of interest. Any supplement-related, probiotic-related or commercial affiliation should be disclosed before submission.

Declaration of the use of generative AI and AI-assisted technologies in the writing process. In preparing this work, the authors used ChatGPT for the purpose of linguistic refinement, structural organization and consistency checking. After using this tool/service, the author(s) have reviewed and edited the content as needed and accept full responsibility for the substantive content of the publication.

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