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Gut Microbiome and Training Adaptation: A Narrative Review of Evidence from 2018–2026

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Abstract

Background: The gut microbiome has emerged as a potential modifier of exercise physiology through effects on nutrient metabolism, intestinal barrier integrity, immune regulation, microbial metabolite production, and gut–brain communication. However, microbial changes reported after training are heterogeneous, and their contribution to persistent human training adaptation remains uncertain.

Aim: This narrative review examines evidence from 2018 to 23 July 2026 on how endurance, resistance, and combined exercise affect the gut microbiome and whether microbiome-related pathways influence performance, recovery, adherence, and long-term adaptation.

Material and Methods: A targeted search of PubMed/MEDLINE, official publisher platforms, and reference lists was performed for peer-reviewed human interventions, athlete studies, systematic reviews, meta-analyses, and mechanistic animal experiments. Evidence was synthesized qualitatively because of substantial heterogeneity in populations, exercise prescriptions, dietary control, sequencing methods, metabolite assays, and performance outcomes.

Results: Human endurance and combined training produce modest, variable, and often reversible microbiome changes influenced by factors such as diet, age, adiposity, exercise dose, medications, and baseline microbial composition. Resistance training improves neuromuscular function but usually causes limited group-level microbiome changes. Athlete microbiomes differ from those of sedentary individuals, although dietary and lifestyle confounding remains substantial. Animal studies suggest mechanisms involving microbial propionate, dopaminergic

signaling, and bile-acid pathways, but direct human evidence that microbiome modification improves training adaptation remains insufficient.

Conclusion: Current evidence supports a bidirectional exercise–microbiome relationship but does not establish a universal athletic microbiome or justify routine microbiome-based training prescriptions.

Keywords: gut microbiome; gut microbiota; exercise; training adaptation; athletes; short-chain fatty acids; gut–muscle axis; endurance; resistance training

1. Introduction

Exercise training produces coordinated adaptations across skeletal muscle, the cardiovascular system, immune regulation, substrate metabolism, and the central nervous system. During the last decade, the gut microbiome has been added to this network as a potentially important, but still incompletely characterized, component of the exercise response. Reviews published since 2018 have concluded that physical activity can alter microbial composition and function, while microbial metabolites may, in turn, influence host metabolism, inflammation, intestinal integrity, and exercise tolerance [1,2]. More recent meta-analyses suggest a small overall increase in microbial alpha diversity after exercise interventions, although the magnitude is modest and heterogeneity between studies remains substantial [3]. A 2026 synthesis further emphasized that human responses are usually individualized, reversible after detraining, and smaller than the effects often implied in non-academic discussions [4].

The term training adaptation refers here to the persistent physiological and functional changes that occur after repeated exercise exposure, including improvements in maximal oxygen uptake, endurance capacity, strength, muscle mass, metabolic control, recovery, and tolerance of subsequent exercise. A microbiome change should not automatically be interpreted as an adaptation. To contribute meaningfully to training adaptation, a microbial shift would ideally precede or accompany a change in host function, display a plausible dose–response relationship, and be supported by mechanistic or mediation evidence.

Interpretation is further complicated by methodological variability. Studies differ in stool collection, storage, DNA extraction, 16S ribosomal RNA regions, sequencing platforms, bioinformatic pipelines, taxonomic databases, normalization strategies, and metabolite assays.

Recent best-practice recommendations for sport microbiome research stress standardized procedures, repeated sampling, detailed dietary assessment, medication reporting, and functional analyses rather than reliance on single taxonomic ratios [5]. Accordingly, the present review distinguishes evidence from human interventions, cross-sectional athlete studies, and mechanistic animal experiments, and avoids treating the *Firmicutes-to-Bacteroidetes* ratio or the abundance of a single genus as a universal marker of gut health.

The aim of this narrative review is to evaluate evidence from 2018–2026 concerning: whether structured exercise modifies the gut microbiome; whether endurance, resistance, and combined training produce different microbial responses; whether athlete-associated microbial features are related to performance; and which microbial pathways could plausibly influence training adaptation.

2. Materials and Methods

2.1. Review design and research questions

This manuscript was designed as a narrative review. The review focused on peer-reviewed evidence addressing the bidirectional relationship between exercise and the gut microbiome. The principal questions were whether regular exercise modifies microbial diversity, composition, or function; whether microbiome characteristics are associated with endurance, strength, or recovery outcomes; and which microbial metabolites or signaling pathways may mediate these relationships.

The primary publication window was 1 January 2018 to 23 July 2026. This interval was selected to capture the rapid expansion of human exercise-intervention studies, athlete metagenomics and mechanistic gut–muscle and gut–brain research.

2.2. Search strategy

A targeted literature search was conducted in PubMed/MEDLINE and supplemented by searches of publisher platforms and the reference lists of relevant reviews and original studies. The final search was completed on 23 July 2026. Search concepts included combinations of “gut microbiome”, “gut microbiota”, “intestinal microbiota”, “exercise”, “physical activity”, “endurance training”, “resistance training”, “athlete”, “sports performance”, “training adaptation”, “cardiorespiratory fitness”, “short-chain fatty acids”, “gut–muscle axis”, “gut–brain axis”, “intestinal permeability” and “recovery”. Additional searches used the names of repeatedly investigated taxa and pathways, including *Akkermansia muciniphila*, *Veillonella*,

Bifidobacterium, *Faecalibacterium*, butyrate, propionate, lactate metabolism, bile acids, and dopamine signaling.

Priority was given to original human interventions, randomized or controlled trials, longitudinal athlete studies, shotgun metagenomic or metabolomic studies, and mechanistic experiments. Systematic reviews and meta-analyses were used to assess consistency across studies and identify methodological limitations.

2.3. Eligibility and evidence synthesis

Publications were considered relevant when they directly assessed exercise or habitual physical activity in relation to gut microbial composition, microbial functional capacity, microbial metabolites, intestinal barrier markers, inflammation, exercise performance, or physiological adaptation. Because the included studies were heterogeneous in terms of populations, exercise protocols, microbiome assessment methods, and reported outcomes, a quantitative meta-analysis was not performed. Findings were synthesized qualitatively and organized into thematic sections covering athlete-associated microbiome characteristics, endurance training, resistance training, combined exercise, microbial metabolites, intestinal-barrier and immune mechanisms, the gut–muscle axis, the gut–brain axis, and implications for performance and recovery. Greater interpretative weight was assigned to controlled human interventions than to cross-sectional or preclinical studies. Causal language was avoided when evidence was observational, and findings from animal models were treated as mechanistic rather than directly applicable to athletes. Non-peer-reviewed reports, conference abstracts without sufficient data, commercial webpages, and articles without direct microbiome assessment were excluded.

Data considered during synthesis included population characteristics, sample size, exercise type and dose, dietary control, microbiome method, metabolite measurement, performance outcomes, and major limitations. Owing to heterogeneity in populations, protocols, sequencing approaches, and outcomes, no new quantitative pooling was performed. Evidence was organized thematically and interpreted according to study design, consistency, biological plausibility and the distinction between association and causation.

3. Exercise Training and Gut Microbial Ecology

3.1. Endurance and aerobic exercise interventions

Several human interventions indicate that endurance exercise can modify gut microbial composition and metabolic output, but the response is not uniform. In a six-week supervised

endurance program, Allen et al. observed obesity-dependent changes in microbial community structure. Fecal short-chain fatty acids increased in lean participants but not in participants with obesity, and many changes were reversed after a sedentary washout [6]. This finding is important because it demonstrates that exercise-related microbial responses can depend on host metabolic status and may require continued training.

In previously sedentary women with overweight, six weeks of cycling improved maximal power and oxygen uptake and produced modest taxonomic and metagenomic changes without broad systemic metabolomic changes [7]. The study suggested that *Akkermansia* and some *Proteobacteria* may be exercise-responsive, but the small sample and heterogeneous individual trajectories limited strong taxon-specific conclusions. A short intervention in older men similarly produced selective changes rather than a uniform restructuring of the community [8].

The strongest early randomized evidence came from a six-month trial in adults with overweight or obesity. Structured exercise changed beta diversity, and vigorous exercise increased the Shannon diversity index by approximately 5% at three months, but no genus changed significantly after correction for multiple testing [9]. This combination of a detectable community-level effect and absent genus-level consensus illustrates why broad claims about specific “exercise bacteria” should be treated cautiously.

Age may modify responsiveness. An eight-week program in physically inactive older women changed selected taxa, including groups related to *Actinobacteria* and *Bifidobacterium*, without a clear increase in overall alpha diversity [10]. A 24-week combined cardiovascular and resistance intervention in older sedentary adults increased *Bifidobacterium*, *Oscillospira*, and *Anaerostipes* and increased fecal butyrate, although only 15 participants provided complete samples [11]. These results support the possibility that longer programs can affect microbial metabolic output even when global diversity changes are small.

Exercise intensity may also matter. In a small randomized substudy of people with type 2 diabetes, moderate-intensity continuous combined training and high-intensity interval combined training produced different compositional and functional responses [12]. However, the sample was too small to establish a general intensity threshold. Across human research, exercise dose is intertwined with baseline fitness, adherence, diet, medication, and energy balance, making simple moderate-versus-vigorous conclusions premature.

3.2. Temporal dynamics, acute exercise and detraining

The timing of stool collection is a central interpretative issue. A sample obtained shortly after the final training session may capture an acute response to altered intestinal transit, exercise-induced lactate production, heat stress, hydration changes, or the pre-exercise meal rather than a stable training adaptation. Conversely, sampling several days after the last session may miss short-lived functional responses. Best-practice recommendations therefore favor repeated sampling and precise reporting of the interval between exercise, food intake, and specimen collection [5]. This distinction is particularly important when studies use only a baseline sample and a single post-intervention sample, because transient and persistent effects cannot be separated.

Evidence from detraining also argues against interpreting every exercise-associated shift as permanent ecological remodeling. In the six-week intervention by Allen et al., several compositional and short-chain-fatty-acid changes diminished after participants returned to sedentary behavior [6]. Reversibility is compatible with biological responsiveness, but it means that the microbial ecosystem may track current energy expenditure and dietary substrate flow rather than retain a long-lasting training memory. Longer interventions suggest that some changes can be maintained during continued exercise, yet persistence after training cessation has rarely been evaluated systematically [9-12].

A further complication is that group averages can obscure heterogeneous trajectories. Participants with similar improvements in fitness may show different microbial responses, while individuals with comparable microbial shifts may not achieve the same physiological outcome. Baseline community structure, metabolic health, habitual fiber intake, and adherence may determine whether a taxon or pathway has ecological space to increase. Future studies should predefine responder analyses, but they should also avoid post hoc taxon selection without correction for multiple testing. The most informative design would combine repeated microbiome measurements with repeated performance tests and quantify whether within-person microbial changes precede, accompany, or follow the host adaptation.

3.3. Resistance training

Resistance-training evidence has expanded but remains less consistent than endurance evidence. In young adults with overweight or obesity, six weeks of resistance training improved strength and cardiometabolic measures, whereas microbiome changes were limited and variable [13]. A ten-week resistance-training study in younger and older adults likewise reported

minimal changes in fecal microbiota and associated metabolites despite expected muscular adaptations [14].

A larger 2026 longitudinal intervention of 150 sedentary adults found no significant change in microbial diversity, community composition, or fecal metabolomics after eight weeks of supervised resistance training [15]. These null findings are scientifically important. They suggest that meaningful neuromuscular adaptation can occur without detectable remodeling of the fecal microbiome, at least over short periods and with current analytical methods. Resistance exercise may still influence microbial function in subgroups, but the evidence does not support a reproducible resistance-specific microbiome signature.

The same study nevertheless identified a responder-related signal: within-person community change was weakly associated with strength improvement, and participants with the greatest strength gains displayed more pronounced time-dependent taxonomic shifts, including enrichment of selected Lachnospiraceae-related organisms [15]. These observations are hypothesis-generating rather than proof of mediation. The reported association was small, subgroup definitions were based on achieved strength gain, and the intervention did not demonstrate that the microbial changes caused the superior response. Still, the findings illustrate why analyses limited to group-level diversity can miss potentially relevant interindividual patterns.

The contrast between robust strength gains and inconsistent microbiome changes indicates that the gut microbiome is not required as a measurable intermediate for every training adaptation. It may function as a modifier of response in selected individuals rather than a universal driver. Future resistance studies should use repeated stool sampling, dietary standardization, direct SCFA and bile-acid measurement, and sufficient statistical power for responder analyses.

3.4. Combined training and populations with metabolic impairment

Combined aerobic and resistance programs are common in clinical exercise because they target cardiorespiratory fitness, muscle function, glucose regulation, and mobility simultaneously. Their microbiome effects are difficult to attribute to a single training modality, but they may be especially relevant in older adults and people with metabolic disease, whose baseline microbial ecology, medication exposure, and physiological reserve differ from those of young athletes. In older sedentary adults, a 24-week intervention altered selected stool microbial and metabolite features alongside improvements in physical function, although the study design could not establish that these microbial changes mediated the functional response [11].

In people with type 2 diabetes, different combined-training intensities produced distinct compositional and predicted functional responses [12]. This finding is compatible with an intensity-dependent effect, but it should be interpreted cautiously because clinical populations are heterogeneous and often use medications that independently influence the microbiome. Changes in body mass, glycemic control, dietary behavior, and intestinal transit can also accompany exercise and may act as parallel causes of the microbial shift. Consequently, an exercise-associated microbiome change in a clinical trial should not automatically be classified as the biological mechanism responsible for improved glucose control.

The clinical value of microbiome measurement will depend on whether it adds information beyond established predictors such as baseline fitness, age, body composition, medication, dietary adherence, and training compliance. A microbial marker could still be useful even if it is not causal—for example, as an indicator of dietary exposure or recovery status—but predictive utility must be demonstrated prospectively. Models developed in one cohort should be tested in an external population, and apparent prediction should be compared with simpler clinical models to determine whether sequencing materially improves accuracy.

Combined-training research also offers an opportunity to study whether multiple physiological stimuli produce additive, opposing or independent microbial effects. Aerobic work may alter substrate flux, transit and cardiorespiratory demand, whereas resistance work produces different mechanical and metabolic signals. Factorial designs, or carefully sequenced training blocks, could help distinguish these components. Repeated stool and metabolite sampling should be synchronized with standardized meals and with the timing of the final endurance and resistance sessions. Without this control, modality-specific conclusions are vulnerable to the acute effects of whichever session occurred closest to specimen collection.

4. Athlete Microbiomes: Training Effect or Lifestyle Signature?

Cross-sectional studies consistently report differences between athletes and less active controls but these studies capture the combined effects of training, diet, body composition, travel, supplementation, sleep and other exposures. In professional rugby players, metagenomic pathways and fecal metabolites separated athletes from controls more clearly than taxonomic composition alone. Athletes showed enrichment of pathways related to amino-acid biosynthesis and carbohydrate metabolism and higher concentrations of acetate, propionate and butyrate [16]. The findings support functional adaptation of the microbial ecosystem, but the athletes also consumed distinct diets, including higher protein and energy intakes.

Elite Irish athletes participating in different sport classifications displayed distinct microbial functional capacity and metabolomic profiles despite limited dietary differences detected by food-frequency assessment [17]. This suggests that training type or load could contribute to sport-specific patterns. Polish endurance athletes also differed from sedentary controls in diversity and in the abundance of several taxa; numerous associations with fiber, fatty acids, folate and sucrose again demonstrated that diet and training are difficult to separate [18].

Longitudinal profiling of Chinese rowing athletes identified microbial clusters associated with elite status, dietary factors, physical characteristics, and performance [19]. However, because age and elite status were partly intertwined, the reported “elite” profile cannot be interpreted as a causal performance determinant. In elite female football players, microbiota composition remained stable across an international tournament despite multiple training sessions and matches, suggesting resilience to short-term competition stress in well-adapted athletes [20].

A matched study comparing elite athletes, physically active young adults and inactive controls found selected taxonomic differences after adjustment for diet but no clear differences in global alpha or beta diversity [21]. A large reanalysis of 418 publicly available athlete and sedentary metagenomes identified enrichment of several SCFA-producing species and biosynthetic capacities in athletes, supporting the view that functional pathways may be more reproducible than individual taxa [22].

Collectively, athlete studies show that there is no single taxonomic “athletic microbiome.” Different sports, diets, geographical regions, sexes, and training phases produce different profiles. The most defensible generalization is that high-level training and its associated lifestyle are often linked to altered microbial functional potential, particularly pathways related to carbohydrate fermentation, amino-acid metabolism, and SCFA production.

4.1. Functional convergence and ecological interpretation

The apparent inconsistency of taxonomic results does not necessarily imply that the microbiome is biologically irrelevant. Different organisms can perform overlapping metabolic functions, a property commonly described as functional redundancy. One athlete cohort may therefore be enriched in one butyrate-producing species, whereas another cohort may achieve a similar fermentation capacity through a different consortium. This may explain why metagenomic pathways and metabolomic outputs have separated athletes from controls more consistently than genus-level abundance in several studies [16,17,22]. It also supports the use of strain-

resolved metagenomics, metatranscriptomics, and direct metabolite measurements rather than assuming that a named taxon has the same function in every community.

Athlete status should also be treated as a composite exposure rather than a single variable. Years of training are accompanied by sport-specific diets, energy expenditure, body composition, sleep schedules, travel, competition stress, supplement use, and medical care. Matching athletes and controls for a short dietary record does not eliminate these long-term differences. Longitudinal within-athlete designs, such as sampling across preparation, competition, recovery and off-season phases, are therefore more informative than a single cross-sectional comparison [19-21]. Even in longitudinal research, training load should be recorded objectively and analyzed together with diet, illness, medication, and gastrointestinal symptoms.

5. Mechanisms Linking the Microbiome to Training Adaptation

5.1. Lactate cross-feeding and short-chain fatty acids

The best-known performance-related mechanism involves microbial utilization of exercise-derived lactate. Scheiman et al. observed an increase in *Veillonella* after marathon running and identified enrichment of genes that convert lactate to propionate. Administration of *Veillonella atypica* or rectal propionate increased treadmill run time in mice [23]. The study established a plausible pathway in which systemic lactate enters the intestinal lumen, is metabolized by bacteria and generates a metabolite capable of affecting performance. Nevertheless, the performance experiment was conducted in mice, and the study did not demonstrate that supplementation improves adaptation in human athletes.

SCFAs may influence training through several routes. Butyrate is a primary energy substrate for colonocytes and contributes to epithelial integrity; acetate and propionate can enter the circulation and participate in hepatic and peripheral metabolism. SCFAs also signal through G-protein-coupled receptors and histone deacetylase inhibition, potentially affecting inflammation, insulin sensitivity, mitochondrial function, and appetite. Reviews focused on athletic performance regard these pathways as biologically plausible but emphasize that direct human ergogenic evidence remains insufficient.

An important distinction is required between fecal SCFA concentration and SCFA production. Fecal values reflect the balance between production, absorption, utilization and transit and may not reliably represent systemic exposure. Future studies should combine fecal and plasma

measurements with isotope tracers, dietary fiber assessment and microbial gene or transcript analysis [24].

5.2. Gut–brain signaling and exercise motivation

A major mechanistic advance came from a mouse study demonstrating that the intestinal microbiome can influence motivation for exercise. Microbiome-derived fatty-acid amides stimulated CB1- and TRPV1-related sensory pathways, increased dopamine signaling in the ventral striatum during exercise, and improved running performance. Antibiotic depletion, sensory-neuron disruption, or dopamine blockade reduced exercise capacity, whereas restoration of the pathway rescued performance.

This work expands the concept of training adaptation beyond muscle and metabolism by suggesting that microbial signals can influence the willingness to sustain exercise. Its translational significance remains uncertain because human motivation is shaped by psychological, social and environmental factors that are not captured in mouse models. Still, the study provides a testable framework for human investigations using metabolomics, neuroimaging, perceived exertion, reward responses, and adherence outcomes [25].

5.3. Bile acids and the gut–muscle axis

Microbial bile-acid metabolism represents another possible link between the gut and skeletal muscle. In mice, transplantation of exercise-acclimated microbiota improved skeletal muscle metabolism through changes in circulating bile-acid deconjugation. Bile acids act not only as detergents but also as signaling molecules through receptors such as FXR and TGR5, which can influence glucose metabolism, energy expenditure, inflammation, and mitochondrial function [26].

The gut–muscle axis should therefore be viewed as a network rather than a single pathway. Microbial SCFAs, bile acids, tryptophan metabolites, inflammatory molecules, and intestinal hormones may interact with host-derived myokines and exercise metabolites. At present, however, most causal evidence comes from animal models, and human studies rarely demonstrate that a microbiome-derived metabolite mediates changes in VO₂max, muscle strength or recovery.

5.4. Intestinal barrier and immune regulation

Moderate, repeated exercise is generally associated with improved metabolic and inflammatory health, whereas prolonged strenuous exercise can acutely reduce splanchnic blood flow,

increase heat stress, and compromise epithelial integrity. In people with type 2 diabetes, chronic exercise was associated with changes in gut microbiota and intestinal-barrier-related measures [27]. Mechanistic reviews propose that exercise-induced microbial metabolites could reduce endotoxin translocation, improve vascular function and modify immune signaling.

The direction of effect depends on training status and dose. A transient increase in permeability after a demanding session is not equivalent to chronic dysbiosis, and trained athletes may develop protective adaptations. Conversely, inadequate energy intake, high heat load, gastrointestinal illness, non-steroidal anti-inflammatory drug use, or excessive training may overwhelm these adaptations. Therefore, microbiome findings should be interpreted alongside gastrointestinal symptoms, permeability markers, inflammatory markers and training load [28].

5.5. Evidence from controlled animal training models

Controlled animal experiments help isolate exercise modality from diet and environment. In mice, endurance and resistance training produced different taxonomic changes, and several taxa correlated differently with endurance and resistance performance. Endurance-trained animals showed higher diversity and distinct abundance of *Desulfovibrio*, whereas resistance training was associated with other genus-level changes. These data support modality-specific biological effects but also illustrate the difficulty of translating taxa from a controlled mouse colony to free-living humans.

Animal models are most informative when they use microbial depletion, colonization, transplantation, or metabolite rescue to test causality. Simple correlations between a taxon and running time-even in a controlled experiment-do not establish that the organism causes the performance difference [29].

5.6. From association to causal mediation

Evidence in this field can be arranged along a causal hierarchy. At the lowest level, a study identifies a correlation between a microbial feature and an exercise phenotype. Stronger evidence requires temporality, replication, and a dose-related relationship. Experimental depletion or transplantation can then test whether the microbiome is necessary or sufficient for part of the phenotype, while metabolite supplementation or receptor blockade can identify the relevant signaling step. Finally, a human intervention must show that deliberately modifying the candidate pathway changes a prespecified performance or adaptation outcome without unacceptable adverse effects.

The *Veillonella*, gut-brain and bile-acid studies illustrate different stages of this hierarchy. The *Veillonella* work combined athlete metagenomics with bacterial administration and metabolite experiments in mice [23]. The gut-brain study used antibiotics, neuronal manipulations, metabolite restoration, and dopamine-related experiments to build a detailed mechanistic chain [25]. The exercise-acclimated microbiota study used transplantation and bile-acid-related analyses to connect microbial adaptation with skeletal muscle metabolism [26]. These designs are more persuasive than taxonomic association alone, but each still requires independent replication and direct human testing.

For human training studies, formal mediation analysis would provide a useful intermediate step. A candidate microbial function should change after the exercise exposure, predict subsequent change in the host outcome and explain a measurable proportion of the training effect after relevant confounders are considered. Such analyses require larger samples and more time points than most current studies. They also require prespecified pathways because testing thousands of taxa as potential mediators creates a severe multiple-testing problem. Ultimately, causality will be strengthened by randomized trials of defined dietary substrates, microbial strains or metabolites combined with standardized training, rather than by commercial microbiome scores that have not been validated against adaptation outcomes [5].

6. Implications for Endurance, Strength, Recovery, and Health

6.1. Endurance adaptation and substrate metabolism

For endurance adaptation, the most plausible microbial contributions involve SCFA availability, lactate cross-feeding, intestinal integrity, substrate metabolism, and inflammation. Human interventions show that endurance training can modify microbial function, but the association with VO₂max is inconsistent and often secondary. The clearest performance-enhancing microbial experiment remains the *Veillonella* mouse model [23], not a human training trial.

A central question is whether microbial metabolism can meaningfully supplement substrate availability during repeated endurance exercise. Fermentation of nondigestible carbohydrate generates SCFAs, but their contribution should not be equated with the direct oxidation of ingested carbohydrate during competition. Their potential value is more likely to involve chronic support of epithelial metabolism, insulin sensitivity, immune regulation, and mitochondrial signaling than a large immediate energy contribution. The propionate-related *Veillonella* model is an important exception because it links an exercise-derived host metabolite

with a microbial product and a performance effect, although this pathway has not yet been shown to enhance human training responses.

Endurance athletes may also experience a trade-off between high carbohydrate availability for performance and sufficient fermentable substrate for the distal colon. Low-residue strategies can be appropriate before selected events to reduce gastrointestinal symptoms, but chronically restrictive eating patterns may reduce substrate diversity for the microbiota. Periodized nutrition research should therefore distinguish temporary competition strategies from habitual diet. Studies that report only macronutrient percentages cannot determine whether participants consumed diverse whole-plant foods, resistant starch, or specific fibers capable of supporting microbial fermentation [23,24].

6.2. Strength, hypertrophy, and the gut–muscle axis

For strength and hypertrophy, current evidence is weaker. Several resistance interventions produce substantial gains in strength without significant microbiome remodeling [13-15]. This does not exclude subtle functional effects, but it argues against presenting microbiome optimization as a necessary component of muscle adaptation. Protein intake is also a major confounder because high-protein diets can alter fermentation substrates and microbial metabolites independently of resistance exercise.

Theoretical gut–muscle mechanisms include modulation of systemic inflammation, insulin sensitivity, amino-acid availability, mitochondrial function, and bile-acid signaling. However, these pathways should not be used to infer that a larger abundance of a selected bacterium will increase muscle protein synthesis. Resistance adaptation is dominated by mechanical tension, training volume, progressive overload, protein intake, energy availability, sleep and individual responsiveness. Microbial effects, if present, are likely to modify this background rather than replace established determinants. The current human data therefore support studying the microbiome as a possible source of response heterogeneity, not as a prerequisite for strength gain [13-15].

6.3. Recovery, illness and gastrointestinal tolerance

Recovery may be influenced through gastrointestinal tolerance, immune regulation, and inflammatory signaling. Athlete-focused reviews discuss potential roles for SCFA-producing bacteria and probiotics in reducing respiratory or gastrointestinal illness and supporting recovery, but results are strain-specific and should not be generalized to all probiotic products

[30,31]. Evidence for direct improvements in performance is considerably less consistent than evidence for selected symptom or immune outcomes.

Recovery outcomes also need clearer definition. Reduced delayed-onset muscle soreness, lower inflammatory markers, fewer upper respiratory symptoms, improved stool consistency, and faster restoration of performance represent different endpoints and may involve different mechanisms. A probiotic that reduces gastrointestinal symptoms during travel should not automatically be described as performance-enhancing. Athlete-focused reviews indicate that benefits depend on the strain, dose, duration, host population, and outcome selected. Pooling distinct products under the term “probiotic” can obscure both beneficial and null results [30,31].

The intestinal barrier is especially relevant during prolonged exercise in heat, when hypoperfusion, hyperthermia, dehydration and mechanical stress can increase gastrointestinal disturbance. Chronic training may improve tolerance, yet high training load combined with inadequate energy intake, illness, or non-steroidal anti-inflammatory drug use may increase vulnerability. Microbiome measurements are therefore most useful when integrated with symptom logs, core or skin temperature, hydration status, permeability markers, inflammatory measures, and objective workload. Without this context, a post-exercise taxonomic difference cannot identify the cause of gastrointestinal distress or recovery failure [27,28].

6.4. Practical translation for athletes and clinicians

The most practical interpretation is that established behaviors – adequate energy availability, dietary diversity, sufficient fermentable fiber, appropriate carbohydrate intake, sleep, hydration, and progressive training—support both host adaptation and microbial ecology. Current evidence does not justify selecting training programs or supplements from a commercial stool profile.

A food-first approach is consistent with the available evidence. Adequate total energy, varied plant foods, whole grains, legumes, fruits, vegetables, nuts and other tolerated sources of fermentable carbohydrate can support microbial metabolic diversity while also meeting conventional nutritional goals. These principles should be individualized for athletes with irritable bowel syndrome, inflammatory bowel disease, food allergy or exercise-associated gastrointestinal symptoms. A strategy that improves microbial diversity but worsens symptom burden or compromises carbohydrate availability would not represent a successful sports-nutrition intervention.

Commercial stool tests currently have limited capacity to prescribe training or predict adaptation. Relative abundance can vary between samples, reference ranges are not standardized for athletic populations, and many algorithms are proprietary. A report may identify organisms or pathways associated with health in population studies without demonstrating that changing them will improve the individual athlete's performance. Until prospective validation is available, testing may be useful in research or selected clinical evaluation, but it should not displace medical assessment, established nutrition practice, or objective monitoring of training response [5,30].

7. Major Moderators and Sources of Heterogeneity

Diet may explain as much or more variance than exercise. Fiber quantity and diversity affect SCFA production, while protein source and dose influence proteolytic fermentation. Athletes also differ in carbohydrate periodization, supplements, energy intake, and food timing. Short dietary records may not capture habitual exposure.

Adiposity and metabolic health. The response to exercise differs between lean and obese participants. Insulin resistance, medication use, and baseline dysbiosis may change both the magnitude and direction of microbial responses [6].

Age and sex. Older adults may have lower baseline diversity, altered transit, different medication exposure, and greater potential for change. Female athletes remain underrepresented, and menstrual status, contraceptive use and energy availability are rarely integrated into microbiome analyses.

Training dose and status. Acute, unaccustomed exercise differs from chronic training. A workload that is moderate for an athlete may be vigorous for a sedentary participant. Studies should report intensity relative to individual capacity, session duration, weekly volume, adherence and detraining.

Sampling and sequencing. A single stool sample captures a limited time point. Technical choices can influence detected taxa and 16S sequencing provides limited strain and functional resolution. Standardization recommendations now favor repeated sampling, harmonized metadata, shotgun metagenomics where feasible and transparent bioinformatic reporting [5].

Compositional data and multiple testing. Relative abundance data are constrained and can produce misleading changes when one taxon expands or contracts. Microbiome studies test

hundreds or thousands of features, making false-positive control essential. Null findings after multiple-testing correction should not be reframed as proof of a broad beneficial shift.

Medication and supplement exposure. Recent antibiotics can cause large and persistent disturbances that are difficult to distinguish from training effects. Proton-pump inhibitors, metformin, laxatives, non-steroidal anti-inflammatory drugs and other commonly used medications may also modify microbial ecology or gastrointestinal physiology. Supplements add further complexity: probiotics are strain-specific, prebiotic fibers vary in fermentability, and high-dose protein, iron, polyphenols, sweeteners, or sports foods can alter substrate availability. Detailed exposure reporting and appropriate washout or stratification are therefore essential [5,30,31].

Geography, travel, and temporal behavior. Microbial communities reflect long-term geography, food culture, household contacts, sanitation, and environmental exposure. International athletes may experience repeated travel, jet lag, altered meal timing, unfamiliar food and acute competition stress. These factors can coincide with the most intensive training periods and may be mistakenly attributed to exercise. Studies should record travel, time-zone change, sleep timing and competition phase and should avoid combining geographically distinct cohorts without appropriate statistical control. The marked differences among athlete studies from Ireland, Poland, China, and other settings reinforce the need for replication across populations [16-22].

8. Limitations of the Evidence and of This Review

The evidence base is limited by small samples, short interventions, incomplete dietary control, varied exercise prescriptions, and inconsistent microbiome methods. Many studies are exploratory secondary analyses and are not powered for microbiome endpoints. Cross-sectional athlete studies cannot establish directionality, and animal mechanisms may not operate with the same effect size in humans.

Systematic reviews published through 2026 conclude that intervention evidence remains heterogeneous and that links between microbial changes and actual performance outcomes are rarely demonstrated [32]. Publication bias is also possible because taxon-level positive findings are easier to emphasize than null community-level results.

Outcome selection is another limitation. Many exercise studies designate microbial diversity or the abundance of selected taxa as an outcome but do not measure training adaptation beyond

body composition or a single fitness test. Conversely, performance studies may collect stool samples without sufficient power or temporal resolution for microbiome analysis. A clinically or athletically useful result requires alignment among the exercise stimulus, the expected physiological adaptation, the proposed microbial pathway, and the timing of each measurement. The systematic review evidence indicates that few interventions have directly connected microbiota change with performance change, and even fewer have tested whether the relationship is causal [32].

This article is a narrative review. Although the search was targeted and the references and DOI records were verified, the review did not use duplicate screening, a registered protocol, a formal risk-of-bias instrument, or quantitative reanalysis. Study selection and interpretation may therefore reflect author judgment. The date range also means that relevant publications appearing after 23 July 2026 are not included.

9. Future Research Priorities

Key priorities include:

Future studies should begin with a defined causal model rather than an unrestricted search for taxa that change after exercise. For example, a trial focused on lactate cross-feeding should measure exercise lactate exposure, microbial genes or transcripts involved in lactate utilization, relevant circulating and fecal metabolites, and a performance outcome expected to respond to that pathway. A trial focused on intestinal integrity should align training stress with symptoms, permeability, inflammation, and recovery. This pathway-first approach would reduce interpretative ambiguity and improve reproducibility.

- Diet-controlled randomized trials lasting at least 12–24 weeks, with sufficient sample size for microbiome outcomes and predefined primary hypotheses.
- Repeated sampling before training, during adaptation, after key sessions and during detraining to separate transient responses from persistent adaptation.
- Integration of shotgun metagenomics, metatranscriptomics, targeted and untargeted metabolomics, and host measures rather than reliance on 16S taxonomic profiles alone.
- Direct testing of mediation: a microbial or metabolite change should statistically and experimentally explain part of the change in VO₂max, strength, recovery, or adherence.

- Human translation of mechanistic pathways involving *Veillonella*, fatty-acid amides, bile acids, and gut–brain signaling, with careful safety and replication studies.
- Greater inclusion of female athletes, older adults, diverse ethnic and geographic populations, and resistance-trained cohorts.
- Consensus standards for sample handling, diet and medication reporting, training-load quantification, data sharing, and compositional statistical analysis.

Studies should also publish negative and null findings with the same detail as positive taxonomic results. Preregistered hypotheses, shared analysis code, accessible sequencing data and validation in an independent cohort would help distinguish reproducible biology from cohort-specific signals. Where possible, investigators should report absolute microbial abundance in addition to relative abundance, quantify dietary intake repeatedly and specify whether analyses concern acute exercise, chronic training, competition or detraining. These practices would make it possible to determine whether the microbiome is a mediator of adaptation, a marker of the athletic lifestyle, or both.

10. Conclusion

Evidence from 2018–2026 supports a bidirectional relationship between exercise and the gut microbiome, but the strength of evidence differs across questions. Human exercise interventions show that microbial composition and function can change with training, especially after endurance or combined programs, yet effects are generally modest, heterogeneous, and dependent on host and methodological factors. Resistance training often improves strength without detectable fecal microbiome remodeling.

Athletes frequently differ from sedentary controls at taxonomic, functional and metabolomic levels, but no universal athletic microbiome has been identified. The most compelling causal pathways – lactate conversion by *Veillonella*, microbiome-dependent dopamine signaling and bile-acid-mediated effects on muscle metabolism – are supported primarily by animal experiments. Consequently, the microbiome should currently be considered a promising modifier and research target rather than an established determinant of human training adaptation.

The field will advance when studies move beyond descriptive taxonomic associations and demonstrate that specific microbial functions mediate measurable improvements in performance, recovery, health or exercise adherence. Until then, microbiome-oriented

recommendations should remain grounded in established nutrition, training and recovery practices rather than unvalidated personalized testing.

Disclosure

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Declaration on the Use of Artificial Intelligence

AI-assisted tools were used exclusively for linguistic refinement. The authors take full responsibility for the scientific content, interpretation of the data, and final version of the manuscript.

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