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Review

Gut Microbiota and Exercise: A Systematic Review of Interventions and Evidence Limitations

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ABSTRACT

This systematic literature review assessed whether nutritional supplement, dietary and exercise interventions influence gut microbiota and subsequent exercise performance. Following Preferred Reporting Items for Systematic Reviews and Meta-Analyses guidelines, a comprehensive search was conducted across five databases (Ovid MEDLINE, EMBASE, CINAHL Complete, Web of Science and Scopus) up to February 2025. Included studies involved healthy, active adults undergoing nutrient supplementation, dietary and/or exercise interventions with a control or placebo comparator. Outcomes included faecal bacterial composition (α-diversity, relative abundance), short-chain fatty acids, in adjunct with exercise performance (i.e., time-trial, time to exhaustion, maximal strength). Eighteen studies met the inclusion criteria. Due to methodological heterogeneity, a descriptive synthesis was performed. Changes in faecal microbiota diversity and composition were highly variable and largely minimal. Short-chain fatty acid outcomes were infrequently assessed; only one study reported a significant increase in faecal acetate concentration following yoghurt supplementation containing *Bifidobacterium animalis* subsp. *lactis* BL-99. Only seven studies examined the relationship between changes in faecal bacterial profile and performance outcomes, with limited or inconclusive findings. No consistent performance benefits were observed in relation to microbiota changes. Risk of bias and methodological limitations were common, including variation in interventions, outcome measures and microbiota analysis methods. Taken together, the current evidence base remains too limited and heterogeneous to draw firm conclusions about the efficacy of microbiota-targeted interventions for enhancing exercise performance in healthy, active adults. Future studies employing standardised methods, mechanistic outcome measures and longitudinal designs may help clarify the potential of microbiota modulation as a performance-enhancing strategy.

Keywords exercise-induced gastrointestinal syndrome, exercise-associated gastrointestinal symptoms, probiotics, bacteria, short-chain fatty acids, time-trial

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Introduction

General overview of the gut microbiota

The gut microbiome comprises approximately 40 trillion microorganisms, including bacteria, viruses, archaea and fungi. Of these, bacteria are the most abundant, residing throughout the gastrointestinal tract, with the highest density in the large intestine and lowest in the stomach.[1,2] This distribution reflects compositional differences between sections of the gastrointestinal tract.[3] The human gut microbiota plays essential roles in immunomodulation, food digestion, nutrient absorption, metabolite production and vitamin metabolism, and the maintenance of intestinal epithelium integrity.[4–6] Most gut bacteria in humans belong to four dominant phyla: Bacillota (previously Firmicutes), Bacteroidota (previously Bacteroidetes), Actinomycetota (previously Actinobacteria) and Pseudomonadota (previously Proteobacteria),[6] according to the International Code of Nomenclature of Prokaryotes (ICNP).

Athlete-specific microbial profiles

This bacterial distribution is similarly observed in athletes, with Bacillota (62–74 %), Bacteroidota (10–34 %), Actinomycetota (1–3 %) and Pseudomonadota (1–9 %) consistently reported as predominant phyla. At the family level, Lachnospiraceae (25–33 %), Ruminococcaceae (20–34 %), Bacteroidaceae (5–20 %) and Prevotellaceae (5–10 %) are the most prevalent.[7–10] However, considerable variation exists at the genus and potentially species levels (e.g., *Bacteroides*, *Alistipes*, *Blautia*, *Agathobacter*, *Faecalibacterium*, *Phascolarctobacterium*, *Prevotella*, *Roseburia*, *Subdoligranulum*), with limited consistency across studies despite comparable experimental designs and attempts to control for key confounders.[7–10] This variability complicates the interpretation of microbial profiles in athletic cohorts.

Influences on gut microbiota and rationale for interventions

The human gut microbiota, defined as the collective genomes of microorganisms residing within the gut microbiome, is influenced

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by numerous factors, including age, medication, early-life exposures, diet, supplementation, pharmaceuticals, psychological stress, exercise and disease or illness.[11] Although relatively stable over time,[12] it remains responsive to external factors, with nutrient supplementation, diet and exercise recognised as primary modulators.[13–17] These alterations may, in turn, influence host metabolic and immune functions, potentially contributing to enhanced exercise performance, as suggested by Di Dio et al.,[18] Jarrett et al.,[19] Patel et al.,[20] and Santibanez-Gutirrrez et al.[21] Despite growing interest in this area, a gap remains in the scientific literature regarding the effects of intervention-induced changes in gut microbiota on direct exercise performance outcomes. These outcomes include time to exhaustion (TTE), time-trial (TT), Cooper's test, strength and anaerobic performance, as well as functional performance measures, such as balance, flexibility and muscular endurance, as well as physiological indicators like $\dot{V}O_{2max}$ or $\dot{V}O_{2peak}$. These outcomes are variably assessed across studies included in this review.[22–40]

Short-chain fatty acids and proposed mechanistic links

Growing interest in the role of the gut microbiota in physical performance has prompted exploration of both its compositional and functional attributes.[41] Research has proposed that characteristics of the gut microbiota, such as microbial diversity (e.g., α-diversity) and the abundance of short-chain fatty acid (SCFA) producing commensal bacterial species, differ between physically active individuals, including athletes, and their sedentary or non-athlete counterparts.[42] Exploratory studies have reported that SCFA-producing commensal bacteria (e.g., *Faecalibacterium prausnitzii*, *Roseburia hominis*, *Akkermansia muciniphila*), microbial metabolic pathways (e.g., amino acid and carbohydrate metabolism) and faecal metabolites (e.g., SCFAs such as acetate, butyrate and propionate) are positively correlated with physical activity. In some studies, physical activity was objectively measured using accelerometer-derived activity counts to distinguish sedentary, light and moderate-to-vigorous intensities,[43] whereas others classified participants by training status or activity level using self-reported questionnaires.[44, 45] Additionally, total exercise volume has been positively associated with a greater abundance of *Prevotella*, which in turn correlates with amino acid and carbohydrate metabolic pathways.[46] Sport-specific differences have also been reported; for example, genera such as *Faecalibacterium*, *Sutterella*, *Clostridium*, *Haemophilus* and *Eisenbergiella* have been observed in higher abundance in bodybuilders compared with sedentary controls and distance runners.[47]

Animal studies have proposed mechanistic links between specific bacterial taxa and performance outcomes.[48, 49] For instance, provision of *Veillonella atypica* improved treadmill performance in mice, potentially due to its capacity to metabolise lactate into propionate, a SCFA-linked hypothesis to influence performance. However, the precise mechanisms remain unclear.[12] While *Veillonella* has been identified in the faecal samples of highly trained endurance athletes compared with sedentary controls,[12] its abundance has not been consistently observed across other exploratory studies in endurance athletes.[7–9, 44–46] The proposal that *Veillonella* increases exercise performance has been contested. Notably, the control group in the *Veillonella* study received *L. delbrueckii* subsp. *bulgaricus*, a lactate-producing bacterium, which may have confounded the performance outcomes.[50]

More broadly, the hypothesis that propionate, or SCFAs more generally, enhances exercise performance in humans is not strongly supported. For instance, increases in faecal or plasma SCFAs (i.e., acetate, butyrate and/or propionate) following high FODMAP intakes in endurance-trained individuals have not translated to improved performance outcomes, though they may offer some protection against disturbances to gastrointestinal integrity associated with exercise-induced gastrointestinal syndrome.[8, 51, 52] SCFAs are critical to host energy metabolism and may influence skeletal muscle activity[53] via enhanced carbohydrate uptake, lipid metabolism and fatty acid oxidation,[54] processes associated with improved exercise performance.[15] Nonetheless, it remains uncertain whether performance improvements observed in animal or human studies are directly attributable to changes in gut microbiota composition, SCFA production or the intervention itself.

Biotics and microbiota-targeted strategies

Prebiotics, probiotics and synbiotics, collectively referred to as biotics, are well-studied functional food strategies for modifying the gut microbiota.[55] Prebiotics are non-digestible substrates that are fermented by gut bacteria [56]; probiotics are live microorganisms that, when consumed in sufficient quantities, confer health benefits to the host [57]; and synbiotics are combinations of the two, proposed to confer beneficial effects on gastrointestinal health [58]. Following the 2019 consensus definition, postbiotics, preparations containing inactivated microorganisms and/or their components that confer health benefits to the host, have also gained attention in scientific research and commercial applications.[59] In sport and exercise contexts, biotics are of interest both for managing gastrointestinal issues among athletes and for potentially enhancing performance.[60] Proposed mechanisms include improved nutrient absorption and gastrointestinal integrity, modulation of immune function via strengthening of the gut barrier, increased production of antimicrobial proteins (e.g., β-defensin, IgA) and regulation of cytokine secretion via NFκB and MAPK pathways.[55–57, 61, 62] Collectively, these mechanisms may contribute to enhanced physiological resilience and nutrient availability.[7, 53, 63–66] However, there remains a lack of systematic reviews assessing whether observed performance benefits are specifically linked to microbiota changes and whether methodological confounders such as diet, training load and study design are adequately addressed.

Other dietary and exercise interventions

In addition to biotics, other strategies have been explored for their microbiota-modulating potential and possible effects on exercise performance. These include α-cyclodextrin (αCD), a non-digestible carbohydrate with selective microbial effects and links to relevant metabolic pathways.[67–70] High-carbohydrate diets, particularly those rich in fermentable substrates like FODMAPs, have also been shown to modulate bacterial composition and increase SCFA production, potentially supporting gastrointestinal integrity and fuel availability during exercise.[8, 51] Physical activity itself, including both aerobic and resistance training, can independently modulate gut microbial diversity and metabolite profiles, with possible implications for host physiology and training adaptations.[20, 44, 45, 55]

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Aim and objectives

Despite growing interest and emerging evidence that both support and challenge the performance benefits of gut microbiota-targeted interventions, a systematic and comprehensive synthesis of the literature is lacking. This systematic literature review aims to critically evaluate current evidence to determine whether exercise performance can be improved through microbiota modulation via nutritional supplementation, dietary modification or exercise interventions. The review will also highlight methodological limitations and propose directions for future research in this evolving area.

Materials and methods

This systematic review was conducted following the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines[71] and registered with PROSPERO (CRD42024551751) (http://www.crd.york.ac.uk/PROSPERO).

Search strategy

A three-step search strategy was developed in consultation with an academic librarian to identify relevant English-language studies. Searches were conducted across five electronic databases, Ovid MEDLINE, EMBASE, CINAHL Complete, Web of Science and Scopus from inception to February 2025. Reference lists of included studies and additional sources known to the authors were screened to identify missed publications. The keywords applied in the literature search are shown in **Table 1**. Search terms were tailored to each database's structure (e.g., CINAHL complete, Scopus and Web of Science) to ensure comprehensive retrieval of relevant literature. Keyword selection was guided by an *a priori* gold set of known eligible articles and refined iteratively to maximise sensitivity and specificity. Athlete-related terms (e.g., triathlete, cyclist, badminton player) were included based on terms found in this gold set, while broader terms such as athlete, sports and exercise ensured inclusion of other athletic populations not explicitly named.

Eligibility criteria

Eligibility criteria were established using the Participant Intervention Comparator Outcomes Study design (PICOS) framework (**Table 2**). Studies were included if they met the following PICOS criteria: population: healthy, active adults; intervention: exercise/physical training, and/or diet, and/or nutrient supplementation; comparator: control or placebo groups; outcomes: quantified gut microbiome measures and exercise performance metrics; and study design: original human research studies. Exclusion criteria included sedentary individuals, those with disease states or established gastrointestinal disorders, populations undergoing uncontrolled dietary, exercise or supplementation modifications and studies lacking a control or placebo group.

Study selection

Search results were imported into Endnote for deduplication before being uploaded to Covidence for study selection. Two independent reviewers (SKG and IGM) screened titles and abstract for eligibility, with full-text assessment conducted for studies meeting inclusion criteria. A third reviewer (RC) resolved conflicts when consensus was not reached.

Data extraction

Two reviewers (SKG and IGM) independently extracted study data using a standardised data extraction table, with verification by a third reviewer (RC). Extracted variables included study characteristics, such as sample size, age, biological sex and training status, as well as details of the intervention, including duration, type, dose, and bacterial species or strain. Microbiome-related outcomes, such as microbial relative abundance, α-diversity and SCFA concentrations, were recorded alongside exercise performance outcomes, including time-trial, time to exhaustion and maximal strength measures. Data extraction focused on interpretable numerical results, with graphical data digitised using WebPlotDigitizer,[72] where applicable. Studies that presented results in non-extractable formats, such as heat mapping or unclear data visualisations, were excluded from analysis. Any discrepancies in data extraction were resolved by discussion and consensus, in accordance with the third reviewer (RC). Given the heterogeneity of interventions, methodologies, outcome measures and varying degrees of meeting best

Table 1 General search strategy for the systematic literature review on the effect of nutritional supplements, diet and/or exercise interventions on inducing alterations of the gut microbiota and its effects on exercise performance in a healthy active adult population

Field one (combine with OR): population		Field two (combine with OR): intervention and comparison		Field three (combine with OR): outcome
Keywords: athlete[a], athletes, walking, sports, triathlete[a], run[a], race walker[a], sportspeople, exercise[a], cyclist, cycling, ((badminton or basketball) adj2 (player[a]), ((physical or recreational or elite or competitive or endurance) adj2 (exercise or activity or performance))	AND	Keywords: short-chain fatty acids, SCFA, microbial composition, microbiome, microbiota, microflora, intestine flora, gastrointestinal microbiome, *Lactobacillus plantarum*, *Bacillus coagulan*, *Lactobacillus paracasei* PS23, *Bacillus subtilis*, *Bifidobacterium longum*, ((*Lacticaseribacillus* or *Lactobacillus*) adj2 (*casei*))	AND	Keywords: athletic performance, training, aerobic, fitness, physical fitness, skeletal muscle, isokinetic, isometric, neuromuscular, force, torque, power, strength, max[a] voluntary contraction, ((exercise or athletic or physical or endurance or sport or muscular) adj2 (performance)), ((time) adj2 (trial or exhaustion)), ((aerobic or anaerobic) adj2 (capacity))

[a]Used to retrieve unlimited suffix variations.

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Table 2 PICOS table, showing the inclusion and exclusion criteria for the study population, intervention, comparator, outcome/s and study design

PICOS	Inclusion	Exclusion
Population	Human.	Animals and in vitro studies.
	Recreational and competitive active adults (≥ 18 yr).	Infants or children.
	Male and female biological sex.	Pregnancy or lactating.
		Sedentary individuals (i.e., no adherence to exercise or structured physical activity programs). Diagnosed disease or syndrome states (i.e., all clinical populations; gastrointestinal disease/disorders (e.g., irritable bowel syndrome, inflammatory bowel disease, coeliac disease or any other functional gastrointestinal disorder/infections). Population adhering to dietary modifications and/or dietary supplementation, including pre-/pro-/syn-biotics in the 3 mo before experimental protocols. Antibiotic and/or other drugs intake (e.g., non-steroidal anti-inflammatory or stool-altering medications) within 1 mo of experimental protocols.
Intervention	Exercise and/or physical training OR diet OR nutrient supplementation OR prebiotic/s, probiotic/s and symbiotic/s blends (i.e., prebiotic + probiotic, with or without other nutrient inclusion) (e.g., vitamins, minerals, lipids, phytochemicals and/or volatiles).	No exercise and/or physical training OR dietary modification OR nutrient supplementation OR provisions of prebiotic/s, probiotic/s and symbiotic blends.
Comparator	Placebo group or control group.	No placebo or control.
Outcome	Gastrointestinal microbiota: e.g., bacterial taxonomy (ASV or OTU) including α-diversity and relative abundance, bacterial functional markers including SCFA concentration (e.g., butyrate, propionate and/or acetate).	Failure to meet outcome criteria.
	AND	
	Exercise performance: e.g., time to exhaustion, time trial, maximal strength.	
Study design	RCT or randomised crossover trial.	All other study designs.

ASV, amplicon sequence variant; OTU, operational taxonomic units; RCT, randomised control trial; SCFA, short-chain fatty acid.

practice guidelines and recommendations checklist in exercise gastroenterology research that included experimental control of confounding factors for primary variables explored in this systematic literature review,[73] a meta-analysis was not feasible and results were synthesised descriptively.

Risk of bias assessment

Risk of bias was independently assessed by two reviewers (SKG and IGM) using the Cochrane Risk-of-Bias Tool (RoB 2),[74] following established criteria. Discrepancies were resolved through discussion and consensus. The overall risk of bias rating for each study reflects the highest level of risk across any domain, in accordance with RoB 2 guidance. No studies were excluded based on their risk of ratings; however, these assessments were integral to our interpretation of findings. Studies with a higher risk of bias, particularly due to inadequate blinding and important methodological limitations such as insufficient dietary or exercise control, were interpreted with caution when considering their reported performance outcomes. While industry funding and author affiliations were noted as part of the study context, they were not used as direct indicators of study reliability. Throughout the results and discussion, risk of bias concerns and methodological limitations were explicitly highlighted to provide a nuanced understanding of the evidence, acknowledging that these factors contributed to heterogeneity and uncertainty in the overall conclusions.

Results

Search results

Results of the literature search are shown in **Fig. 1**. A total of 3,779 non-duplicate studies, including those identified via citation searching, were screened. After title and abstract screening, 3,750 were excluded. Of the 29 studies sought for retrieval (26 from databases and registers and 3 from other sources), 27 full-text articles were successfully retrieved and assessed for eligibility. Studies were excluded due to wrong study design ($n=2$), wrong study population ($n=3$) and wrong outcomes ($n=4$), resulting in 18 studies being included in the final review (**Fig. 1**).

Study characteristics

Outcomes are reported from a total of 588 participants. The majority were male (67 %), with participant ages ranging from 19 to 69 years. The populations studied included $n=5$ active adults,[22,33,38] $n=1$ active elderly,[26] $n=2$ football players (semi-professional and professional soccer; Tier 2–3),[29,32] $n=1$ triathletes (described as

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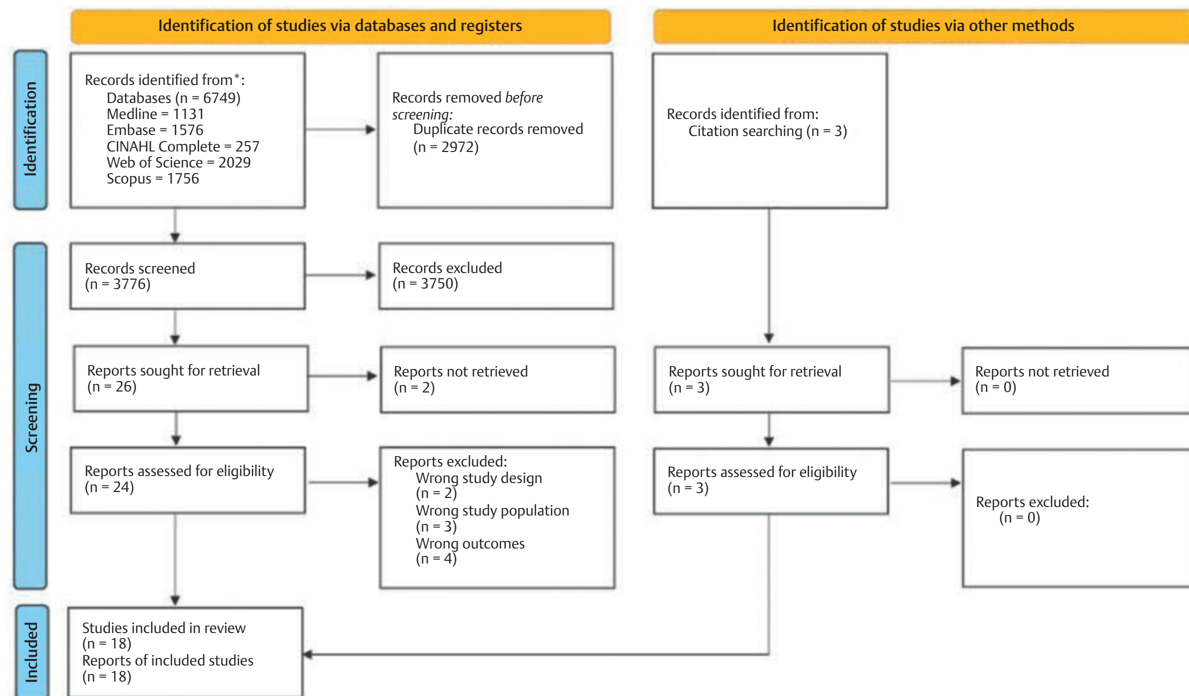

Fig. 1 PRISMA diagram illustrating the systematic review process and the inclusion and exclusion of research papers. [71]

elite but lacking sufficient detail to classify by Tier),[23] *n* = 1 national level cross-country skiers (Tier 3),[28] *n* = 1 cyclists (Tier 2),[37] *n* = 1 well trained MMA (Tier 2),[34] *n* = 4 runners (Tier 2–3)[25,27,30,36] and *n* = 1 race walkers (Tier 4).[31]

Interventions included *n* = 11 probiotic and/or postbiotic trials (*n* = 8 single strain, *n* = 3 multi-strain);[22,23,25,28,30,32,34,36–38,40] and *n* = 2 using other nutritional supplements containing α-Cyclodextrin.[33,39] Other interventions included *n* = 3 dietary modifications: (1) a high protein diet (40 % protein/30 % carbohydrate/ 30 % fat) or a high carbohydrate diet (60 % carbohydrate/10 % protein/30 % fat)[27]; (2) a ketogenic Mediterranean diet with phytoextracts (< 30 g/d carbohydrate, comprising < 10 % of total energy intake, 25–30 % of energy from protein, with fat consumed *ad libitum* and three herbal extracts)[29]; and (3) high-carbohydrate (HCHO) diet (60 % carbohydrate/16 % protein/20 % fat) and periodised carbohydrate (PCHO) diet (same macronutrient composition as HCHO, but periodised across days/sessions).[31] Additionally, a low-carbohydrate, high-fat (LCHF) diet was employed, consisting of 78 % fat, 17 % protein and < 50 g/d carbohydrate, comprising approximately 3.5 % of total energy intake. Furthermore, *n* = 1 exercise intervention was included, which involved a 60-minute exercise program performed four times per week. The program comprised a 10-minute warm-up, 20 minutes of aerobic exercise, 25 minutes of resistance exercise and a 5-minute cool-down.[26]

Among the probiotic and/or postbiotic interventions, delivery formats included capsules (*n* = 7),[22,23,25,30,37,38,40] tablets (*n* = 1),[34] sachets (*n* = 1),[36] yoghurt (*n* = 1)[28] and kefir (*n* = 1).[32] Intervention durations ranged from 2 to 11 weeks. Outcomes assessed across studies included direct exercise performance measures such as TTE, TT, distance test, vertical jump height, strength assessments (i.e., knee extensor/flexor, grip and isometric quadriceps), peak power output, fatigue index, single leg standing with eyes closed and the 2-minute step test. In addition, physiological indicators of aerobic capacity, such as $\dot{V}O_{2max}$, were also reported in several studies.

Due to the heterogeneity of study designs, interventions and outcome measures, results are presented descriptively in **Table 3** (study characteristics) and **Table 4** (study outcomes); expanded outcome data corresponding to **Table 4** are provided in **Supplementary Table S1** (available in the online version only). Wu et al.[38] conducted a post hoc analysis of data from Lee et al.,[24] and Murtaza et al.[31] performed a post-hoc analysis of data originally published by Burke et al.[75] Przewłócka et al.[34,35] reported different performance outcomes from the same trial in two separate publications.

Risk of bias assessment

Results of the risk of bias assessment are presented in **Table 5**. Several studies did not explicitly state their blinding or randomisation procedures, or blinding was not possible due to the nature of the intervention. These studies were therefore assessed as having 'some concerns' or 'high risk' in certain domains.[23,25,31,32,40] Most included studies, however, had an overall low risk of bias. More than half were either directly funded by, received supplementation and/or placebo products from or included authors affiliated with, commercial product manufacturers (i.e., probiotic, herbal or

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Table 3 Systematic literature review search results and study characteristics of included studies investigating the impact of nutritional supplement-, dietary- and exercise-associated changes to the gut microbiota and their impact on athletic performance

Author, country, study design	Population characteristics	Intervention protocol (vs. placebo or control)	Dietary control (DC) / Physical activity (PA)	Faecal collection technique	Faecal analysis technique[b]	Funding source and conflict(s) of interest
Probiotic and postbiotic studies—single strain						
Gross et al.,[22] USA, RXT	*N* = 7 (3 males and 4 females), age: 31 ± 8 yr, physically active, Tier 1, $\dot{V}O_2$ peak (running) (mL/kg/min): 49.2 ± 8.4.	*Veillonella atypica* FB0054 (VA), 1 × 10^{10} CFU/cap, 1× capsule consumed with 8–12 ounces water, Pla: corn starch, duration: 14 d with 21 d washout period.	DC[a]: Completed 2-d food/fluid log, instructed to replicate diet before each study visit. Fasted overnight (e.g., avoiding food, caffeine, nicotine). Dietary records analysed for nutritional composition (energy, carbohydrate, protein, fat). PA: Avoided vigorous exercise at least 24 h before each visit. Required to engage in aerobic exercise at least 2 × /wk; typically exercised 5.4 ± 1.5 d/wk, with 6.7 ± 0.8 d of overall PA.	Participants self-collected stool samples using provided kits, froze them at −20 °C with preservation reagents (RNA Later and OMNIgene) and transported them on ice to the lab for storage at −80 °C.	Shotgun metagenomics analysis, α-diversity, Shannon entropy, SCFA not measured.	Funded by FitBiomics (NY) and Increnovo, LLC (WI). One author is a scientific advisor to FitBiomics and others have ownership stakes and patents on the probiotic strain used. These authors had no role in data collection or analysis.
Huang et al.,[23] Taiwan, RCT	*N* = 20 (male), age: Int: 21.6 ± 1.3; Pla: 21.9 ± 1.4 yr, Triathletes, $\dot{V}O_{2max}$ (running) (mL/kg/min) Int: 55.5 ± 8.6; Pla: 56.6 ± 9.0.	*Lactiplantibacillus plantarum* PS128, 1.5 × 10^{10} CFU/cap + 100 mg microcrystalline cellulose, 2 capsules after training/before sleeping, Pla: 400 mg microcrystalline cellulose, duration: 4 wk.	DC[a]: Instructed to avoid fermented foods, probiotics, prebiotics and antibiotics. Dietary records analysed for nutritional composition and caloric intake (data not reported). PA[a]: Daily training for 4 wk, not monitored/reported. Maintain a regular lifestyle, avoiding any strenuous exercise.	Fresh stool samples collected; collection procedure and handling prior to lab processing not specified.	16S rRNA gene amplicon sequencing targeting V1–V3, α-diversity, Shannon index, GC–MS analysis.	Supported by Ministry of Science and Technology in Taiwan (grant nos. MOST 107-2321-B-158-001 and MOST 108-2410-H-038-017).
Lee et al.,[40] Taiwan, RCT	*N* = 53 (26 males and 27 females), age: control: 21.6 ± 1.6; TWK-10 21.3 ± 1.7; TWK10-hk 21.6 ± 2.5 yr, physically active, Tier 0–1, $\dot{V}O_{2max}$ (running) (mL/kg/min): Pla: 47.3 ± 8.3; TWK10: 46.8 ± 9.3; TWK10-hk: 47.5 ± 10.2.	TWK10: viable *Lactiplantibacillus plantarum* TWK10-hk: heat-killed *Lactiplantibacillus plantarum*, 1.0 × 10^{10} CFU/cap, 3 capsules, Pla: maltodextrin, microcrystalline cellulose, duration: 6 wk.	DC[a]: Instructed to maintain usual diet, to cease supplements (e.g., probiotics, prebiotics, antibiotics) 2 wk pre-intervention. Baseline energy intake recorded. PA[a]: Instructed to avoid any strenuous physical activity for 3 d before $\dot{V}O_{2max}$ and exercise performance tests. No activity monitoring stated.	Fresh stool samples collected; collection procedure not specified prior to lab processing.	16S rRNA gene amplicon sequencing targeting V3–V4, α-diversity, Shannon index, HPLC.	Some authors employed by Synbio Tech Inc.
Li et al.,[28] China, RCT	*N* = 16 (male), age control: 19.3 ± 0.7; Int: 19.6 ± 1.1 yr, national top-level cross-country skiers, Tier 3, $\dot{V}O_{2max}$ (running) (mL/kg/min): control: 55.9 ± 4.4; Int: 55.8 ± 5.4.	Yogurt with *Bifidobacterium animalis* subsp. *lactis* BL-99, 1 × 10^9 CFU, control: ordinary yoghurt, yoghurt with each of 3 meals and at 21:00, 4× d, duration: 8 wk.	DC[a]: Instructed to maintain usual diet; no dairy, yogurt-containing foods and supplements 1 wk pre-trial. Completed 2-d weighed food/fluid diary. [NS] differences in energy or macronutrient intake between groups. PA: Firstbeat Sport Sensor and Bodyguard 2 monitored training load and energy expenditure. [NS] changes in TRIMP or energy expenditure between groups.	Faecal samples collected by researchers; participant collection procedure not described.	DNA Nanoball Sequencing (DNB-seq) using combinatorial probe-anchor synthesis (cPAS), α-diversity, Shannon index, plasma-targeted metabolomic analysis via LC-MS.	Funded by the National Key R&D Program of China (grant no. 2019YFF0301700).

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Table 3 Continued

Author, country, study design	Population characteristics	Intervention protocol (vs. placebo or control)	Dietary control (DC) / Physical activity (PA)	Faecal collection technique	Faecal analysis technique[b]	Funding source and conflict(s) of interest
Lin et al.,[25] Taiwan, RCT	*N* = 21 (14 males and 7 females), age: Pla (mean ± SEM): 21.2 ± 0.4; Int: 21.6 ± 0.7 yr, healthy, well-trained middle and long distance runners, Tier 2, fitness status not reported.	OLP-01, human strain probiotic *Bifidobacterium longum* subsp. *longum*, 5 × 10⁹ CFU/cap, after each meal, 3 × d, Pla: Maltodextrin, duration: 5 wk.	DC[a]: Instructed to avoid nutritional supplements, yogurt, Yakult, probiotic products, antibiotics. Team dietitian 'specified the diet' and 'provided the same meal' to ensure consistency of the diet. Nutrition composition data not reported, monitoring of adherence not reported. PA: Experiment included 3 wk regular training and 2 wk of de-training. All subjects followed team's work-rest schedule (data not reported).	Faecal samples were self-collected using DNA/RNA Shield-preserved tubes with attached spoons and stored at −80 °C for subsequent DNA extraction and sequencing; further collection details not provided.	16S rRNA gene amplicon sequencing targeting V3–V4, SCFA not measured.	Funded by projects from the university-industry cooperation fund (NTSU No. 1091038), National Taiwan Sport University, Taoyuan, Taiwan. Glac Biotech Co., Ltd. (Tainan City, Taiwan) provided probiotics.
McDermott et al.[30] USA RCT	*N* = 28 (13 males and 15 females), age: Pla: 25.6 ± 4.9; Int: 24.6 ± 5.1 yr, Healthy, adult runners, Tier 2, $\dot{V}O_{2max}$ (running) (mL/kg/min): Pla 48.8 ± 6.5; Int: 49.4 ± 6.1.	*Lactobacillus helveticus* Lafti L10 with same excipients as Pla capsule, 5 × 10⁹ CFU/cap, 1 × d with a meal, Pla: potato starch, ascorbic acid, and magnesium stearate, duration: 6 wk.	DC[a]: Instructed to consume standardised low-fat breakfast. Dietary habits assessed at baseline and post-intervention. [NS] between groups for fibre intake and total diet quality scores. PA: PA self-reported (questionnaires) and tracked (accelerometer). Int: performed fewer aerobic sessions ($p = 0.02$) and less vigorous exercise ($p = 0.007$) than Pla. [NS] difference in strength, combination training or moderate/very vigorous exercise minutes.	Stool samples collected using a commode specimen collection system (Thermo Fisher Scientific) and a nucleic acid preservation tube (Norgen Biotek). Pea-sized samples were taken from three different stool sites and mixed with preservative. If a sample was not provided during the first visit, participants were instructed to collect it at their next bowel movement. Samples	Real-Time qPCR, SCFA not measured.	Funded by Lallemand Health Solutions Inc. and USDA National Institute of Food and Agriculture Hatch project (FLA-FOS- 510 006391.
West et al.[37] Australia RCT	*N* = 88 (64 male and 35 female recruited), Competitive cyclists, Tier 2, Age: Int: M: 35.2 ± 10.3, F: 36.5 ± 8.6; Pla: M: 36.4 ± 8.9, F: 35.6 ± 10.2 yr, $\dot{V}O_{2max}$ (mL /kg/min): Int: M: 56.5 ± 6.2, F: 53.0 ± 5.0; Pla: M: 55.8 ± 5.6, F: 51.6 ± 7.4.	*Limosilactobacillus fermentum* VRI-003 PCC, 1 × 10⁹ CFU/cap, 1 × d, consume any time with or without food. Pla: microcrystalline cellulose, duration: 11 wk.	DC[a]: 4-d food diary. Usual diet, without probiotic foods. No substantial differences between groups for energy, macronutrients, fibre. PA: Training log kept. No substantial differences between groups.	Faecal samples collected in sealable plastic bags and immediately frozen in a portable −20 °C freezer for transport to the laboratory, though the time between collection and lab arrival is not specified.	16S rRNA gene diversity analysis using DGGE and qPCR, SCFA not measured.	Funded by Christian Hansen A/S, Probiomics, and the Australian Institute of Sport. Two of the authors held full-time positions with Christian Hansen A/S and Probiomics Ltd, respectively.

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Table 3 Continued

Author, country, study design	Population characteristics	Intervention protocol (vs. placebo or control)	Dietary control (DC) / Physical activity (PA)	Faecal collection technique	Faecal analysis technique[b]	Funding source and conflict(s) of interest
Wu et al.,[38] Taiwan, RCT	*N* = 105 (75 males and 30 females), age: Pla: 21.6 ± 2.0; L-PS23 21.4 ± 1.3; HT-PS23: 21.8 ± 2.5 yr, physically active, Tier 0–1, not well described.	*Lacticaseibacillus paracasei* PS23 (L-PS23) (DSM 32322) previously isolated from healthy human faeces, HT-PS23: heat-treated L-PS23, 1 × 10 billion CFU/cap, 2 × d, Pla: microcrystalline cellulose, duration: 6 wk.	DC[a]: Instructed to maintain usual diet, to cease supplements (e.g., probiotics, prebiotics, antibiotics). 3-d food diary with meal photos including scale completed at baseline and end. [NS] differences in energy, macronutrient or fibre, non-starch polysaccharides, intake between or within groups. PA[a]: Not monitored.	Fresh stool samples were collected and immediately placed in containers with 95 % ethanol for DNA preservation, then transported to the laboratory and stored at − 80 °C until DNA extraction. The time between collection and transportation to the lab is not specified.	16S rRNA gene amplicon sequencing targeting V3–V4, α-diversity, Shannon index and Chao1, UPLC–MS/MS.	Bened Biomedical Co., Ltd provided the capsules. The study was supported by the University-Industry Cooperation Fund, National Taiwan Sport University (NTSU No. 1111040) and partial funding to SIW from the Department of Medical Research, Mackay Memorial Hospital (MMH-113-19, MMH-110-110, MMH-109-112, MMH-109-14, MMH-108-121, MMH-108-146, MMH-TT-10804, MMH-TH-10804). Funders had no role in study design, data collection, analysis, interpretation, writing or publication.
Probiotic studies—multi-strain						
Przewłócka et al.,[35] Study 1 Poland, RCT	*N* = 23 male well-trained MMA athletes, Tier 2, age: control: 26.02 ± 4.00; Int: 24.70 ± 6.50 yr, $\dot{V}O_{2max}$ (cycling) (mL/kg/min): control 52.33 ± 5.06; Int: 56.92 ± 0.83.	*Bifidobacterium lactis* W51, *Levilactobacillus brevis* W63, *Lactobacillus acidophilus* W22, *Bifidobacterium bifidum* W23 and *Lactococcus lactis* W58 + 5 mL Vit D3 (0.5 mg Cholecalciferol/mL), 5×10^8/tablet, 4 × d with meals, control: maltodextrin and plant proteins + 5 mL Vit. D3, duration: 4 wk.	DC[a]: Participants were instructed to maintain their usual diet. Pre-exercise breakfast was standardised. Dietary intake was assessed via a 3-d food interview and FFQ by a qualified sports nutritionist. Nutrient intake (energy, carbohydrate, fat, protein) was analysed using professional software; nutritional composition data were not reported. PA: Instructed to maintain regular training (at least 5 × wk MMA sessions, 60–90 min, including combat, grappling, striking, strength and endurance). No exercise 24 h before testing. Training log kept and qualified sports nutritionist conducted interview before and after intervention to assess training load.	This was assessed in Przewłócka et al.[34]	This was assessed in Przewłócka et al.[34]	Partially funded by project from Ministry of Education and Science: IDUB 664/306/63/73-3326.
Przewłócka et al.,[34] Study 2 Poland, RCT	As per Przewłócka et al.[35]	As per Przewłócka et al.[35]	As per Przewłócka et al.[35]	Faecal samples collected by participants into a standardised container before and after the intervention period, following instructions provided by the researchers. Samples were immediately frozen and stored at − 80 °C for analysis.	*Shallow shotgun sequencing*, α-diversity, Inverted Simpson, Chao1, ACE, Shannon, gas chromatography with flame ionisation detection.	As per Przewłócka et al.[37]

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Table 3 Continued

Author, country, study design	Population characteristics	Intervention protocol (vs. placebo or control)	Dietary control (DC) / Physical activity (PA)	Faecal collection technique	Faecal analysis technique[b]	Funding source and conflict(s) of interest
Wang et al.,[36] China, RCT	*N* = 19 (15 males and 4 females), active amateur marathon runners, Tier 2, age: Int: 28.50 ± 12.18; Pla: 29.78 ± 12.39 yr, $\dot{V}O_{2max}$ (cycling) (mL/kg/min): not reported, training hx: Int: 5.10 ± 2.02 yr; 188 ± 41.31 km/mo; Pla: 5.00 ± 2.35 yr; 183 ± 40.93 km/mo.	*Lactobacillus acidophilus* and *Bifidobacterium longum*, 1.5 g/d (1.5×10^9 CFU: 1.02×10^9 CFU, *Lactobacillus acidophilus* and 4.95×10^8 CFU *Bifidobacterium longum*), 1 × d sachet, Pla: Sachet containing maltodextrin, duration: 5 wk.	DC[a]: Received 'assigned diet'—no details about this diet. Participants were instructed to avoid supplements, probiotics, prebiotics, yogurt and antibiotics. Alcohol prohibited. PA: Instructed to maintain regular training, logged intensity, duration and distance of aerobic sessions.	Stool samples collected in freeze-dried tubes containing a freeze-dried solution. Samples immediately frozen at −80 °C for analysis.	16S rRNA gene amplicon sequencing targeting V4, SCFA not measured.	Zhejiang Provincial Natural Science Foundation (Grant Number: TGY24H180038); Zhejiang Medical and Health Science and Technology (Grant Numbers: 2022KY258 and 2024KY198); Hangzhou Health, Science and Technology Plan (Grant Numbers: A20210057 and A20230654); Hangzhou Biomedical and Health Industry Development Support Science and Technology (Grant Number: 2021WJCY052).
Önes et al.,[32] Turkey, RCT	*N* = 21 females, professional soccer players, Tier 3, age: Int: 24.42 ± 2.52; control: 22.14 ± 3.61 yr, $\dot{V}O_{2max}$ measured, not reported.	Kefir from a single, commercially available, standardised brand. The product, with quality control measures, provided 140 kcal, 7.3 g fat, 4.2 g CHO, 9.2 g protein/250 mL. 200 mL/d, after training or any time on rest days, control: continue daily diet routine, duration: 28 d.	DC[a]: Nutritional status assessed via 3-d food records (single, double, off days). Dietitian-guided record-keeping, analysed via Nutrition Information Systems. Players instructed to maintain usual diet. Nutritional composition not reported. PA[a]: Players' actual physical activity not recorded.	Stool collection method not reported.	16S rRNA gene amplicon sequencing targeting V3–V4, α-Diversity, Shannon index and Chao1, SCFA not measured.	Supported by Acibadem University Scientific Research Projects Coordination Unit (ABAPKO) (Grant: TDK-2023-91).
Nutrient						
Morita et al.,[39] Japan, RCT	*N* = 31 (males), healthy, non-athletes, exercise regularly (1–2 × wk), Tier 0–1, age: Pla: 36.3 ± 9.6; FL: 33.9 ± 10.0; αCD: 34.5 ± 10.9 yr, $\dot{V}O_{2max}$ (mL/kg/min): Pla: 46.40 ± 6.45; FL: 43.20 ± 6.83; αCD: 45.67 ± 8.66.	FL: 200 mg/d, NIPPN flaxseed lignans, ~40 % (w/w) SDG and ~40 % (w/w) αCD, αCD: 200 mg/d, Dexypearl-α (Ensuiko Sugar Refining, Tokyo, Japan), αCD (> 98 %), 3 × tablets/d, ingested with water at the same time of day, Pla: maltitol, duration: 9 wk.	DC[a]: Instructed to maintain normal diet and avoid prohibited foods, including performance enhancing foods/beverages, pharmaceuticals or quasi-drugs for recovery/fatigue/strength and dietary supplements. Nutritional composition of diets not reported. PA[a]: Instructed to maintain their normal physical activity, defined as 1–2 sessions (≥ 30 min each) per wk of exercise ≥ 5 METs, excluding resistance training. Physical activity was not monitored or reported in this study.	Participants collected stool samples at home using Sarstedt containers within 5 d prior to each visit, froze them at approximately −30 °C and transported them to the clinic.	16S rRNA gene amplicon sequencing targeting V4, complemented by species-specific qPCR for *Bacteroides uniformis* quantification, α-Diversity, Shannon, Chao1, Phylogenetic distance, SCFA not measured.	Partially supported by JSPS KAKENHI (22H03541), JST ERATO (JPMJER1902), AMED-CREST (JP22gm1010009), the Food Science Institute Foundation and Asahi Quality & Innovations Ltd. Some authors are employees of Asahi Quality & Innovations Ltd. and filed patents related to antifatigue and strength-enhancing substances using *B. uniformis*, its derivatives, FL or αCD. One author is the founder and CEO of Metagen Inc., which had no role in the study's interpretation, writing or publication.

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Table 3 Continued

Author, country, study design	Population characteristics	Intervention protocol (vs. placebo or control)	Dietary control (DC) / Physical activity (PA)	Faecal collection technique	Faecal analysis technique[b]	Funding source and conflict(s) of interest
Onishi et al.,[33] Japan, RCT	*N* = 81 (males), healthy, non-athletes, with regular exercise habits, Tier 0–1, age: median [first quartile–third quartile] Pla: 40.00 [29.50–44.00]; αCD: 40.00 [30.50–44.50] yr, $\dot{V}O_{2max}$ (mL/kg/min): Pla: 44.05 [39.63–50.10]; αCD: 42.30 [38.15–51.60].	αCD: 4× tablets d, 1 g/d, Pla: maltitol, duration: 8 wk.	DC[a]: Participants received a prescribed dinner the day before and a prescribed breakfast on the day of the clinic visit; otherwise asked to maintain their normal diet. Daily meal intake recorded, showing no change during the supplementation period for both groups. [10] PA[a]: Recorded daily exercise habits during supplementation period. [NS] differences in exercise frequency between groups.	Participants collected stool samples at home in faecal collection containers within 5 d before each visit, froze them at ~ −30 °C and delivered them to the clinic; samples were thawed at room temperature before analysis.	16S rRNA gene amplicon sequencing targeting V4, complemented by species-specific qPCR for *Bacteroides uniformis* quantification, SCFA not measured.	Funded by Asahi Quality & Innovations, Ltd. Some authors are employees of Asahi Quality & Innovations Ltd. and have filed a patent for sport performance-improving agents containing αCD.
Diet						
Furber et al.,[27] UK, RCT	*N* = 16 (males) highly trained endurance runners, Tier 3, age: HPD: 25 ± 3.6; HCD: 27 ± 5.0 yr, $\dot{V}O_{2max}$ (mL/kg/min): HPD: 63.1 ± 4.8; HCD: 65.3 ± 6.4.	Phases: 7-d habitual diet → 7-d isocaloric HPD (protein 40/CHO 30/fat 30 %EI) or HCD (CHO 60/protein 10/30 % Fat EI) → 7-d habitual diet, duration: 22 d.	DC: Rigorously controlled diets (specific macronutrient ratios) were prescribed to match energy expenditure, supplemented by a daily 500 kcal meal. Participant compliance monitored using a 3-d food record for each diet phase, which was analysed. PA: Training volume controlled; instructed to maintain the same weekly program, replicating sessions on the same day and time, logging all sessions with the provided GPS watch, automatically uploaded to Garmin Connect.	Participants collected the first stool of the day, which was transferred to −80 °C storage within 2 h of passage and stored there until analysis.	16S rRNA gene amplicon sequencing targeting V4, ITS1–ITS2 amplicon sequencing for fungal taxa and viral metagenomics, Fisher-alpha diversity, SCFA not measured.	The authors declare a conflict of interest. Two authors are former employees of GlaxoSmithKline and two have received funding for gut microbiota research. Previous employers and funders had no role in the study's design, participant recruitment, results or conclusions.
Mancin et al.,[29] Italy, RCT	*N* = 16 (males), semi-professional soccer players, Tier 2, age: KD: 25.5 ± 2.5; WD: 25.5 ± 3.1 yr.	Diets: KEMEPHY ketogenic Mediterranean diet (KD) or Western diet (WD), both isocaloric and isoproteic (1.8 g/kg BW/d protein), KD: <30 g/d carbs (<10 %EI), 25–30 %EI protein, fat *ad libitum*, 3 herbal extracts, nutritional counselling, meal plans, WD: 50–55 %EI carbs, 30 %EI protein, 20–25 %EI fats (<10% SF, <300 mg cholesterol), whole cereals, legumes, moderate wine, Compliance: Weighed food records, urine ketone testing, duration: 30 d.	DC: Participants received nutritional counselling, meal plans and recipes and were provided with specific items like ready-to-eat ketogenic products, herbal extracts and MCT oil (KEMEPHY group) and pre-sleep protein (both groups). Dietary adherence was rigorously monitored using food diaries, ketone testing (KEMEPHY group) and regular follow-ups with a nutritionist. PA: Players asked to keep their normal training schedule (8 h of training/wk), managed by team structure and explicit instructions to players.	Faecal samples (100–150 mg) collected using sterile swab tubes with preservative buffer on the morning of day 0 and day 30, sent to the lab within 2 d and stored at −20 °C until DNA extraction.	16S rRNA gene amplicon sequencing targeting V3–V4, α-diversity, OTU and Shannon's ENS, SCFA not measured.	Funded by Department of Biomedical Sciences, University of Padua Institutional Grant. One author received, and another is supported by a research grant from Gianluca Mech S.p.A., a company specialising in herbal products and dietary keto-foods, which had no role in study design, data collection, analysis, interpretation or writing. Two authors were employed by BMR Genomics srl.

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Table 3 Continued

Author, country, study design	Population characteristics	Intervention protocol (vs. placebo or control)	Dietary control (DC) / Physical activity (PA)	Faecal collection technique	Faecal analysis technique[b]	Funding source and conflict(s) of interest
Murtaza et al.,[31] Australia, RCT	*N* = 29 (males), highly competitive race walkers, Tier 4, age: HCHO 25.4 ± 4.0; PCHO: 27.4 ± 4.6; LCHO 28.3 ± 3.5 yr, $\dot{V}O_2$ peak (mL/kg/min): HCHO: 61.6 ± 6.8; PCHO: 64.6 ± 5.3; LCHO: 66.3 ± 4.8.	3-wk training camp, diets assigned based on performance beliefs, HCHO: 60 %EI CHO (~8.5 g/kg BM/d), 16 %EI protein (~2.1 g/kg BM/d), 20 %EI fat, PCHO: same macronutrient composition as HCHO but periodised across d/sessions, LCHF: 78 %EI fat, 17 %EI protein (~2.2 g/kg BM/d), < 50 g/d CHO (~3.5 %EI).	DC: All food and fluids were provided and prepared by professional staff. Individualised meal plans were served in a controlled setting, with intake weighed, monitored and cross-checked daily for compliance. PA: 3-wk intensified training block with race walking, resistance and cross-training (running, cycling, swimming). 6 mandatory sessions under standardised conditions with external monitoring; additional sessions based on athlete preference, logged.	Stool samples were collected from athletes at start and end of training-diet intervention using the OMNIgene stool collection and preservative kit.	16S rRNA gene amplicon sequencing targeting V6–V8, α-Diversity, Shannon and Simpson, SCFA not measured.	This research was supported by CRN AESS, an Australian Catholic University Research Fund Program Grant (No. 201300800), and the Australian Institute of Sport's High-Performance Sport Research Fund. One of the authors is supported by a University of Queensland International Postgraduate Fellowship.
Exercise						
Zhong et al.,[26] China, RCT	*N* = 14 (females), physically active elderly, Tier 1, age: Int: 66.38 ± 4.07; control: 68.50 ± 3.78 yr, Physical Activity Scale for Elderly (PASE) scores: Int: 117.11 ± 24.49; control: 105.36 ± 49.90.	Int: exercise program: 4× wk, 60 min, including warm-up (10 min), aerobic exercise (20 min), resistance exercise (25 min) and cool down (5 min), control: maintain daily life, 8 wk.	DC[a]: Diet not monitored. PA[a]: Maintain daily life activities along with intervention. No specific methods for monitoring or assessing physical activity during intervention period were described.	Stool samples collected before baseline test and after intervention, no further details on collection or storage method provided.	16S rRNA gene amplicon sequencing targeting V3–V4, α-diversity, Sobs, Chao, Ace, Shannon, Simpson, SCFA not measured.	Supported by Hangzhou Philosophy and Social Science Project (Z20JC074), China; The Fundamental Research Funds for the Central Universities.

Abbreviations: ax, assessment; BM, body mass; CFU, colony forming units; CHO, carbohydrate; DGGE, denaturing gradient gel electrophoresis; EI, energy intake; GC–MS, gas chromatography–mass spectrometry; HCD, high carbohydrate diet; HCHO, high carbohydrate; HPD, high protein diet; HPLC, high-performance liquid chromatography; ITS1, internal transcribed spacer 1; ITS2, internal transcribed spacer 2; LC_MS, liquid chromatography–mass spectrometry; LCHF, low carbohydrate, high fat; LCHO, low carbohydrate; MMA, mixed martial arts; no., number; [NS], not significant; PCHO, periodised carbohydrate; Pla, placebo; QPCR, quantitative polymerase chain reaction; RCT, randomised control trial; RXT, randomised crossover trial; SDG, secoisolariciresinol diglucoside; UPLC–MS/MC, ultra-performance liquid chromatography–tandem mass spectrometry; αCD, α-cyclodextrin. Mean age is ± SD unless otherwise stated.

[a]Not in accordance with best practice guidelines and recommendations in exercise gastroenterology research- did not follow minimal confounder control for diet and/or physical activity Costa et al.[73]

[b]Faecal analysis technique- bacterial taxa, α-diversity and short-chain fatty acids.

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Table 4 Systematic literature review search results and study characteristics of included studies investigating the impact of nutritional supplement-, dietary- and exercise-associated changes to the gut microbiota and their impact on athletic performance

Author, study design	*N*_Intervention protocol	Microbiota changes (Δ)	SCFA changes (Δ)	Performance changes (Δ)	Key takeaway
Probiotic and postbiotic studies—single strain					
Gross et al.,[22] RXT	*N* = 7 (3 males and 4 females), physically active	NS changes in α-diversity (Shannon entropy) between baseline, Pla, washout and Int ($p > 0.05$).	Not reported	NS difference in treadmill TTE between Int and Pla	*Veillonella* supplementation did not significantly alter gut microbiota diversity or improve exercise performance, though individual microbiota changes were noted.
	Probiotic vs. placebo	β-diversity (Bray Curtis) showed no group-level changes, some individual variability observed.		Mean TTE Δ	
	Veillonella atypica FB0054, 14 d.	No changes in specific taxa or microbial functions after intervention.		Int: 13.29 ± 100.13 s, ($p = 0.738$)	
				Pla: 61.14 ± 72.04 s ($p = 0.066$)	
				No correlation between changes in β-diversity and TTE ($r = 0.09$).	
Huang et al.,[23] RCT	*N* = 20 males, triathletes	α-Diversity (Shannon index) ↓ with Int vs. Pla (Int 4.4, Pla 4.7; $p < 0.05$)	Measured only at post-intervention time points.	Treadmill TTE was significantly greater in Int compared to Pla (Int: 1,679 Pla: 1,083 s, $p < 0.05$)	Post-intervention, probiotic group showed higher beneficial genera, SCFAs, and TTE vs. Pla; no baseline data reported, limiting interpretation.
	Probiotic vs. placebo	No baseline data reported.	Significant ↑ observed in acetic (4.7 vs. 3.8 ng/mL), propionic (1.18 vs. 0.5 ng/mL), and butyric acid (0.5 vs. 0.3 ng/mL) in the Int compared to Pla ($p < 0.05$).	NS difference observed in $\dot{V}O_{2max}$ between groups (Int: 59.2, Pla: 57.2 mL/kg/min)	
	Lactiplantibacillus plantarum PS128, 4 wk.	NS difference in phylum-level RA between groups.	NS differences for decanoic, heptanoic, hexanoic, isobutyric, isovaleric, octanoic and valeric acids.	No correlation analysis between performance and microbiota reported.	
		Significant ↓ in RA of multiple genera in Int vs. Pla (e.g., *Anaerotruncus*, *Caproiciproducens*, *Coprobacillus*, *Desulfovibrio*, *Dielma*, Family_XIII_UCG_001, *Holdemani*, *Oxalobacter*) ($p < 0.05$).			
		Significant ↑ in RA of beneficial genera (*Akkermansia*, *Bifidobacterium*, *Butyricimonas*, *Lactobacillus*) in Int vs. Pla ($p < 0.05$).			
Lee et al.,[40] RCT	*N* = 53 (26 males and 27 females), physically active	NS change in α-diversity (Shannon index) within or between groups	Acetate ↑ post-intervention in both TWK10 and TWK10-hk ($p < 0.05$); NS between group differences.	Postintervention treadmill, TTE significantly ↑ in both TWK10 (17.55 ± 3.98 min) and TWK10-hk (16.72 ± 5.91 min) vs. control (12.23 ± 2.08 min) ($p < 0.001$)	Both TWK10 and TWK10-hk increased endurance and acetate (no between-group difference).
	Probiotic vs. heat-killed probiotic vs. placebo	β-Diversity (UniFrac): Post-intervention differences observed between TWK10-hk vs. control ($p = 0.036$) and TWK10 vs. TWK10-hk ($p < 0.0001$); no difference between TWK10 vs. control.	Propionate ↑ trend only in TWK10-hk ($p = 0.0857$)	Within-group TTE: ↑ 1.38-fold (TWK10) and 1.33-fold (TWK10-hk) (both $p < 0.001$)	TWK10-hk altered β-diversity and showed a trend for propionate ↑.

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Table 4 Continued

Author, study design	*N*_Intervention protocol	Microbiota changes (Δ)	SCFA changes (Δ)	Performance changes (Δ)	Key takeaway
	TWK10: viable *Lactiplantibacillus plantarum*	Within-group shifts significant in TWK10-hk and control only.	Butyrate ↑ trend in TWK10 ($p = 0.0744$)	TTE positively correlated with Coriobacteriaceae family (TWK10; $r = \sim 0.5$, $p < 0.01$) and Veillonellaceae family (TWK10-hk; $r = 0.65$, $p < 0.01$); negative correlation observed in TWK10-hk with *Eubacterium coprostanoligenes*, Erysipelatoclostridiaceae and Lachnospiraceae (correlation coefficient represented on heatmap, difficult to extract data, $p < 0.05$).	TWK10 had within-group microbiota shifts and a trend for butyrate ↑.
	TWK10-hk: heat-killed *Lactiplantibacillus plantarum*, 6 wk.	No between-group differences in phylum-, family- or genus-level RA.			Performance correlated with specific taxa.
		Within-group changes (post vs. baseline): TWK10-hk ↑ *Pseudomonadota* (phylum, $p = 0.030$), ↑ *Lactococcus*, *Escherichia-Shigella* (genus, $p < 0.05$), ↓ *Peptostreptococcaceae* (family, $p = 0.030$), ↑ trend *Enterobacteriaceae* (family, $p = 0.072$).			
		TWK10 ↑ *Lactobacillaceae* (family, $p = 0.039$), ↓ *Lachnospira* (genus, $p = 0.022$), ↓ trend *Faecalibacterium* (genus, $p = 0.077$)			
Li et al.,[28] RCT	$N = 16$ males, National top-level cross-country skiers	^{NS} between group differences in α-diversity (Shannon index) or β-diversity (data not shown).	Acetic acid	Treadmill, $\dot{V}O_{2max}$ (mL/kg/min): ↑ in both groups ($p < 0.01$); post-intervention Int > control ($p < 0.02$)	*B. animalis* abundance ↑ in Int (40-fold vs. 2-fold); acetic acid ↑ significantly vs. control ($p < 0.05$).
	Probiotic vs. control	Within group: RA of *Bifidobacterium animalis* ↑ ~40-fold in intervention vs. ~2-fold in control.	↑ 4.5 fold (Int), ↑ 3.4 fold (control); Int > control ($p < 0.05$)	Knee strength (Nm/kg)	Improvements in $\dot{V}O_{2max}$ and knee extensor strength observed in the intervention group, with positive correlations between *B. animalis* and multiple SCFAs.
	Yogurt with *Bifidobacterium animalis* subsp. *lactis* BL-99, 8 wk.	Statistical significance between groups for this change not reported.	Propanoic, butyric, valeric acids: ↑ in both groups (2–5 fold); ^{NS} difference between groups	60 °/s extensors: ↑ in Int ($p < 0.01$), ^{NS} in control; Int > control at post ($p < 0.05$)	Improvements in $\dot{V}O_{2max}$ and knee extensor strength observed in the intervention group, with positive correlations between *B. animalis* and multiple SCFAs.
			Positive correlations ($r > 0.5$, $p < 0.05$) between *Bifidobacterium animalis* and acetic, propanoic, butyric and valeric acids	60 °/s flexors: ↑ in Int ($p < 0.05$); ^{NS} in control	
				180 °/s extensors: ↑ in both groups ($p < 0.05$)	
				180 °/s flexors: ^{NS} in both groups	

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Table 4 Continued

Author, study design	*N*_Intervention protocol	Microbiota changes (Δ)	SCFA changes (Δ)	Performance changes (Δ)	Key takeaway
Lin et al.,[25] RCT	*N* = 21 (14 males and 7 females), well-trained middle and long-distance runners	RA	Not measured	12 min Cooper's test (running distance by minute):	Although there were [NS] between-group differences in running distance at any time point during the 12-min Cooper's test, the Int demonstrated significantly greater pre-to-post improvements at 6, 9 and 12 min compared to Pla (all $p < 0.01$).
	Probiotic vs. placebo	Phylum (post only):		No between-group differences at any time point ($p > 0.05$)	
	Bifidobacterium longum subsp. *longum*, 5 wk.	↑ *Actinomycetota* and *Bacillota*		Δ Pre- to postintervention	
		↓ *Pseudomonadota* in Int vs. Pla (no *p*-values reported)		6th minute: Int 72 ± 14 m Pla 4 ± 9 m ($p = 0.0014$)	
		Genus: ↑ *Bifidobacterium* in Int vs. Pla ($p = 0.0027$); ninefold ↑ in *Lactobacillus* count (no *p*-value provided)		9th minute: Int 116 ± 17 m Pla −27 ± 21 m ($p = 0.0001$)	
		Species: *Bifidobacterium longum* subsp. *Longum* ↑ 8.63-fold in Int to 0.95 % ($p = 0.0178$); Pla post-intervention was 0.11 %		12th minute: Int 105 ± 16 m Pla −56 ± 29 m ($p = 0.0001$)	
		[NS] differences in the abundance of common and pathogenic strains between groups		Within group (post vs. pre):	
				Int: ↑ at 3rd ($p = 0.0051$), ↑ at 6th ($p = 0.0004$), ↑ at 9th ($p < 0.0001$) and ↑ at 12th min ($p < 0.0001$)	
				Pla: ↑ at 3rd min ($p = 0.0051$)	
McDermott et al.,[30] RCT	*N* = 28 (13 males and 15 females), runners	RA, baseline	Not measured	Treadmill, TTE (s):	Despite recovery of *Lactobacillus helveticus* Lafti L10 in over half of Int, no performance benefit was observed.
	Probiotic vs. placebo	No detectable levels of *Lactobacillus helveticus* Lafti L10 in either group.		Int: ↓ (1,655 ± 230–1,547 ± 215; $p = 0.23$)	Treadmill TTE decreased in Int and increased in Pla, with post-intervention TTE significantly greater in Pla vs. Int ($p = 0.01$).
	Lactobacillus helveticus Lafti L10, 6 wk.	Post intervention:		Pla: ↑ (1,344 ± 188–1,565 ± 219; $p = 0.01$)	SCFAs were not measured, and no statistical comparison of microbiota changes between groups was reported.
		Lactobacillus helveticus Lafti L10 detected in 8/14 participants in Int (median: $10^{4.37}$ bacteria/mL); not detected in Pla		Post intervention:	
		No statistical analysis of between-group differences reported		Int < Pla ($p = 0.01$)	

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Table 4 Continued

Author, study design	*N*_Intervention protocol	Microbiota changes (Δ)	SCFA changes (Δ)	Performance changes (Δ)	Key takeaway
West et al.,[37] RCT	*N* = 88 (64 males and 35 females recruited), competitive cyclists	[NS] differences in diversity (16S rRNA, data not shown)	Not measured	Incremental cycling ergometer test ($\dot{V}O_{2max}$): No substantial difference between groups (data not shown). Training patterns unaffected.	[NS] changes in microbiota diversity or $\dot{V}O_{2max}$ were observed between groups following *Limosilactobacillus fermentum* supplementation.
	Probiotic vs. placebo	No statistical testing performed; only raw bacterial count data reported by sex.			Bacterial counts were reported descriptively (by sex), with no statistical analysis of between-group changes.
	Limosilactobacillus fermentum VRI-003 PCC, 11 wk.	Total bacteria:			SCFAs were not measured.
		Males: ↓ in both Int and Pla (0.5×10^{10})			
		Females: ↑ in Int (0.7×10^{10}), ↓ in Pla (1.0×10^{10})			
		C. coccoides			
		Males: ↓ in both groups			
		Females: ↔ in Int, ↓ in Pla			
		E. coli			
		↑ in both sexes, greater increase in Int			
		Bifibacteria			
		Males: ↓ in both groups			
		Females: ↑ in Int, ↓ in Pla			
		Bacteroides			
		Males: ↑ in both groups			
		Females: ↑ in Int, ↓ in Pla			
		Lactobacillus			
		Males and females: ↑ in Int			
		Males: ↓ in Pla Females: ↑ in Pla			

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Table 4 Continued

Author, study design	*N*_Intervention protocol	Microbiota changes (Δ)	SCFA changes (Δ)	Performance changes (Δ)	Key takeaway
Wu et al.,[38] RCT	$N = 105$ (75 males and 30 females), physically active	[NS] change in α-diversity (Shannon index and Chao1) within or between groups	[NS] differences in acetic, propionic, butyric, isobutyric, valeric or isovaleric acid within or between groups	CMJ[a]	L-PS23 and HT-PS23 improved post-EIMD performance recovery (CMJ, RFD, WAnT); post-intervention microbiota differences observed (e.g., ↑ *Lacticaseibacillus*, ↓ *Prevotella*) but baseline data not reported; no SCFA changes.
	Probiotic vs. heat-treated probiotic vs. placebo	β-Diversity, post only: L-PS23 significantly different from Pla ($p = 0.006$); HT-PS23: not different from Pla ($p = 0.868$)		Post-EIMD Recovery:	
	Lacticaseibacillus paracasei PS23 (L-PS23)	No baseline data reported.		All groups showed reduced RFD and relative force peak post EIMD (3, 24, 48 h) ($p < 0.05$)	
	HT-PS23, 6 wk.	RA (post-intervention, log10)		L-PS23 and HT-PS23 less reduction in RFD and relative force peak vs. Pla at multiple time points ($p < 0.05$ to $p < 0.0001$)	
		Genus: L-PS23 > *Pla* for *Lacticaseibacillus*, *Streptococcus*, *Blauti*, *Lactobacillus*		HT-PS23 showed less reduction in CMJ vs. Pla at 24 and 48 h ($p < 0.05$)	
				Time effects significant for RFD, relative peak force and CMJ ($p < 0.0001$)	
		L-PS23 < Pla for *Prevotella* ($p < 0.05$)		Isotonic Muscle Strength (IMTP)	
		HT-PS23 > Pla for *Lacticaseibacillus* and *Collinsella* ($p < 0.05$)		L-PS23 and HT-PS23 showed significantly less reduction in relative force peak vs. Pla at various time points post-EIMD ($p < 0.05$)	
				RFD:	
				L-PS23 and HT-PS23 had less loss of peak RFD at all post-EIMD time points vs. Pla ($p < 0.05$)	
				Wingate anaerobic test (WAnT):	
				L-PS23 and HT-PS23 less reduction of relative mean and peak power vs. Pla at 3, 24 and 48 h post-EIMD ($p < 0.05$)	
				Fatigue index:	
				All groups increased fatigue index post-EIMD ($p < 0.0001$)	
				HT-PS23 and L-PS23 had smaller increases vs. Pla at 24 and 48 h ($p < 0.05$).	
				Correlation: Data difficult to extract (heat map).	
				Positive correlations: *Lacticaseibacillus*, *Streptococcus*, *Blautia*, *Lactobacillus*: with improved performance in CMJ, IMTP, and WAnT, weak to moderate ($r = 0.1–0.3$).	
				Negative correlations: *Prevotella* with exercise performance including RFD % 24 h ($r = -\sim 0.3$), Wingate relative peak power (%) 48 h and Wingate Fatigue Index (%) 24 h ($r = \sim -0.2$)	

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Table 4 Continued

Author, study design	*N*_Intervention protocol	Microbiota changes (Δ)	SCFA changes (Δ)	Performance changes (Δ)	Key takeaway
Probiotic studies—multi-strain					
Przewłócka et al.[35] Study 1 RCT	*N* = 23 males, well-trained MMA athletes	This was assessed in Przewłócka et al.[45]	This was assessed in Przewłócka et al.[45]	Cycle ergometer, supramaximal sprints, triple WAnT	↑ W_{tot} and MP within intervention group (p = 0.04); no between-group differences in performance.
	Probiotic vs. control			↑ within-group W_{tot} (J/kg) and MP (W/kg) in Int; no between-group differences in WAnT performance outcomes (i.e., W_{tot}, P_{max}, MP or FI)	
	Bifidobacterium lactis W51, *Levilactobacillus brevis* W63, *Lactobacillus acidophilus* W22, *Bifidobacterium bifidum* W23 and *Lactococcus lactis* W58				
	+ 5 mL Vit D3, 4 wk.				
Przewłócka et al.,[34] Study 2 RCT	*N* = 23 males, well-trained MMA athletes	NS difference between groups in α-diversity (Inverted Simpson, p = 0.086)	NS differences between groups for acetic, propionic, butyric and valeric acid	Graded cycle ergometry test	NS changes in α- or between-group β-diversity.
	Probiotic vs. control	β-Diversity (Bray–Curtis), significant within group Δ in Int (p = 0.0005); NS in control (p = 0.145)	Post-intervention: ↓ propionate in both groups, statistically significant in control (p = 0.004)	NS within or between group differences in $\dot{V}O_{2max}$ or MAP (W)	Int altered specific gut taxa (↑ Negativicutes, Faecalibacterium; ↓ Firmicutes, Lachnospiraceae).
	Bifidobacterium lactis W51, *Levilactobacillus brevis* W63, *Lactobacillus acidophilus* W22, *Bifidobacterium bifidum* W23 and *Lactococcus lactis* W58	No between-group differences reported	Int propionate tended to be lower than control (p = 0.061)	TTE (s) significantly improved in Int (p = 0.023) but not in control (p = 0.685); between-group difference at post-intervention not reported.	No group differences in SCFAs; propionate decreased in both groups.

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Table 4 Continued

Author, study design	*N*_Intervention protocol	Microbiota changes (Δ)	SCFA changes (Δ)	Performance changes (Δ)	Key takeaway
	+ 5 mL Vit D3, 4 wk.	Total abundance			No effect on $\dot{V}O_{2max}$ or power; TTE improved only in Int (between-group difference not reported).
		Between group (post-intervention, Int vs. control)			
		Class: ↑ Negativicutes (Est pairwise = 2.23, $p = 0.032$), ↓ Firmicutes (Est pairwise = −0.93, $p = 0.038$)			
		Family: ↓ Lachnospiraceae (Est pairwise = −1.27, $p <$ 0.001), ↓ Peptostreptococcaceae (Est pairwise = −2.69, $p = 0.004$), ↓ Lactobacillaceae (Est pairwise = −2.04, $p = 0.018$)			
		Genus: ↑ *Faecalibacterium* (Est pairwise = 1.21, $p = 0.013$), ↑ *Prevotella* (Est pairwise = −1.01, $p = 0.002$), ↓ *Collinsella* (Est pairwise = −0.93, $p = 0.038$), ↓ *Bacteroides* (Est pairwise = −0.041, missing *p*-value)			
		Species: ↑ *Bacteroides fluxus* (Est pairwise = 2.02, $p = 0.002$), ↑ *Roseburia inulinivorans* (Est pairwise = 1.40, $p = 0.005$)			
		Within group changes (Int)			
		Class: ↑ Negativicutes (Est = 1.98, $p = 0.006$), ↓ Firmicutes (Est = −1.68, $p < 0.001$).			
		Family: ↓ Lachnospiraceae bacterium (Est = −1.55, $p < 0.001$), ↓ Peptostreptococcaceae bacterium (Est = −1.86, $p = 0.005$), genus: Int: ↑ *Bacteroides* (Est = 0.259, $p < 0.001$), ↑ *Faecalibacterium* (Est = 1.03, $p = 0.002$), ↑ *Prevotella* (Est = 3.62, $p < 0.001$), ↓ *Collinsella* (Est = −1.28, $p = 0\ .003$)			
		Species: Int: ↑ *Bacteroides fluxus* (Est = 2.11, $p <$ 0.001), ↑ *Roseburia inulinivorans* (Est = 0.74, $p = 0.030$)			
		Within-group changes (control)			
		Class: ↓ Firmicutes (Est = −0.75, $p = 0.021$).			

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Author, study design	*N*_Intervention protocol	Microbiota changes (Δ)	SCFA changes (Δ)	Performance changes (Δ)	Key takeaway
Wang et al.,[36] RCT	*N* = 19 (15 males and 4 females), active amateur marathon runners	RA (Phylum)	Not measured	Cooper's test	Intervention increased abundance of several genera (e.g., *Lacticaseibacillus*, *Olsenella*) and decreased others (*Cloacibacillus*) vs. placebo.
	Probiotic vs. placebo	*Bacteroidota*, *Bacillota*, *Pseudomonadota* and *Actinomycetota* more abundant in Int vs. Pla (qualitative, no *p*-values shown).		Total distance (km) improved within Int ($p < 0.05$), but not in Pla; [NS] between-group difference ($p = 0.323$)	*B. longum* increased ([NS]).
	Lactobacillus acidophilus and *Bifidobacterium longum*, 5 wk.	Genus: ↑ *Lacticaseibacillus* ($p = 0.001$), *Olsenella*, *Weissella*, *Anaerostipes* ($p < 0.05$) in Int vs. Pla; ↓ *Cloacibacillus* and *Alphaproteobacteria*_unclassified in Int vs. Pla ($p < 0.01$).			Running distance improved within intervention only; no between-group difference.
		Species: *Bifidobacterium longum* increased from pre- to post-intervention in Int ([NS])			
Önes et al.,[32] RCT	*N* = 21 females, professional soccer players	[NS] between-group differences in α-diversity (Shannon index, Chao1)	Not measured	*30–15 intermittent fitness test*	No change in α-diversity; β-diversity differed between groups.
	Probiotic vs. control	β-Diversity:		$\dot{V}O_{2max}$ and finishing speed data not reported numerically	Kefir ↑ *Akkermansia*, *Bifidobacterium* and *A. muciniphila*.
	Kefir, 28 d.	Significant difference between groups (Bray–Curtis, $p = 0.018$; Jaccard, $p = 0.016$).		Subgroup analysis:	No numerical performance data; exploratory findings linked certain taxa (e.g., *F. prausnitzii*, *P. copri*) with higher fitness.
		[NS] within group changes in Int		High performers: ↑ *Faecalibacterium* and *P. copri*	
		RA		Low performers: ↑ *Dorea formicigenerans* and *Oxalobacter formigenes*	
		Phylum: dominant: *Bacillota* and *Bacteroidota* in all groups.		No statistical comparisons between groups reported	
		Int: ↓ *Pseudomonadota*, ↑ *Verrucomicrobiota* and *Euryarchaeota* (no *p*-values reported)			
		Genus: dominant: *Prevotella*, *Bacteroides* and *Faecalibacterium* in all groups.			
		Int: ↓ *Bacteroides* and *Faecalibacterium*			
		↑ *Akkermansia* and *Bifidobacterium* (no *p*-values reported)			
		Species:			
		Int: ↑ *A. muciniphila* and *Roseburia faecis* (no *p*-values reported)			
Nutrient					

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Table 4 Continued

Author, study design	*N*_Intervention protocol	Microbiota changes (Δ)	SCFA changes (Δ)	Performance changes (Δ)	Key takeaway
Morita et al.,[39] RCT	*N* = 31 males, physically active	α-Diversity and β-diversity: [NS] difference between groups.	Not measured	10 km TT exercise bike	No changes in α- or β-diversity.
	Nutrient vs. nutrient vs. placebo	RA		FL or Pla: NS at any time point	*B. uniformis* increased within FL and αCD groups over time, but between-group differences were [NS].
	FL	Between groups: Non-significant increase in *B. uniformis* in FL and αCD vs. Pla at wks 4 and 8		αCD: ↓ time to complete at 4 wk ($p = 0.004$) and 8 wk ($p = 0.014$) vs. baseline	αCD group showed improved 10 km TT at 4 and 8 wk compared to baseline; significantly faster than Pla at 8 wk.
	αCD, 9 wk.	Within groups:		Between groups at 8 wk: αCD fasters than Pla ($p = 0.010$)	
		FL: ↑ *B. uniformis* at wks 4 ($p = 0.038$) and 8 ($p = 0.011$)			
		αCD: ↑ *B. uniformis* at wk 8 ($p = 0.036$)			
		Pla: [NS] ↑ *B. uniformis* at wks 4 and 8			
		Δ *B. uniformis abundance*, (0–8 wk): median copies/g:			
		αCD: 3.99×10^{10}			
		FL: 1.46×10^{10}			
		Pla: 3.74×10^{8}			
		[NS] between groups			
Onishi et al.,[33] RCT	*N* = 81 males, physically active	[NS] difference between groups in *B. uniformis* abundance post-intervention.	Not measured	10 km TT (s) exercise bike	No between-group difference in *B. uniformis* abundance.
	Nutrient vs. placebo	Both αCD and Pla showed significant within-group increases over time.		10 km TT completion times improved significantly from baseline in both groups. At 8 wk, αCD group times were significantly faster than Pla ($p = 0.016$).	Significant within-group increases observed in both αCD and Pla
	αCD, 8 wk.			[NS]*changes within or between groups for* $\dot{V}O_{2max}$ or PWC 75 % HR_{max}	10 km TT improved from baseline in both groups; αCD group was significantly faster than Pla at 8 wk, although between-group change over time was not assessed.
					[NS] changes in $\dot{V}O_{2max}$ or PWC 75 % HR_{max}
Diet					
Furber et al.,[27] RCT	*N* = 16 males, highly trained endurance runners	*HPD:* ↓ Fisher-alpha diversity of inducible viruses (IV) ($p = 0.04$), altered viral composition ($R^2 = 0.15–0.16$), ↑ Sk1virus, correlated with ↑	Not measured	*Treadmill*, *10 km TTE* (*s*)	HPD impaired performance and altered viral composition (↑ Sk1virus, ↓ *Bifidobacterium* spp.); HCD mostly improved performance, linked to microbial stability. All effects reversed after washout.
	Diet vs. diet vs. diet	*Leuconostoc* and ↓ *Bifidobacterium* spp.		HPD: ↓ 23.3 % ($p < 0.001$); linked with greater FVP shifts, performance returned to baseline after washout	
	Habitual diet vs. HPD vs. *HCD*, 22 d.	*HCD:* Bacterial shifts included		HCD: ↑ 6.5 % ($p = 0.05$); linked with microbial stability and subject-specific profiles, performance returned to baseline after washout	
		↑ *Leuconostoc*, *Lactococcus*, *Collinsella*. *Ruminococcus*; ↓ Streptococcus; viral changes included ↑ Cc31virus			

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Table 4 Continued

Author, study design	*N*_Intervention protocol	Microbiota changes (Δ)	SCFA changes (Δ)	Performance changes (Δ)	Key takeaway
Mancin et al.,[29] RCT	*N* = 16 males, semi-professional soccer players	No change in α-diversity (OTU count, Shannon's ENS)	Not measured	*Maximal strength*, *isometric*, *quadriceps*	KDP reduced *Actinobacterota* vs. WD ($p = 0.021$); no changes in α-diversity. Performance improved in both diets with no between-group differences.
	Diet vs. diet	RA		[NS] within or between groups	
	KEMEPHY ketogenic Mediterranean diet (KDP) vs. Western diet, 30 d.	Phylum: *Actinobacterota* ↓ in KDP and ↑ *in WD*; KDP < WD ($p = 0.021$)		*Yo-yo Intermittent Test* (*m*) *and CMJ* (*cm*) ↑ within both groups ($p < 0.001$); [NS] between-group differences	
		Post intervention:			
		KDP: ↑ *Clostridia UCG-014*, *Butyricimonas*, *Odoribacter* and *Ruminococcus*			
		WD: ↑ *Bifidobacterium*, *Butyricicoccus* and *Acidaminococcus*			
		CHO intake negatively correlated with Δ'sof *Odoribacter* genus ($r = -0.59$); fat intake negatively with *Fusicatenibacter* genus ($r = -0.53$)			
Murtaza et al.[31] RCT	*N* = 29 male, highly competitive race walkers	At baseline, participants clustered into gut microbiota enterotypes: 7/29 were *Prevotella* dominant, 20/29 *Bacteroides* dominant and 2 *Bacillota* dominant. These enterotypes remained relatively stable post-intervention.	Not reported.	$\dot{V}O_2$ peak ↑ in all groups[b] ($p < 0.001$):	LCHF diet altered β-diversity and increased *Bacteroides* and *Dorea*, with a reduction in *Faecalibacterium*. These changes were associated with impaired performance.
	Diet vs. diet vs. diet	No changes in α-diversity		Submaximal O_2 cost[b] ↓ in	Enterotypes (mainly *Bacteroides* or *Prevotella*) remained stable and may influence responsiveness to dietary interventions.
	HCHO vs. PCHO vs. LCHO, 3 wk.	HCHO and PCHO: subtle, non-significant microbiota shifts.		HCHO/PCHO; unchanged in LCHF	
		LCHF: Significant shifts in β-diversity (RDA $p = 0.02$; anosim $p = 0.029$).		10-km walk time[b] improved in HCHO (↓ 6.6 %) and PCHO (↓ 5.3 %), not in LCHF (↑ 1.6 %)	
		↓ *Faecalibacterium* spp. ($p = 0.0003$), ↑ *Dorea* spp. ($p = 0.007$) and ↑ *Bacteroides* ($p = 0.002$).		Significant negative correlations observed between *Bacteroides* and fat oxidation and *Dorea* spp. and exercise economy following LCHF. [NS] baseline correlations were reported.	
Exercise					

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Author, study design	*N*_Intervention protocol	Microbiota changes (Δ)	SCFA changes (Δ)	Performance changes (Δ)	Key takeaway
Zhong et al.,[26] RCT	*N* = 14 females, physically active	α-Diversity	Not reported.	NS differences in 2-min step-test, Grip strength, 30-s chair stand test, Timed up and go test.	Exercise increased microbial richness within group, with no between-group differences in α-diversity. Some taxa differed between groups post-intervention.
	Exercise vs. control	NS differences between groups.		Chair sit-and-reach (cm) and single-leg standing with eyes closed (s) significantly improved in Int vs. control	No between-group differences in most functional measures. Chair sit-and-reach and balance improved in Int vs. control; both outcomes negatively correlated with specific taxa.
	4 × wk, 60 min, including warm-up (10 min), aerobic exercise (20 min), resistance exercise (25 min) and cool down (5 min), 8 wk.	Within Int, richness indices (Sobs, Chao, Ace index) increased significantly, but diversity indices (Shannon, Simpson) did not change significantly.		Chair sit-and-reach: negatively correlated with *Betaproteobacteria* ($r = -0.704$, $p = 0.005$).	
		Post intervention, Int showed significantly lower RA (vs. control) of:		Single-leg standing with eyes closed: negatively correlated with *Holdemania* ($r = -0.553$, $p = 0.040$).	
		Class: Betaproteobacteria ($p = 0.008$)		Grip strength: negatively correlated with *Betaproteobacteria* ($r = -0.551$, $p = 0.041$).	
		Order: Burkholderiales ($p = 0.008$)			
		Family: Sutterellaceae ($p = 0.008$), Bacteroidaceae ($p = 0.009$)			
		Genus: *Holdemania* ($p = 0.030$)			
		Anaerostipes ($p = 0.008$)			
		Bacteroides ($p = 0.009$)			
		Bacilli and *Lactobacillales* were higher in Int (both $p = 0.027$)			
		Within-group increases in Int vs. control:			
		Order: Coriobacteriales ($p = 0.012$)			
		Family: Coriobacteriaceae ($p = 0.012$)			
		Genus: *Asaccharobacter* ($p = 0.028$), *Collinsella* ($p = 0.028$), *Fusicatenibacter* ($p = 0.049$)			

Abbreviations: CHO, carbohydrate; EIMD, exercise-induced muscle damage; FI, fatigue index; HCD, high carbohydrate diet; HCHO, high carbohydrate; HPD, high protein diet; IMTP, isometric mid-thigh pull; Int, Intervention; LCHF, low carbohydrate; LCHO, low carbohydrate; MMA, mixed martial arts; MP, mean power; NS, not significant; PCHO, periodised carbohydrate, high fat; Pla, Placebo; Pre, Prebiotic intervention; RA, relative abundance; RCT, Randomised control trial; RFD, rate of force development; RXT, randomised crossover trial; WAnT, Wingate anaerobic test based.

Notes: Δ Indicates the change in mean or median from pre- to post-intervention, unless otherwise specified.

[a]Performance data from Lee et al.[24]

[b]Performance data from Burke et al.[75]

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Table 5 Risk of bias assessment

Reference	Randomisation process	Period and carryover effects	Deviations from intended interventions – effect of assignment of intervention	Deviations from intended interventions – effect of adhering to intervention	Missing outcome data	Measurement of outcome	Selection of the reported result	Overall risk of bias
Furber et al. [27]	🟢	N/A	🟢	🟢	🟢	🟢	🟢	🟢
Gross et al. [22]	🟢	🟢	🟢	🟢	🟢	🟢	🟢	🟢
Huang et al. [23]	🟠	N/A	🟢	🟢	🟢	🟢	🟠	🟠
Lee et al. [40]	🔴	N/A	🟢	🟢	🟢	🟢	🟠	🔴
Li et al. [28]	🟢	N/A	🟢	🟢	🟢	🟢	🟢	🟢
Lin et al. [25]	🟠	N/A	🟢	🟠	🟢	🟢	🟠	🟠
Mancin et al. [29]	🟢	N/A	🟢	🟢	🟢	🟢	🟢	🟢
McDermott et al. [30]	🟢	N/A	🟢	🟢	🟢	🟢	🟢	🟢
Morita et al. [39]	🟢	N/A	🟢	🟢	🟢	🟢	🟢	🟢
Murtaza et al. [31]	🟠	N/A	🟢	🟢	🟢	🟢	🟢	🟠
Önes et al. [32]	🟠	N/A	🔴	🟢	🟢	🔴	🟠	🔴
Onishi et al. [33]	🟢	N/A	🟢	🟢	🟢	🟢	🟢	🟢
Przewłócka et al. [35]	🟢	N/A	🟢	🟢	🟢	🟢	🟢	🟢
Przewłócka et al. [34]	🟢	N/A	🟢	🟢	🟢	🟢	🟢	🟢
Wang et al. [36]	🟢	N/A	🟢	🟠	🟢	🟢	🟢	🟠
West et al. [37]	🟢	N/A	🟢	🟢	🟠	🟢	🟢	🟠
Wu et al. [38]	🟢	N/A	🟢	🟢	🟢	🟢	🟢	🟢
Zhong et al. [26]	🟢	N/A	🟢	🟠	🟢	🟢	🟢	🟠

🟢 Low risk of bias, 🔴 High risk of bias, 🟠 Indicates some concern

dietary products) (**Table 3**).[22,25,27,29,30,33–35,37–40] Most studies included in this review did not adhere to best practice guidelines for exercise-gastroenterology research,[73] which emphasises rigorous methodology and control of confounding variables, especially those related to diet and exercise (**Table 3**).

Dietary monitoring and control

Dietary monitoring and control strategies varied widely, with inconsistent implementation across studies. The majority (*n* = 13) did not provide participants with food; three studies partially provided meals,[25,29,33] and only two implemented fully controlled dietary interventions.[31,36] Most trials monitored dietary intake to some extent rather than supplying all meals. In studies investigating single-strain probiotics or postbiotics, dietary monitoring was often reported, but comprehensive control and detailed reporting were uncommon. For instance, Gross et al.[22] used 2-day food and fluid logs and required participants to replicate intake before each study visit and fast overnight. Despite this dietary control method within the experimental procedures being inappropriate,[73] macronutrient intake was reported, but fibre and FODMAPs were not. Huang et al.[23] asked participants to avoid fermented foods, prebiotics, probiotics and antibiotics and analysed dietary records; yet, detailed results were not reported. Lee et al.[40] required cessation of supplements two weeks prior to intervention and recorded baseline energy intake, but did not monitor fibre or FODMAPs. Similarly, Li et al.[28] reported no between-group differences in energy or macronutrients using 2-day weighed food diaries, but did not include fibre or FODMAP data. Lin et al.[25] employed a dietitian to prescribe a diet and provide the same meal, while restricting probiotic- and antibiotic-containing foods; however, no dietary intake data were reported. West et al.[37] used 4-d food diaries and instructed participants to maintain habitual diets while avoiding probiotic foods, reporting no between-group differences in energy, macronutrients or fibre intake. Wu et al.[38] collected 3-day food diaries with photographic records and reported no significant differences between or within groups, although diets were not otherwise controlled. McDermott et al.[30] standardised pre-trial breakfasts and evaluated dietary habits at baseline and post-intervention, reporting no changes in fibre or diet quality.

Among multi-strain probiotic studies, dietary control and reporting were similarly limited. Przewłócka et al.[34,35] assessed dietary intake using a 3-day interview and food frequency questionnaire and standardised the pre-exercise breakfast, but did not report nutrient data. Wang et al.[36] prescribed an ‘assigned diet’ and restricted supplements, fermented foods and alcohol, yet provided no dietary details. Önes et al.[32] used dietitian-guided 3-day food records across training phases but again did not report nutritional composition. In nutrient-based intervention studies, dietary control varied. Morita et al.[39] asked participants to maintain their usual diets while avoiding functional foods and supplements but did not report dietary composition. Onishi et al.[33] provided participants with a prescribed dinner the day prior and a prescribed breakfast on the day of the clinic visit. Outside of this, participants were instructed to maintain their usual diet. Daily meal intake was recorded, though nutrient composition was not analysed. In contrast, Furber et al.,[27] Murtaza et al.[31] and Mancin et al.[29] implemented tightly controlled diets with detailed dietary analysis, more closely aligning with methodological best practice.[73] In the sole exercise-only study, Zhong et al.[26] did not monitor or report dietary intake, representing a significant limitation given the influence of diet on gut microbiota outcomes. Overall, while many studies incorporated some level of dietary monitoring, such as food diaries, exclusion criteria or standardised meals, few provided detailed nutrient data, especially on fibre and FODMAP intake, which are key confounding nutrients to gut microbiota change. Only a minority provided food directly to participants. These inconsistencies limit the interpretability of intervention effects (e.g., any positive outcome could simply be due to artefact subsequent to the experimental design) and underscore the need for improved methodological rigour, as recommended by Costa et al.[73]

Exercise monitoring and control

Exercise control and reporting were similarly variable (**Table 3**). Most studies provided only general instructions, such as maintain-

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ing a regular lifestyle and avoiding strenuous exercise, with some specifying a restriction period of 1–3 days before testing. However, $n = 3$ studies[23,38,40] did not monitor or record participants' physical activity levels at all. Eleven studies[22,23,25,27,29,32,36–40] lacked detailed exercise data, while five studies[28,30,31,33,35] provided overall comprehensive information. Given the influence of physical activity on gut microbiota, this lack of standardisation limits result interpretation (e.g., any positive outcome could simply be due to an artefact subsequent to the experimental design).

Stool sample collection and storage

Seventeen studies assessed microbial composition in stool samples during the intervention period, reflecting changes in luminal microbiota. However, collection protocols varied considerably, complicating direct comparison across studies (**Table 3**). In probiotic and postbiotic interventions, Gross et al.,[22] participants used kits containing preservation reagents (RNA Later and OMNIgene); samples were initially stored at − 20 °C, transported on ice and then stored at − 80 °C. Lin et al.[25] used DNA/RNA Shield tubes for self-collection, with storage at − 80 °C. McDermott et al.[30] employed a commode system and nucleic acid preservation tubes, storing samples at − 80 °C. In contrast, West et al.[37] used sealable plastic bags frozen at − 20 °C, although the interval between collection and lab delivery was not reported. Wu et al.[38] preserved fresh samples in 95 % ethanol prior to transport and stored them at − 80 °C. Li et al.,[28] Huang et al.[23] and Lee et al.[40] collected fresh stool samples but did not describe collection and handling protocols. In multi-strain probiotic studies, Przewłócka et al.[34] used standardised containers with immediate freezing at − 80 °C, while Wang et al.[36] used freeze-dried collection tubes with similar freezing procedures. Önes et al.[32] did not report their collection or storage methods. Of the two other nutritional supplement studies, Morita et al.[39] and Onishi et al.[33] provided Sarstedt containers for home collection, instructing participants to freeze samples at approximately − 30 °C before transport to the clinic. Dietary intervention protocols also employed varied approaches. Furber et al.[27] collected the first stool of the day, freezing samples at − 80 °C within 2 hours. Mancin et al.[29] used sterile swab tubes containing preservative, stored at − 20 °C. Murtaza et al.[31] used OMNIgene kits for samples collected before and after the training-diet intervention in athletes. In the sole exercise-only intervention, Zhong et al.,[26] stool samples were pre- and post-intervention, but the collection and storage protocols were not described.

Microbial, α-diversity, and SCFA techniques

Most probiotic and postbiotic interventions used 16S rRNA gene amplicon sequencing, targeting different hypervariable regions: V1–V3,[23] V3–V4[25,32,38,40] and V4.[36] Shannon index was commonly used to assess α-diversity,[23,28,32,38,40] sometimes alongside Chao1,[32,38] Inverted Simpson, ACE and others.[34] Two studies employed alternative sequencing: Gross et al.[22] used shotgun metagenomic (Shannon entropy) and Li et al.[28] used DNB-seq with combinatorial probe-anchor synthesis. West et al.[37] used denaturing gradient gel electrophoresis (DGGE) and qPCR and McDermott et al.[30] used real-time qPCR, though α-diversity was not reported in either. Of the two other nutritional supplement studies, Morita et al.[39] and Onishi et al.[33] used 16S rRNA gene amplicon sequencing, targeting the V4 region and employed species-specific qPCR to quantify *Bacteroides uniformis*. Morita et al.[39] also used Shannon, Chao1 and phylogenetic distance indices, but SCFA concentrations were not measured in either study.

SCFA quantification methods varied and were inconsistently reported. They included gas chromatography–mass spectrometry (GC-MS),[2] high-performance liquid chromatography (HPLC),[40] ultra-performance liquid chromatography tandem mass spectrometry (UPLC-MS/MS),[38] and gas chromatography with flame ionisation detection (GC-FID),[34] while several studies did not report SCFA analysis.[22,25,30,32,36–37]

Diet-based interventions applied 16S rRNA gene amplicon sequencing, targeting V3–V4,[29] V6–V8,[31] and V4.[27] Furber et al.[27] also included ITS1–ITS2 sequencing for fungal taxa and viral metagenomics. Reported diversity metrics included Fisher's alpha,[27] Shannon's effective number of species (ENS) and operational taxonomic units (OTUs)[29] and Shannon and Simpson indices.[27] None of the dietary studies assessed SCFA concentrations. In the sole exercise-only intervention, Zhong et al.[26] employed 16S rRNA gene amplicon sequencing (V3–V4), with multiple α-diversity metrics reported: Sobs, Chao1, ACE, Shannon and Simpson. SCFA concentrations were not reported.

Faecal microbial composition and diversity

Two probiotic studies showed increased levels of supplemented bacterial species: *B. longum* subsp. *longum*[25] and *Limosilactobacillus fermentum* VRI-003 PCC.[37] Przewłócka et al.[34] found increases in *Bacteroides fluxus* and *Roseburia inulinivorans* after 4 weeks of a multi-strain probiotic with vitamin D3. No other significant species-level changes were found.[22,25,28,32,36,38,40] McDermott et al.[30] detected *Lactobacillus helveticus* Lafti L10 in 57.1 % of participants post-intervention, though without statistical group comparisons.

At the phylum level, Lin et al.[25] noted an increase in Actinomycetota and Bacillota and a reduction in Pseudomonadota following *Bifidobacterium longum* subsp. *longum* supplementation, although significance was not stated. Gross et al.[22] found no changes in *Veillonella* abundance after *Veillonella atypica* FB0054 supplementation. Huang et al.[23] reported reductions in several bacterial groups (e.g., *Anaerotruncus*, *Caproiciproducens*, *Coprobacillus*, *Desulfovibrio*, *Dielma*, Family_XIII_UCG_001, *Holdemania* and *Oxalobacter*) and increases in *Akkermansia*, *Bifidobacterium*, *Butyricimonas* and *Lactobacillus*, though no baseline data were reported. Wang et al.[36] noted increases in *Bacteroidota*, *Bacillota*, *Pseudomonadota*, and *Actinomycetota*, after 5 weeks of *Lactobacillus acidophilus* and *Bifidobacterium longum* supplementation but statistical significance was not reported.

At the family and class levels, Przewłócka et al.[34] found reductions in the families Lachnospiraceae, Peptostreptococcaceae and Lactobacillaceae, all within the phylum Bacillota, alongside an increase in the class Negativicutes and an overall reduction in Bacillota abundance. No further faecal sample bacterial taxa changes were observed with probiotic supplementation. At the genus level, Lin et al.[25] showed significant increases in *Bifidobacterium* and members of the Lactobacillaceae family, including genera formerly classified under *Lactobacillus*. Wu et al.[38] found *Lacticaseibacillus paracasei* PS23 supplementation led to increases in the genera *Lacticaseibacillus*, *Streptococcus*, *Blautia* and other members of the Lac-

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tobacillaceae family (formerly grouped under *Lactobacillus*), alongside a decrease in *Prevotella*. Postbiotic supplementation with heat-killed *Lacticaseibacillus paracasei* PS23 led to an increase in the genera *Lacticaseibacillus* and *Collinsella*; however, baseline microbiota data were not reported.[38] Przewłócka et al.[34] reported an increase in the genera *Faecalibacterium* and *Prevotella* and a decrease in *Collinsella* and *Bacteroides* after supplementation with a multi-bacterial formulation. Wang et al.[36] noted increases in the genera *Lacticaseibacillus*, *Olsenella*, *Weissella* and *Anaerostipes* and decreases in *Cloacibacillus* and *Alphaproteobacteria*_unclassified.

Seven studies reported no significant changes in α-diversity following probiotic supplementation [22,28,32,34,37,38,40] and two reported no significant *β*-diversity shifts.[22,28] However, three studies observed significant *β*-diversity shifts following probiotic use.[32,38,40] Lee et al.[40] reported differences in β-diversity between groups receiving heat-killed *Lactiplantibacillus plantarum* TWK10 and controls and between live and heat-killed TWK10. Wu et al.[38] found significant β-diversity changes after 6 weeks of *Lacticaseibacillus paracasei* PS23 supplementation compared to placebo. Finally, Önes et al.[32] observed significant β-diversity differences following kefir supplementation over four weeks. Of the two other nutritional supplement studies, one examined both α- and β-diversity and reported no significant differences between groups following 9 weeks of supplementation with flaxseed lignans, αCD or a placebo.[39] Additionally, no significant changes in species-level relative abundance were reported.[33,39]

Among the three dietary studies, two assessed α-diversity and found no significant differences between dietary intervention groups.[29,31] At the phylum level, Mancin et al.[29] reported a decrease in the abundance of Actinomycetota following adherence to the KEMEPHY ketogenic Mediterranean diet (KDP) compared to a Western diet over 30 days. Carbohydrate intake was negatively correlated with the genus, *Odoribacter*, and fat intake was negatively associated with *Fusicatenibacter*. Murtaza et al.[31] identified three dominant gut microbiota profiles at baseline: *Prevotella* predominant, *Bacteroides* dominant and a profile dominated by members of the phylum *Bacillota* in one participant. These profiles remained relatively stable after participants adhered to one of three prescribed diets (high carbohydrate, protein carbohydrate and low carbohydrate).

Furber et al.[27] did not compare α- or β-diversity between intervention groups (high protein diet vs. high carbohydrate diet), instead focusing on within-group changes over time. The high protein diet was associated with a significant reduction in Fisher-α diversity of inducible viruses, which did not return to baseline levels post-intervention. Significant shifts were observed in both free viral particles and inducible virus communities. The high-carbohydrate diet appeared to have a greater impact on bacterial community composition than on the viral community; however, these shifts were not statistically significant. Notably, the high carbohydrate diet was associated with increased relative abundance in *Leuconostoc*, *Lactococcus* and *Collinsella*, while *Streptococcus* decreased.

In the sole exercise-only intervention, Zhong et al.[26] reported no significant changes in α-diversity between intervention and control groups. At the class level, the exercise intervention was associated with an increase in *Bacilli* and a decrease in *Betaproteobacteria*. At the order level, *Lactobacillales* increased while *Burkholderiales* decreased. At the family level, reductions were noted in Sutterellaceae and Bacteroidaceae. At the genus level, the intervention resulted in a decreased abundance of *Holdemania*, *Anaerostipes* and *Bacteroides*.

Faecal SCFA concentration

Faecal SCFA concentrations were assessed pre- and post-intervention in $n = 4$ studies (**Table 4**), all of which investigated probiotic and/or postbiotic supplementation.[28,34,38,40] One additional study[23] reported only post-intervention SCFA values. Significant increases in faecal acetate concentrations were observed following 6 weeks of supplementation with *L. plantarum* or heat-killed *L. plantarum* (formerly *Lactobacillus plantarum*). No significant changes were detected in the control group, and no significant between-group differences were reported.[40] Huang et al.[23] observed higher post-intervention concentrations of acetic acid, propionic acid and butyric acid in the probiotic group (*L. plantarum* PS128) compared to placebo; however, the absence of baseline data limits the ability to attribute these changes to the intervention. Li et al.[28] reported a 4.5-fold increase in faecal acetate following 8 weeks of yoghurt supplementation containing *Bifidobacterium animalis* subsp. *lactis* BL-99 compared to control. In contrast, two studies[34,38] found no significant post-intervention changes in faecal SCFA concentrations.

None of the studies assessing SCFA outcomes adhered to best practice guidelines for the control of experimental confounders in exercise–gastroenterology research, as outlined by Costa et al.[73] As described previously, dietary control across studies varied with those measuring SCFA mostly relying on simplistic strategies, such as requesting participants to maintain usual intake or record dietary logs, which are insufficient for standardising pre-trial diet. Several studies failed to report quantitative dietary intake or key microbiota-modulating components.

Exercise performance outcomes in response to interventions

Eighteen studies assessed exercise performance outcomes across various domains, including TTE, TT, Cooper's test, strength and anaerobic performance and functional capacity. Several also evaluated physiological parameters, such as aerobic capacity, and examined correlations against faecal microbiota composition (**Table 4**). Six studies examined TTE outcomes, $n = 5$ probiotics and/or postbiotics [22,23,30,34,40] and $n = 1$ dietary study.[27] Results from single-strain probiotic trials were mixed: *Veillonella atypica* FB0054[22] and *Lactobacillus helveticus* Lafti L10 had no effect on TTE;[30] whereas *L. plantarum* PS128 [23] increased post-intervention TTE compared to placebo. However, baseline values were not reported, limiting the interpretation of the magnitude of change. In that study, TTE was significantly greater in the intervention group than placebo (1,679 s vs. 1,083 s, $p < 0.05$). Both viable and heat-killed forms of *L. plantarum* TWK10 improved TTE at 85 % $\dot{V}O_{2max}$.[40] A multi-strain probiotic containing *B. lactis* W51, *L. brevis* W63, *L. acidophilus* W22, *B. bifidum* W23, *Lc. lactis* W58 and vitamin D_3 improved TTE by approximately 12.6 % in mixed martial arts athletes.[34] TTE increased significantly in the intervention group (496.30 ± 89.98 to 559.00 ± 68.99 s, $p = 0.023$), while no significant change occurred in the control group (489.91 ± 72.02 to 468.55 ± 102.03 s, $p = 0.685$). Howev-

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er, as only within-group comparisons were conducted and no between-group statistical analyses were reported, the true effect of the intervention remains unclear. In dietary studies, a high-protein diet led to a significant decrease in TTE, while a high-carbohydrate diet resulted in a modest increase in TTE.[27]

Three studies assessed TT, $n = 2$ focused on nutritional supplementation,[33,39] and $n = 1$ on a dietary intervention.[31] Both supplementation trials demonstrated improved 10 km performance with αCD supplementation in physically active males.[33,39] A dietary intervention comparing HCHO, PCHO and LCHF diets found that only HCHO and PCHO improved 10 km race walking time (average of 4.2 %), despite all groups showing increased aerobic capacity.[31]

Two studies employed Cooper's test both were probiotic studies. Lin et al.[25] found that in well-trained middle and long-distance runners, while no significant differences were observed at any time point, the intervention group showed significantly greater improvements in distance covered at the 6th, 9th, and 12th minutes, compared to the placebo group. Wang et al.,[36] found increased distance in amateur marathon runners post-supplementation, though changes were not significantly different between groups.

Four studies investigated strength or anaerobic performance, $n = 3$ probiotic and/or postbiotics[28,35,38] and $n = 1$ dietary intervention.[29] Probiotic supplementation significantly increased 60 °/s knee joint extensor and flexor strength in skiers compared to control.[28] *Lactobacillus plantarum* PS23 (both viable and postbiotic, i.e., heat-killed), helped mitigate neuromuscular fatigue following exercise-induced muscle damage in physically active adults,[38] showing improved performance countermovement jump height, rate of force development and Wingate anaerobic performance. A multi-strain probiotic did not outperform placebo for anaerobic capacity in MMA athletes.[35] One dietary study comparing a ketogenic Mediterranean diet and a Western diet in semi-professional soccer players found no significant differences between groups in Yo–Yo intermittent test performance, countermovement jump height or maximal isometric quadriceps strength, despite suggesting potential improvements in agility, sprinting and power output.[29] In the sole exercise-only intervention and which involved older adults, Zhong et al.[26] reported improvements in chair sit-and-reach flexibility and single-leg standing balance (eyes closed) following an 8-week aerobic and resistance training program. However, no significant changes were observed in strength or timed functional tests.

Six studies evaluated aerobic capacity, $n = 4$ were probiotics,[23,28,34,37] $n = 1$ nutritional supplementation[33] and $n = 1$ dietary intervention.[42] *Lactobacillus plantarum* PS128 [23] and *Limosilactobacillus fermentum* VRI-003 PCC[37] had no effect on $\dot{V}O_{2max}$, whereas *Bifidobacterium animalis* subsp. *lactis* BL-99 significantly improved $\dot{V}O_{2max}$ in national-level cross-country skiers compared to control.[28] A multi-strain probiotic had no impact on MMA athletes.[34] α-Cyclodextrin supplementation did not change $\dot{V}O_{2max}$.[33] Murtaza et al.[31] reported a significant post-intervention increase in $\dot{V}O_{2peak}$ across all dietary intervention groups (HCHO, PCHO and LCHF diet) ($p < 0.001$, 90 % confidence interval [CI]: 2.55, 5.20 %). However, $\dot{V}O_2$ uptake, at a speed approximating 20 km race pace, decreased in both the high carbohydrate (90 % CI: − 7.05, − 0.244 %) and periodised carbohydrate (90 % CI: − 5.18, − 0.86 %) groups, whereas it remained at pre-intervention levels in the low carbohydrate high fat group. Furthermore, mean 10 km race walk time improved in the high carbohydrate (6.6 %, 90 % CI: 4.1, 9.1 %) and periodised carbohydrate (5.3 %, 90 % CI: 3.4, 7.2 %) but showed no clear improvement in the low carbohydrate high fat group (− 1.6, 90 % CI: − 8.5, 5.3 %).

Correlations between microbiota alterations and exercise performance

Seven studies, $n = 4$ probiotic studies,[22,32,38,40] $n = 2$ dietary studies,[27,31] and $n = 1$ exercise study,[26] explored correlations between changes in the gut microbiota and performance outcomes (**Table 4**). Among the probiotic interventions, Gross et al.[22] reported no correlation between changes in β-diversity and TTE. In contrast, Lee et al.[40] reported a moderate positive correlation between TTE and the Coriobacteriaceae family following TWK10 probiotic supplementation, with an approximate correlation coefficient of $r = {\sim}0.5$ ($p < 0.05$), which was inferred from the heat map visualisation. They also observed a strong positive correlation between TTE and the Veillonellaceae family following the TWK10-hk postbiotic intervention ($r = 0.65$, $p < 0.01$). Other microbial families demonstrated negative correlations with TTE. Wu et al.[38] found weak to moderate positive correlations between the abundance of *Lacticaseibacillus*, *Streptococcus*, *Blautia* and *Lactobacillus* with improved performance across various exercise tests, while *Prevotella* abundance was weakly negatively correlated with performance. Önes et al.[32] reported that high-performance athletes exhibited greater relative abundance of *Faecalibacterium prausnitzii* and *P. copri*, whereas *Dorea formicigenerans* and *Oxalobacter formigenes* were more abundant in lower-performing athletes; however, most correlations between microbiota profiles and performance outcomes were not statistically significant.

Among the dietary studies, Furber et al.[27] found that individuals whose performance declined during a high-protein diet exhibited greater shifts in overall faecal microbial community composition, whereas those on a high-carbohydrate diet showed improved performance and greater microbial stability. Murtaza et al.[31] reported no significant associations between baseline faecal bacterial profiles and performance; however, following a low-carbohydrate high-fat dietary intervention, strong negative correlations were found between *Bacteroides* and fat oxidation and between *Dorea* and exercise economy.

The exercise-only study by Zhong et al.[26] identified negative associations between specific faecal bacterial taxa and physical performance measures. A strong negative correlation was found between *Betaproteobacteria* and chair sit-and-reach performance ($r = -0.704$, $p = 0.005$), while moderate negative associations were reported between *Holdemania* and single-leg standing with eyes closed ($r = -0.553$, $p = 0.040$) and *Betaproteobacteria* and grip strength ($r = -0.551$, $p = 0.041$).

Discussion

This systematic literature review represents, to our knowledge, the first comprehensive synthesis examining whether nutritional supplement, dietary and exercise interventions can modulate gut microbiota composition and subsequently enhance exercise performance in healthy active adult populations. Previous narrative and systematic reviews in this topic area have typically focused on sub-

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sets of these interventions, often overlooking key studies or methodological limitations. Many have not consistently included concurrent assessments of gut microbial composition alongside exercise performance outcomes, nor examined correlations with SCFAs in plasma or faeces. By including only studies that assessed both gut microbiota and exercise performance, this review addresses these critical gaps and provides a more integrated perspective on the potential interactions between gut microbiota and exercise outcomes.

From a methodological perspective, considerable variability was observed in how studies assessed the impact of interventions on faecal bacterial composition. While differences in stool collection and management procedures were noted, a greater source of inconsistency arose from the analytical techniques employed, most notably, quantitative polymerase chain reaction (qPCR) and next-generation sequencing approaches such as 16S rRNA and shotgun metagenomics. These techniques yield fundamentally different outputs: qPCR provides absolute values (e.g., CFU/g), whereas sequencing typically reports relative abundance (e.g., % of total reads). Compounding this, inconsistency in taxonomic levels, bacterial targets and reporting units hindered direct comparisons across studies. As a result, interpreting intervention-induced microbiota shifts, and, by extension, their effects on exercise performance, requires considerable caution. These methodological discrepancies underscore the urgent need for standardised protocols to enhance comparability and improve the validity of conclusions drawn from this growing body of literature.

Despite methodological limitations, some studies did report changes in bacterial composition following probiotic supplementation. Three probiotic studies reported significant increases in the supplemented strains' relative abundance[25, 30, 37]; however, effects on broader microbiota taxa were negligible or inconsistent. This aligns with previous findings from a systematic review showing limited microbial shifts beyond the administered strains and no substantial effects on gastrointestinal status (i.e., integrity and function) markers or SCFA concentrations.[76] While a subset of studies in the current review reported taxonomic shifts beyond the supplemented strains, such as increases in saccharolytic genera (*Blautia*, *Faecalibacterium*, *Anaerostipes*)[34, 36, 38] and decreases in potentially pro-inflammatory taxa (*Desulfovibrio*, *Dielma*),[23] these findings were inconsistent, often lacked baseline comparisons and did not consistently align with improvements in α- or β-diversity. Notably, only three probiotic studies found significant changes in β-diversity,[32, 38, 40] while three others (two probiotic and one supplement) found no significant shifts.[22, 28, 39] Similar variability was observed in dietary and exercise interventions. For example, a high-carbohydrate diet was associated with increased relative abundance of *Leuconostoc*, *Lactococcus* and *Collinsella*, while high-protein and ketogenic diets were associated with reductions in microbial diversity or shifts in phylum-level taxa.[27] The sole exercise study included in this review reported modest taxonomic changes but no effects on α-diversity.[26] Collectively, while some interventions appeared to modulate saccharolytic or butyrate-producing taxa, the broader impact on faecal bacterial diversity and composition appears limited and inconsistent. A further limitation is that many studies did not adequately control for key confounders known to affect faecal microbial profiles, such as diet composition, faecal water content, physical activity and gastrointestinal transit time.[77–80] The absence of such controls compromises the ability to attribute microbiota shifts to the interventions themselves, raising the possibility that reported improvements in exercise performance may instead reflect suboptimal research design. This aligns with previous findings by Kristensen et al.,[81] who reported no significant probiotic-induced changes in α-diversity among healthy populations. Although probiotic-induced microbial shifts are often small and transient, they may influence bacterial metabolic activity, particularly the production of SCFAs like acetate and butyrate,[82] which have been proposed to support both health[83] and exercise performance.[84] However, it remains unclear whether these changes are biologically meaningful in athletic contexts or if their significance has been overstated.

This variability in intervention outcomes contrasts with the inherent stability observed in gut microbiota profiles among physically trained individuals under controlled conditions. From a historical perspective, repeated assessments of faecal bacterial profiles in endurance-trained individuals, using best practice experimental controls,[73] with test–re-test reliability assessments, have demonstrated consistent and stable microbiota compositions.[9] Specifically, predominant phyla include *Bacillota* (69 % relative abundance), *Bacteroidota* (24 %), *Actinomycetota* (2 %), *Pseudomonadota* (2 %) and *Verrucomicrobiota* (2 %). At the family level, Ruminococcaceae and Lachnospiraceae each comprise 27 %, followed by Bacteriodaceae (13 %), Acidaminococcaceae (6 %) and Prevotellaceae (5 %). Dominant genera include *Bacteroides* (13 %), *Faecalibacterium* (11 %), *Agathobacter* (5.7 %), *Phascolarctobacterium* (5.3 %) and *Prevotella* (4.3 %). Measures of α-diversity indices, assessed using the Shannon Equitability Index (SEI), also showed minimal variation: phyla SEI = 0.188 (95 % CI: 0.166–0.211), family SEI = 0.245 (95 % CI: 0.234–0.256) and genus SEI = 0.282 (95 % CI: 0.269–0.296).[9] These findings highlight the underlying stability of the gut microbiota in physically trained individuals, reinforcing the need for rigorous control of methodological and biological variability in intervention trials. Within this context, the lack of consistent changes in microbiota composition across studies included in this review, particularly at lower taxonomic levels (i.e., more specific classifications such as genus and species), is unsurprising and likely reflects heterogeneity in study protocols, population characteristics, analytical approaches and control of key confounders, rather than genuine intervention effects.

Despite mechanisms by which changes in the gut microbiota may enhance exercise performance being linked to the increased presence of SCFAs, these key microbial metabolites were assessed in only five studies.[23, 28, 34, 38, 40] Of these, just one study[28] reported a significant increase in acetate, while the remainder found no meaningful changes. One study[23] reported post-intervention means only, preventing evaluation of within-group change. Collectively, these findings suggest that the interventions in this review did not reliably modulate microbial fermentation end-products, which are proposed to mediate gut–exercise performance relationships. Given the proposed role of SCFAs in exercise physiology, the limited and inconsistent data further diminish the plausibility of a microbiota-mediated performance benefit. Furthermore, only seven studies directly examined associations between microbiota alterations and performance outcomes, with results

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varying considerably in both the direction and strength of reported correlations. While a small number of studies reported statistically significant associations between specific bacterial taxa and improved or impaired performance,[26, 31, 32, 38, 40] substantial heterogeneity in study designs, microbial endpoints and performance measures limits the ability to draw firm conclusions. Notably, one study reported strong positive correlations between TTE and the relative abundance of Coriobacteriaceae and Veillonellaceae following probiotic or postbiotic TWK10 interventions,[40] suggesting a potential mechanistic role of these taxa in energy metabolism. In contrast, several studies reported no significant associations, or only weak correlations between gut microbial changes and performance outcomes.[22, 32, 38] Taken together, there appear to be no considerable links between intervention-induced changes to the gut microbiota and exercise performance. The dietary intervention by Furber et al.[27] adds complexity to these findings, with a high-protein diet being associated with larger bacterial shifts in participants whose performance declined; whereas HCHO was linked to improved performance and microbial stability. Similarly, the exercise only study by Zhong et al.[26] reported predominantly negative correlations between microbial taxa and physical function measures. However, the physiological relevance of these findings remains uncertain due to small effect sizes and diverse outcome measures. As a whole, it would therefore be premature from a translational practice perspective, to conclude that changes in gut microbiota composition, whether induced through nutritional supplementation, dietary or exercise interventions, directly improve exercise performance. Current evidence does not support such claims, and doing so risks overstating the practical significance of largely speculative or inconsistently observed findings. Given the absence of consistent improvements in performance outcomes across the review, these findings align with the broader conclusion that meaningful microbiota-mediated exercise performance enhancements remain unsubstantiated.

Study limitations

Despite most included studies being rated as low risk of bias, several methodological limitations were identified that constrain the strength of conclusions in this systematic review. Common issues across studies included inadequate blinding, unclear randomisation procedures, inconsistent adherence to exercise-gastroenterology best-practice guidelines,[73] and potential conflicts of interest, with more than half receiving industry funding or product support. A key limitation was the limited and inconsistent investigation of associations between gut microbiota and exercise performance, with only seven studies exploring these relationships and considerable heterogeneity in study design, intervention type, microbial analysis methods and performance assessments, precluding direct comparisons. Substantial variability in faecal sample collection and storage procedures raised concerns. While some studies used validated collection and preservation methods, others provided minimal procedural detail,[23, 28, 40] raising concerns about cross-contamination, microbial degradation and/or compositional shifts due to variable storage conditions. For example, some used freeze-dried collection tubes[36] or home freezing at approximately −30 °C,[33, 39] which may offer some stability. However, inconsistency across studies hampers data comparability. Notably, two studies[26, 32] failed to report collection and storage protocols entirely, limiting both reproducibility and interpretability. Likewise, heterogeneity in microbiota and SCFA assessment methods, including variation in sequencing platforms, depth and bioinformatic pipelines, as well as inconsistent use of SCFA quantification techniques, limited comparability and likely contributed to divergent findings. Inconsistent data reporting, such as the use of fold-changes without absolute values[23] or reliance on heat maps, further impeded interpretation. Additional study design concerns included post-randomisation group switching,[32] unclear performance categorisation and inappropriate group comparisons. Significant heterogeneity in probiotic strain, dose and delivery mode, along with the predominant inclusion of biological sex male participants, also limits generalisability. Finally, although rigorous search strategies were employed across five databases and citation searches, it remains possible that some relevant studies were missed. Collectively, these methodological inconsistencies and data gaps precluded meta-analysis and reduced the overall strength of conclusions regarding the impact of nutritional supplementation, dietary or exercise interventions on gut microbiota and performance in healthy active adults.

Future implications and translational application

This systematic literature review highlights that, at present, there is insufficient high-quality evidence to determine whether gut microbiota manipulation enhances exercise performance. Methodological issues, particularly variability in study designs, inconsistent dietary and exercise controls and inconsistent reporting of microbiota and metabolite outcomes, significantly limit the ability to draw firm conclusions. Given that both diet and exercise independently influence gut microbiota composition,[17, 85–87] future research must rigorously control for these variables to minimise confounding. Few studies adequately monitored or standardised participants' exercise regimens, despite exercise performance being a primary outcome. Consistent documentation of exercise type, intensity, duration and training load using validated tools such as accelerometry should be standard practice,[88] these methods have been successfully used to link physical activity with physiological outcomes, including gut-related physiological responses.[63, 89–91]

Dietary intake also requires greater control. Changes in gut microbiota can occur within 24–48 hours of a dietary manipulation, particularly at the species, genus and family levels.[8, 87] Therefore, all food and fluid consumed at least 48 hours prior to sample collection should be documented,[17] with continued dietary monitoring throughout the intervention period. Standardising intake of microbiota-relevant foods such as FODMAPs and dietary fibre is critical[14] to limit prebiotic effects that may confound outcomes, particularly through modulation of beneficial taxa like *Bifidobacterium* species.[92] However, caution is warranted when considering resistant starch, as its content is highly variable depending on food preparation and is not routinely included in standard nutrient databases, limiting accurate assessment in most dietary studies. Where substantial dietary variation exists, it should be accounted for statistically. Prospective food and fluid diaries can aid protocol adherence, and pre-inter-

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vention controlled feeding phases can help reduce the impact of dietary variability.[17] Crossover designs, where feasible, offer an effective way to reduce inter-individual variability[17] and may be particularly useful in studies involving trained individuals.[9] In addition to improving intervention protocols, future studies should strengthen the measurement and interpretation of outcomes. SCFAs, including acetate, propionate and butyrate, are key microbial metabolites with potential relevance to exercise performance,[8,51] yet were rarely quantified. SCFA measurement should be routinely incorporated to assess the functional impact of microbiota changes. Probiotic timing and adherence also require better reporting. Given that probiotic survival may improve under higher stomach pH[93] and that digestion is influenced by circadian rhythms,[94] future studies should provide clear guidance on intake timing and rigorously monitor adherence to enhance intervention fidelity.[17]

Microbiota analysis and reporting practices also need refinement. Heat maps used in several studies limited taxonomic-level data extraction, and inconsistencies in sequencing methods (e.g., 16S rRNA target regions, sequencing depth and pipelines) contributed to reporting variability. Future work should prioritise quantitative reporting using relative abundance data and incorporate complementary tools such as optical techniques and flow cytometry. When paired with sequencing and metabolomics, these can provide more accurate insights into microbial function and its relationship with host physiology.[95] Limitations of faecal sampling also warrant consideration. While convenient and widely used, faecal samples may not accurately represent microbial communities across the gastrointestinal tract, particularly in the mucosa and small intestine.[3,96] Microbial composition differs markedly across gut regions due to localised chemical, nutritional and immunological environments,[3] and the heterogeneity of faecal bacterial distribution further complicates interpretation.[97] These issues are especially problematic when assessing interventions targeting regions beyond the colon. For instance, one included study attempted to assess *Veillonella atypica*, a predominantly small intestinal species, via faecal sampling.[22] This highlights the need for validated, less-invasive methods to characterise small intestinal and mucosa-associated microbiota, which would improve both mechanistic understanding and intervention precision. Finally, this review identified a significant evidence gap: no study to date has directly examined the effects of prebiotic or synbiotic supplementation on both gut microbiota composition and exercise performance. Although it is possible some studies were missed, this risk was minimised by searching five academic databases and performing citation tracking. One adjacent study by Rauch et al.[10] involving healthy, active adults exposed to repeated exertional-heat stress (EHS) found that 8 weeks of prebiotic supplementation (resistant starch, FOS, β-GOS) attenuated markers of intestinal epithelial injury and thermoregulatory strain, without impairing gastrointestinal tolerance or transit time and/or exacerbating exercise-associated gastrointestinal symptoms. While microbial α-diversity increased, this change was not statistically significant; however, enrichment of commensal taxa (e.g., Ruminococcaceae, Catenibacterium) was observed. These microbial shifts, despite no significant change in faecal SCFA concentrations (including acetate, propionate and butyrate), may enhance gut barrier function and systemic tolerance to exertional stress. This suggests a plausible, but as yet untested, mechanism by which gut microbiota modulation could influence exercise performance outcomes. Targeted trials are needed to test this hypothesis directly.

Conclusions

This systematic review identified limited and inconsistent evidence regarding the effects of gut microbiota-targeted interventions, via nutritional supplements, diet or exercise, on exercise performance in healthy, active individuals. Reported benefits were highly variable, often undermined by poor dietary and exercise control, and frequently absent. Furthermore, changes in faecal microbiota were not consistently linked with exercise performance, and mechanistic insights were scarce. Given the small number of studies and considerable methodological heterogeneity, the current evidence base is insufficient to draw firm conclusions about the effectiveness of microbiota-modifying strategies for performance enhancement. Future research should prioritise well-controlled trials that incorporate standardised dietary intake, consistent exercise monitoring and robust, functional microbiota analysis. In particular, studies that longitudinally assess microbial metabolites (e.g., SCFAs), host physiological markers and individual responses may clarify whether targeted strategies can offer meaningful ergogenic benefits.

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Statements and additional information

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