Food Science of Animal Products

Research Article

Dietary probiotic intervention for childhood obesity management: a systematic review and meta-analysis

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Abstract: Background: Childhood and adolescent obesity are a global health concern, with unclear efficacy of probiotics as an intervention. Objectives: This meta-analysis evaluates probiotics' effects on obesity-related metrics (body mass index (BMI), weight, lipid profiles) in children/adolescents (2–18 years) compared to placebo or no intervention. Methods: We searched PubMed, Scopus, and Web of Science databases for randomized trials up to October 2024. Data extraction and analysis were conducted employing weighted mean differences (WMD) with 95% confidence intervals (CI) via fixed/random-effects models. Study quality, publication bias, and sensitivity were assessed. Results: Nine studies were included. Probiotics significantly reduced BMI (WMD: −1.57; 95% CI: −2.55 to −0.58; $P = 0.002$) but showed no significant impact on weight, waist circumference, lipid parameters, or other anthropometric measures. No publication bias was detected, and sensitivity analysis confirmed robustness. Conclusions: Probiotics effectively reduce BMI in obese children and adolescents, supporting their potential role in obesity management. This meta-analysis has been registered on PROSPERO (CRD42024610790).

Keywords: probiotics; overweight; obesity; children; adolescents; meta-analysis

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1 Introduction

Pediatric obesity is conventionally defined using body mass index (BMI) percentile rankings. Generally, a BMI between the 85th and 95th percentile is defined as overweight, and ≥ 95th percentile as obese[1]. The number of obese children and adolescents has been increasing rapidly, at least fourfold since 1985[2], and the global obesity epidemic has been formally recognized by the World Health Organization (WHO) as constituting one of the most formidable public health challenges confronting contemporary global society. According to the World Obesity Federation in 2020, the aggregate number of children and adolescents suffering from obesity will be approximately 379 million, including 39 million cases occurring in the preschool demographic (< 5 years) and 340 million cases identified among school-aged children and adolescents (5–19 years)[3]. In response, China has made the prevention and control of obesity in children and adolescents a priority of the Healthy China action, and set the goal of controlling the obesity rate of primary and secondary school students to less than 12% by 2030. Obesity places a tremendous burden on the bodies of children and adolescents, increasing the risk of developing several diseases such as asthma, cancer, diabetes, hypercholesterolemia, and cardiovascular disease[4–5]. In addition to the negative physiological effects, obesity can also lead to psychological issues in children and adolescents[6], including loss of self-esteem and depression[7]. The physical and psychological effects of obesity on children and adolescents can ultimately lead to a reduction in their life expectancy[8].

The etiology of obesity is complex but primarily driven by lifestyle factors, including chronic overeating and lower rates of physical activity[9]. Despite existing dietary guidelines, children and adolescents often do not consume enough of the healthier components, such as fruits, vegetables, and whole grains[10]. WHO issues evidence-based guidelines stipulating that pediatric and adolescent populations should accumulate a minimum of 60 min daily of moderate-to-vigorous physical activity to meet international health standards, but most of children and adolescents fall short of this standard of daily physical activity[11–12]. Mainstream treatment options for obesity in children and adolescents include behavioral, pharmacological, and surgical interventions, but due to the complexity of the etiology of obesity, it is difficult for a single treatment option to reduce obesity in children and adolescents at the population level[13]. In terms of behavioral interventions, dietary interventions are one of them, and a commonly used method is the "Traffic Light Diet", which aims to reduce energy intake while improving the nutritional quality of food[14]. Pharmacological interventions are used in scenarios where behavioral interventions are less effective but usually have some adverse effects, common medications used to treat obese children and adolescents include orlistat and phentermine[15–16]. Surgical interventions such as

metabolic and bariatric surgery are required when obesity symptoms in children or adolescents are severe to a certain extent, but fewer studies have been conducted on such approaches, and their safety and efficacy are difficult to ensure[13].

Currently, in the field of adult obesity, it has been shown that probiotics are effective in the treatment of obesity by improving the composition of the human gut microbiome, contributing to the reduction of body weight and adiposity, balancing lipids, insulin, and fasting glucose levels, and reducing the production of inflammatory factors[17–18]. However, some studies have failed to demonstrate statistically significant therapeutic outcomes associated with probiotic interventions in pediatric obesity management on most anthropometric measurements[19], and there is disagreement on the therapeutic efficacy of probiotic interventions in obesity management. The current evidence base remains disproportionately focused on adult populations, with only a limited number of methodologically robust clinical trials systematically investigating the therapeutic potential of probiotic interventions in addressing adiposity pathophysiology in pediatric populations[20]. Evidence regarding their efficacy in pediatric populations remains controversial. While some studies suggest beneficial modulatory effects[21–22], others have reported no significant impact[23] or even adverse outcomes[24]. Considering these evidence gaps, the present investigation was undertaken to systematically evaluate the therapeutic efficacy of probiotic interventions in children and adolescents through a rigorous meta-analytical synthesis of randomized controlled trials. Among them, the primary outcomes included body weight, BMI, total cholesterol (TC), and triglycerides (TG), and the secondary outcomes included waist circumference (WC), waist-to-hip ratio (WHR), low-density lipoprotein cholesterol (LDL-C), and high-density lipoprotein cholesterol (HDL-C).

2 Material and methods

This evidence synthesis was conducted in strict adherence to the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines[25], ensuring compliance with contemporary standards for systematic review methodology. Prior to initiating the research protocol, the study design was prospectively registered with the International Prospective Register of Systematic Reviews (PROSPERO) to ensure methodological transparency and prevent outcome reporting bias (Registration ID: CRD42024610790). The analytical framework incorporated dual independent review processes at all stages of study selection, data extraction, and quality assessment to maintain methodological rigor.

2.1 Search strategy

A systematic literature search was conducted across three major biomedical databases: PubMed, Scopus, and Web of Science. The search strategy employed a dual approach combining controlled vocabulary terms (e.g., MeSH) with text word searches to optimize retrieval sensitivity. The search timeframe encompassed records from database inception through October 2024 to ensure comprehensive coverage of historical and contemporary literature. The first author independently executed the search protocol without imposing temporal, linguistic, or methodological restrictions to minimize selection bias. The following search formula was used: ("Probiotics" [MeSH Terms] OR "Probiotic" [Title/Abstract]) AND ("Pediatric Obesity" [MeSH Terms] OR ("obesity pediatric" [Title/Abstract] OR "childhood obesity" [Title/Abstract] OR "obesity childhood" [Title/Abstract] OR "obesity in childhood" [Title/Abstract] OR "child obesity" [Title/Abstract] OR "obesity child" [Title/Abstract] OR "childhood onset obesity" [Title/Abstract] OR "obesity childhood onset" [Title/Abstract] OR "adolescent obesity" [Title/Abstract] OR "obesity adolescent" [Title/Abstract] OR "obesity in adolescence" [Title/Abstract] OR "childhood overweight" [Title/Abstract] OR "overweight childhood" [Title/Abstract] OR "adolescent overweight" [Title/Abstract] OR "overweight adolescent" [Title/Abstract])).

2.2 Study selection

The study selection process was as follows: first, duplicates were excluded from the search records obtained from the database; then, titles and abstracts were read and compared with the inclusion criteria for initial screening; and finally, the remaining articles were screened by full-text review. Inclusion criteria for the studies included: 1) the type of study was a randomized controlled trial; 2) the study was conducted in children and adolescents between the ages of 2 and 18 years with overweight or obesity; 3) the intervention was probiotic supplementation in the experimental group, and the intervention in the control group was the use of a placebo or no intervention; and 4) the study's outcomes included at least one of the following: body weight, BMI, WC, WHR, TC, TG, LDL-C, and HDL-C. Exclusion criteria for studies included: 1) the type of articles were reviews, letters, reports, and other nonrandomized controlled studies; 2) the studies did not contain the required outcome metrics or the data from the studies could not be extracted. The study selection process was conducted independently by two authors, and any disagreements in the results were reelected by consensus. The process of study selection used the Shiny app to generate the PRISMA flow diagram[26].

2.3 Quality assessment

The methodological rigor of candidate studies was critically appraised through application of the Cochrane Risk of Bias tool, following standardized protocols for systematic reviews. This evaluation framework systematically assessed seven core domains of research quality: 1) randomization sequence generation; 2) allocation concealment procedures; 3) participant/personnel blinding implementation; 4) outcome assessment blinding; 5) completeness of outcome data; 6) selective reporting risks; and 7) identification of other potential bias sources. Each methodological domain received categorical ratings through systematic classification: "Low risk" indicated full compliance with best practices, "High risk" denoted substantial methodological concerns, and "Unclear risk" was assigned when reporting insufficiently addressed the criterion. Subsequently, studies underwent stratified quality classification based on their aggregate low-risk domain counts: methodologically limited (fewer than two low-risk domains), moderate rigor (exactly two low-risk domains), and methodologically robust (more than two low-risk domains). This tripartite classification system enabled systematic differentiation of evidence quality while maintaining alignment with established Cochrane guidelines for bias assessment[27]. The

process of quality assessment was carried out independently by the first author and the other researcher.

2.4 Data extraction

The first author and another researcher jointly extracted the data of the selected studies, including the name of the first author, publication year, study location, number of participants, number of genders, average age, follow-up time, intervention measures (dose and type of probiotic supplementation), and outcome indicators. Where discrepancies existed, the corresponding author was consulted to resolve the differences through discussion. Data extraction was performed using a standardized form in Microsoft Excel.

2.5 Statistical analysis

The analytical protocol incorporated rigorous data harmonization and quantitative synthesis methodologies. Continuous outcome measures underwent systematic unit standardization, with conversion of reported standard errors (SE) to standard deviations (SD) through the computational formula $SD = SE\sqrt{n}$, where n represents sample size. Effect size estimation employed weighted mean differences (WMD) with 95% confidence intervals (CI) as the principal comparative metric, appropriate for continuous variable meta-analyses. Heterogeneity quantification utilized the I^2 statistic, with thresholds operationalized per Cochrane guidelines: I^2 exceeding 50% (indicating substantial heterogeneity) triggered application of random-effects models (using inverse-variance (IV) method with between-study variance estimation), while values ≤ 50% warranted IV fixed-effects approaches[28]. Analytical robustness was verified through iterative exclusion methodology, sequentially removing individual studies to assess effect estimate stability across model iterations. Publication bias evaluation implemented complementary regression-based assessments: Egger's linear regression test examined small-study effects[29], while Begg's rank correlation method detected asymmetry patterns[30].

3 Results

3.1 Selection of studies

The systematic literature search yielded an initial pool of 531 publications from three major academic databases, with distribution as follows: PubMed contributed 45 records, Web of Science 244 records, and Scopus 242 records. Following automated deduplication procedures that eliminated 228 redundant entries, the remaining 303 unique articles underwent rigorous preliminary screening. A triage process involving title and abstract evaluation resulted in the exclusion of 290 publications deemed non-relevant to the research objectives. Of the 13 potentially eligible articles identified through this initial screening protocol, one publication became inaccessible during full-text retrieval attempts. The remaining 12 accessible articles progressed to comprehensive full-text review. Subsequent critical appraisal revealed three articles requiring exclusion due to insufficient data availability or methodological reporting deficiencies. This rigorous multi-stage screening process ultimately resulted in nine methodologically sound articles meeting all predetermined inclusion criteria for systematic analysis[20,31–38]. The flow diagram of study selection is shown in Figure 1.

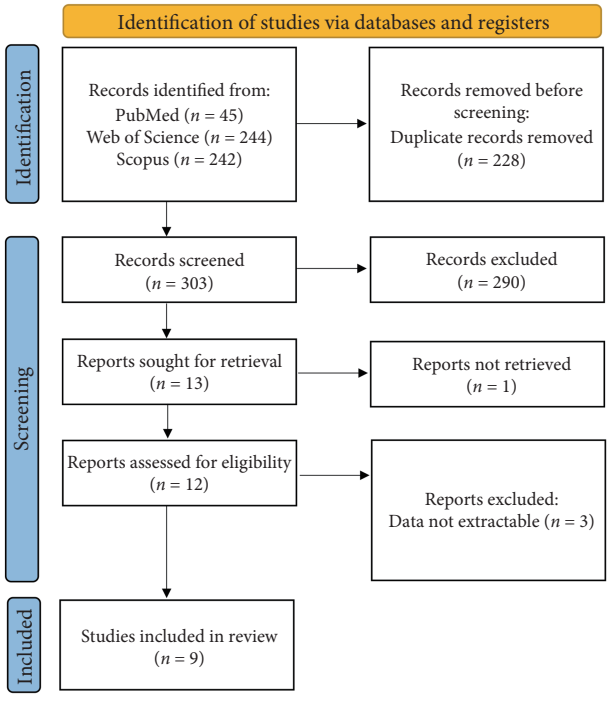

Figure 1 PRISMA flow diagram of study selection process.

3.2 Study characteristics

A total of 464 children and adolescents suffering from overweight, or obesity were involved in the nine included studies, 236 allocated to the probiotic intervention group and 228 in the placebo-controlled comparator group. And the participants were from the United States, China, Turkey, Denmark, Iran, and Bali. The publication years of the articles ranged from 2012 to 2024, and the follow-up periods were eight weeks, 12 weeks, or three months. Eight studies reported the number of participants by gender, and one study did not mentin the number of participants by gender. Table 1 summarizes the characteristics of the included studies.

3.3 Quality assessment of studies

The quality of the included studies was good, and the overall quality of the studies was high, according to the quality assessment results. Figure 2 show detailed information about the quality assessment.

3.4 Effects of probiotics on primary outcomes

The quantitative synthesis incorporated comprehensive outcome data from randomized controlled trials examining probiotic interventions. Complete body weight measurements were available from five studies (Figure 3A), demonstrating non-significant between-group differences (WMD: −3.74; 95% CI: −8.30 to 0.83; $P = 0.11$) with homogeneous effect estimates ($I^2 = 0\%$; $P = 0.52$). Conversely, pooled analysis of five studies reporting BMI (Figure 3B) revealed statistically significant reductions in the probiotic cohort (WMD: −1.57; 95% CI: −2.55 to −0.58; $P = 0.002$), maintaining methodological consistency across trials ($I^2 = 0\%$; $P = 0.77$). Lipid analyses demonstrated divergent heterogeneity patterns. Six studies assessing TC (Figure 3C) showed negligible interventional effects (WMD: 0.01; 95% CI: −0.35 to 0.38; $P = 0.94$) amidst substantial between-study heterogeneity ($I^2 = 87\%$; $P < 0.000\ 01$). Similarly, TG levels from six trials (Figure 3D) exhibited non-significant treatment differences (WMD: −0.12; 95% CI: −0.47 to 0.22; $P = 0.48$) with pronounced variability across

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Table 1 Characteristics of included trials.

Country	Total sample size (male/female)		Mean age (year)		Duration	Intervention		Outcomes	References
	Treatment group	Control group	Treatment group	Control group		Treatment group	Control group		
Iran	17/13	11/19	11.23 ± 2.37	11.07 ± 2.00	8 weeks	Synbiotic capsules (*Lactobacillus coagulans* SC-208 (6×10^9 CFU), *Lactobacillus indicus* HU36 (6×10^9 CFU)), total 12×10^9 CFU	Placebo capsule containing maltodextrin	Body weight, BMI, WC, WHR	[31]
China	14/13	16/10	11.6 ± 0.6	11.2 ± 0.7	3 months	Packages of the supplement (*Lactobacillus salivarius* AP-32 (1×10^9 CFU), *Lactobacillus rhamnosus* bv-77 (1×10^9 CFU), *Bifidobacterium animalis* CP-9 (8×10^9 CFU)), total 10×10^{10} CFU	Packages of the supplement containing the same ingredients without any of the probiotics	Body weight, BMI, WC	[32]
Iran	14/18	18/14	12.7 ± 2.2	12.6 ± 1.7	12 weeks	Probiotic capsule (*Lactobacillus acidophilus* ATCC B3208 (3×10^9 CFU), *Bifidobacterium lactis* DSMZ 32269 (6×10^9 CFU), *Bifidobacterium bifidum* ATCC SD6576 (2×10^9 CFU), *L. rhamnosus* DSMZ 21690 (2×10^9 CFU))	Placebo capsule	WC, TC, TG, LDL-C, HDL-C	[33]
Denmark	11/16	11/12	12.9 ± 1.0	13.4 ± 1.1	12 weeks	*L. salivarius* Ls-33 ATCC SD5208 (1×10^{10} CFU)	Placebo	Body weight, WC, WHR, TC, TG, LDL-C, HDL-C	[34]
Bali	9/18	8/17	14.0 ± 1.0	14.3 ± 1.2	8 weeks	Sachets of probiotic (*Streptococcus thermophilus*, *L. rhamnosus*, *L. acidophilus*, *Bifidobacterium longum*, *B. bifidum* (total 1.25×10^9 CFU))	Placebo containing flour and glucose	TC, TG, LDL-C, HDL-C	[35]
Turkey	12/18	16/15	11.8 ± 3.1	12.4 ± 2.7	12 weeks	Sachets of synbiotic (*L. acidophilus* (4.3×10^8 CFU), *L. rhamnosus* (4.3×10^8 CFU), *B. bifidum* (4.3×10^8 CFU), *B. longum* (4.3×10^8 CFU), *Enterococcus faecium* (8.2×10^8 CFU)), total 2.5×10^9 CFU	Sachets of placebo	Body weight, BMI, WC, WHR, TC, TG, LDL-C, HDL-C	[20]
Iran	29	27	10.75 ± 2.49	10.09 ± 1.93	8 weeks	Synbiotic capsules (*Lactobacillus casei*, *L. rhamnosus*, *S. thermophilus*, *Bifidobacterium breve*, *L. acidophilus*, *B. longum*, *Lactobacillus bulgaricus*), total 2×10^8 CFU	Placebo capsules containing maltodextrine	WC, WHR, TC, TG, LDL-C, HDL-C	[36]
USA	1/3	2/2	15.8 ± 1.8	15.7 ± 1.8	12 weeks	Sachets of symbiotic (*Lactobacillus plantarum* DSM 24730, *L. plantarum* DSM 24731, *L. plantarum* DSM 24735, *L. plantarum* DSM 24801, *L. salivarius* DSM 24800, *Lactobacillus paracasei* DSM 24737, *Lactobacillus delbrueckii* DSM 25998, *Pediococcus pentosaceus* DSM 24734, *B. animalis* DSM 24736, *B. breve* DSM 24732), total 9×10^{11} CFU	Sachets of placebo containing maltose and silicon dioxide	Body weight, BMI	[37]
China	16/14	13/17	13.98 ± 4.25	14.25 ± 4.82	12 weeks	*Bifidobacterium* quadruple live bacteria tablets (*Bifidobacterium infantis*, *L. acidophilus*, *E. faecalis*, *Bacillus cereus*), 0.5 g each tablet	No additional interventions	BMI, WHR, TC, TG, LDL-C, HDL-C	[38]

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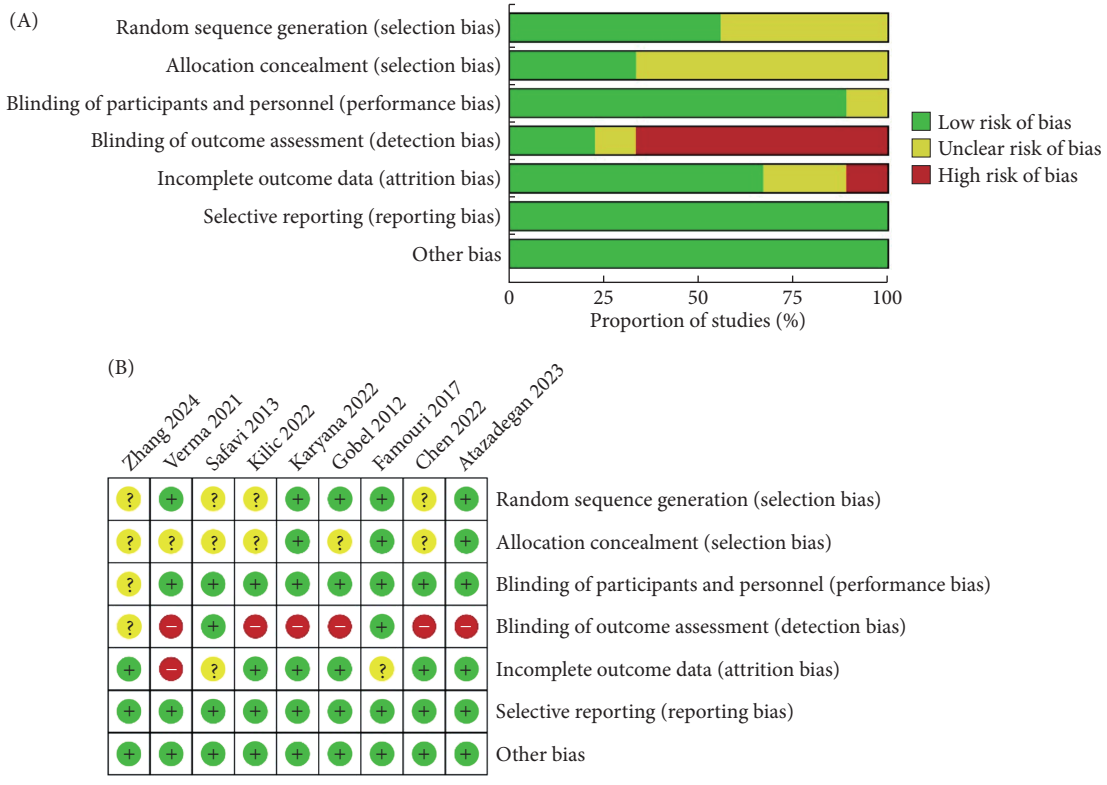


Figure 2 Risk of bias graph. Quality assessment of studies eligible for inclusion was performed using the Cochrane Risk of Bias tool. (A) Risk of bias graph; (B) risk of bias summary. "+" indicates low risk of bias, "−" indicates high risk of bias, and "?" indicates unclear risk of bias.

(A)

Study or subgroup	Probiotics Mean	SD	Total	Placebo/no intervention Mean	SD	Total	Weight (%)	Mean difference IV, Fixed, 95% CI
Atazadegan 2023	54.9	15.15	30	54.89	16.52	30	32.4	0.01 [−8.01, 8.03]
Chen 2022	66.7	21.82	27	66	23.46	26	14.0	0.70 [−11.51, 12.91]
Gobel 2012	81.5	15.9	27	86.5	13.4	23	31.6	−5.00 [−13.12, 3.12]
Kilic 2022	64.4	18.3	30	74.2	21.9	31	20.3	−9.80 [−19.92, 0.32]
Verma 2021	120.8	17.5	4	135.7	30.1	4	1.8	−14.90 [−49.02, 19.22]
Total (95% CI)			118			114	100.0	−3.74 [−8.30, 0.83]

Heterogeneity: $Chi^2 = 3.23$, df = 4 ($P = 0.52$); $I^2 = 0\%$
Test for overall effect: $Z = 1.61$ ($P = 0.11$)

(B)

Study or subgroup	Probiotics Mean	SD	Total	Placebo/no intervention Mean	SD	Total	Weight (%)	Mean difference IV, Fixed, 95% CI
Atazadegan 2023	24.58	3.33	30	25.24	4.35	30	25.2	−0.66 [−2.62, 1.30]
Chen 2022	28.5	5.72	27	29.7	5.1	26	11.4	−1.20 [−4.12, 1.72]
Kilic 2022	26.7	3.7	30	29	5.8	31	16.4	−2.30 [−4.73, 0.13]
Verma 2021	40.9	5.5	4	45.2	5.8	4	1.6	−4.30 [−12.13, 3.53]
Zhang 2024	27.15	2.92	30	28.95	2.85	30	45.4	−1.80 [−3.26, −0.34]
Total (95% CI)			121			121	100.0	−1.57 [−2.55, −0.58]

Heterogeneity: $Chi^2 = 1.80$, df = 4 ($P = 0.77$); $I^2 = 0\%$
Test for overall effect: $Z = 3.12$ ($P = 0.002$)

(C)

Study or subgroup	Probiotics Mean	SD	Total	Placebo/no intervention Mean	SD	Total	Weight (%)	Mean difference IV, Random, 95% CI
Famouri 2017	3.76	1.24	32	2.73	0.61	32	14.8	1.03 [0.55, 1.51]
Gobel 2012	4.15	0.71	27	4.12	0.56	23	16.7	0.03 [−0.32, 0.38]
Karyana 2022	3.97	0.6	27	4.41	0.64	25	16.9	−0.44 [−0.78, −0.10]
Kilic 2022	4.22	0.78	30	4.07	0.91	31	15.6	0.15 [−0.27, 0.57]
Safavi 2013	4.22	0.32	29	4.16	0.36	27	18.9	0.06 [−0.12, 0.24]
Zhang 2024	4.07	0.59	30	4.67	0.67	30	17.2	−0.60 [−0.92, −0.28]
Total (95% CI)			175			168	100.0	0.10 [−0.35, 0.38]

Heterogeneity: $Tau^2 = 0.18$; $Chi^2 = 38.48$, df = 5 ($P < 0.000\,01$); $I^2 = 87\%$
Test for overall effect: $Z = 0.08$ ($P = 0.94$)

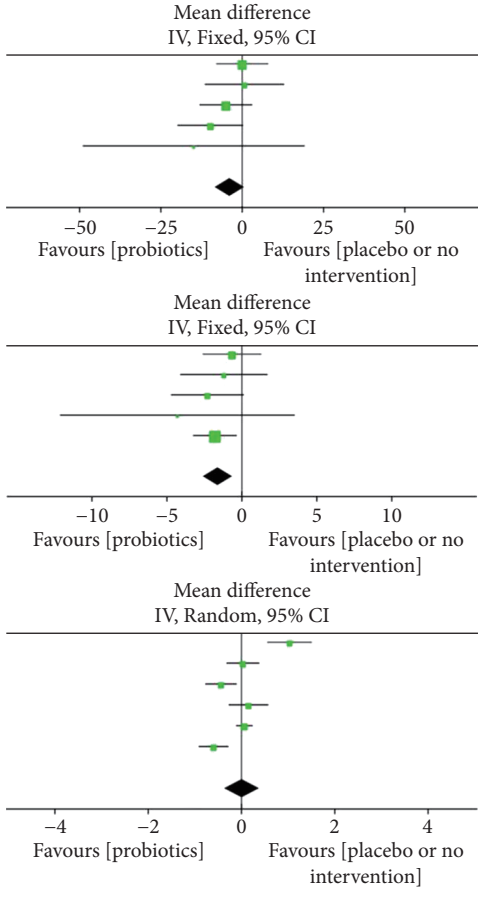


Figure 3 Forest plot of the effect of probiotics on (A) body weight, (B) BMI, (C) TC, (D) TG.

(D)

Study or subgroup	Probiotics Mean	SD	Total	Placebo/no intervention Mean	SD	Total	Weight (%)	Mean difference IV, Random, 95% CI
Famouri 2017	1.14	0.51	32	1.04	0.22	32	18.2	0.10 [−0.09, 0.29]
Gobel 2012	1.04	0.74	27	1.16	0.65	23	15.5	−0.12 [−0.51, 0.27]
Karyana 2022	1.15	0.61	27	1.46	0.68	25	16.0	−0.31 [−0.66, 0.04]
Kilic 2022	1.37	0.7	30	1.18	1.07	31	14.4	0.19 [−0.26, 0.64]
Safavi 2013	1.49	0.43	29	1.3	0.31	27	18.1	0.19 [−0.01, 0.39]
Zhang 2024	1.43	0.35	30	2.19	0.52	30	17.8	−0.76 [−0.98, −0.54]
Total (95% CI)			175			168	100.0	−0.12 [−0.47, 0.22]

Heterogeneity: Tau² = 0.16; Chi² = 48.82, df = 5 (P < 0.000 01); I^2 = 90%
Test for overall effect: Z = 0.71 (P = 0.48)

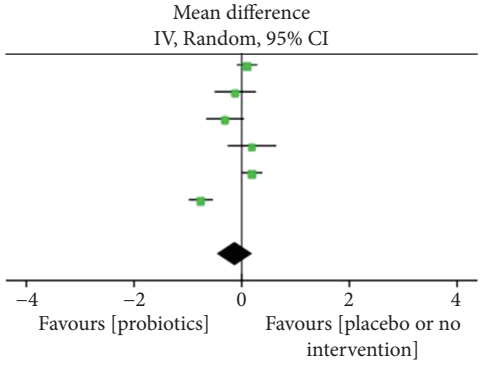


Figure 3 (*Continued*)

studies (I^2 = 90%; P < 0.000 01). This meta-analytical evidence substantiates probiotics' capacity to significantly modulate BMI parameters, while demonstrating statistically inconclusive effects on body weight regulation and lipid metabolism outcomes. The observed heterogeneity in lipid measures suggests potential moderating variables requiring further investigation.

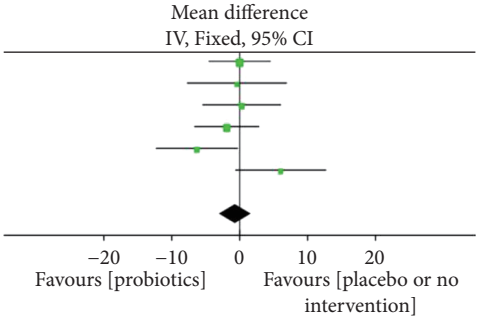
3.5 Effect of probiotics on secondary outcomes

The meta-analytical evaluation of anthropometric and lipid parameters incorporated rigorous examination of body composition metrics across multiple randomized trials. Six studies providing complete WC data (Figure 4A) demonstrated non-significant interventional effects (WMD: −0.63; 95% CI: −2.93 to 1.67; P = 0.59) with acceptable heterogeneity thresholds (I^2 = 35%; P = 0.18). Similarly, pooled analysis of five trials reporting WHR outcomes (Figure 4B) revealed negligible between-group differences (WMD: −0.01; 95% CI: −0.02 to 0.01; P = 0.34), exhibiting methodological consistency across studies (I^2 = 0%; P = 0.48). Lipoprotein analyses demonstrated variable heterogeneity patterns. Six trials evaluating LDL-C (Figure 4C) showed clinically insignificant treatment effects (WMD: 0.04; 95% CI: −0.12 to 0.20; P = 0.63) amidst moderate between-study variability (I^2 = 63%;

(A)

Study or subgroup	Probiotics Mean	SD	Total	Placebo/no intervention Mean	SD	Total	Weight (%)	Mean difference IV, Fixed, 95% CI
Atazadegan 2023	80	8	30	80	10	30	25.2	0.00 [−4.58, 4.58]
Chen 2022	89.3	14.55	27	89.7	12.75	26	9.8	−0.40 [−7.76, 6.96]
Famouri 2017	80.3	15.1	32	80	7.2	32	15.7	0.30 [−5.50, 6.10]
Gobel 2012	101.4	10.5	27	103.3	6.6	23	23.0	−1.90 [−6.69, 2.89]
Kilic 2022	86.8	8.7	30	93.1	14.7	31	14.5	−6.30 [−12.34, −0.26]
Safavi 2013	82.89	15.2	29	76.85	9.99	27	11.8	6.04 [−0.65, 12.73]
Total (95% CI)			175			169	100.0	−0.63 [−2.93, 1.67]

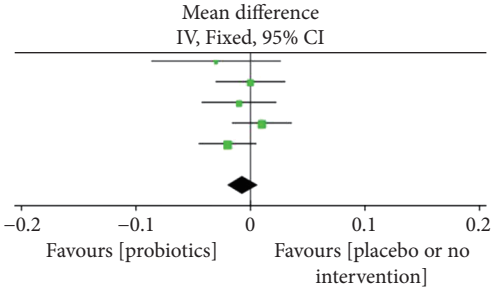
Heterogeneity: Chi² = 7.65, df = 5 (P = 0.18); I^2 = 35%
Test for overall effect: Z = 0.54 (P = 0.59)



(B)

Study or subgroup	Probiotics Mean	SD	Total	Placebo/no intervention Mean	SD	Total	Weight (%)	Mean difference IV, Fixed, 95% CI
Atazadegan 2023	0.84	0.15	30	0.87	0.05	30	5.8	−0.03 [−0.09, 0.03]
Gobel 2012	1	0.06	27	1	0.05	23	20.1	0.00 [−0.03, 0.03]
Kilic 2022	0.88	0.06	30	0.89	0.07	31	17.5	−0.01 [−0.04, 0.02]
Safavi 2013	0.35	0.05	29	0.34	0.05	27	27.3	0.01 [−0.02, 0.04]
Zhang 2024	0.93	0.05	30	0.95	0.05	30	29.2	−0.02 [−0.05, 0.01]
Total (95% CI)			146			141	100.0	−0.01 [−0.02, 0.01]

Heterogeneity: Chi² = 3.50, df = 4 (P = 0.48); I^2 = 0%
Test for overall effect: Z = 0.95 (P = 0.34)



(C)

Study or subgroup	Probiotics Mean	SD	Total	Placebo/no intervention Mean	SD	Total	Weight (%)	Mean difference IV, Random, 95% CI
Famouri 2017	2.11	0.61	32	2.03	0.33	32	18.6	0.08 [−0.16, 0.32]
Gobel 2012	2.54	0.6	27	2.45	0.44	23	15.8	0.09 [−0.20, 0.38]
Karyana 2022	2.57	0.62	27	2.83	0.63	25	13.3	−0.26 [−0.60, 0.08]
Kilic 2022	2.81	0.74	30	2.61	0.92	31	10.3	0.20 [−0.22, 0.62]
Safavi 2013	2.77	0.05	29	2.58	0.05	27	30.6	0.19 [0.16, 0.22]
Zhang 2024	3.12	1.01	30	3.41	0.39	30	11.4	−0.29 [−0.68, 0.10]
Total (95% CI)			175			168	100.0	0.04 [−0.12, 0.20]

Heterogeneity: Tau² = 0.02; Chi² = 13.63, df = 5 (P = 0.02); I^2 = 63%
Test for overall effect: Z = 0.48 (P = 0.63)

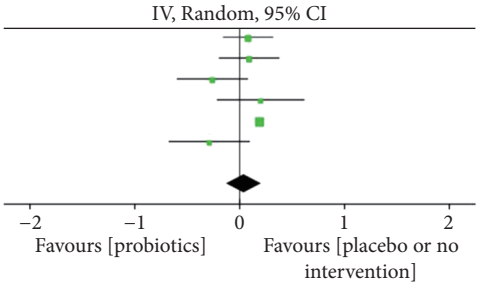


Figure 4 Forest plot of the effect of probiotics on (A) WC, (B) WHR, (C) LDL-C, (D) HDL-C.

(D)

Study or subgroup	Probiotics Mean	SD	Total	Placebo/no intervention Mean	SD	Total	Weight (%)	Mean difference IV, Random, 95% CI
Famouri 2017	1.21	0.39	32	0.95	0.26	32	12.6	0.26 [0.10, 0.42]
Gobel 2012	1.05	0.19	27	1.06	0.22	23	17.7	−0.01 [−0.12, 0.10]
Karyana 2022	1.12	0.19	27	1.14	0.24	25	17.2	−0.02 [−0.14, 0.10]
Kilic 2022	1.12	0.16	30	1.16	0.27	31	18.2	−0.04 [−0.15, 0.07]
Safavi 2013	1.03	0.48	29	1.09	0.52	27	6.5	−0.06 [−0.32, 0.20]
Zhang 2024	1.18	0.08	30	1.1	0.06	30	27.8	0.08 [0.04, 0.12]
Total (95% CI)			175			168	100.0	0.04 [−0.04, 0.11]

Heterogeneity: Tau² = 0.01; Chi² = 14.11, df = 5 (P = 0.01); I^2 = 65%
Test for overall effect: Z = 1.00 (P = 0.32)

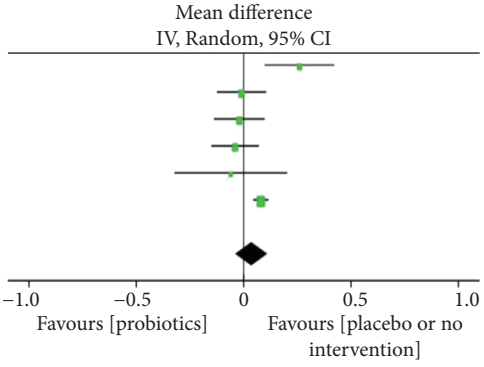


Figure 4 (*Continued*)

P = 0.02). Parallel findings emerged from six studies assessing HDL-C (Figure 4D), with non-significant outcome differences (WMD: 0.04; 95% CI: −0.04 to 0.11; P = 0.32) and marked heterogeneity across trials (I^2 = 65%; P = 0.01). Collectively, these quantitative syntheses indicate probiotic interventions failed to demonstrate statistical significance in modulating central adiposity markers (WC, WHR) or lipoprotein metabolism (LDL-C, HDL-C). The observed heterogeneity in lipid parameters suggests potential moderating variables, possibly related to methodological variations in lipid quantification protocols or differential baseline metabolic profiles across study populations.

3.6 Publication bias and sensitivity analysis

The methodological evaluation of publication bias employed complementary statistical approaches, incorporating both Egger's regression asymmetry test and Begg's rank correlation method. Comprehensive assessment across all outcome measures revealed no substantive evidence of publication bias, with detailed quantitative outputs systematically tabulated in Table 2. A systematic sensitivity evaluation was performed through sequential exclusion modeling, whereby individual studies were iteratively removed from the analytical cohort. This protocol demonstrated methodological robustness, as evidenced by stable pooled effect estimates maintaining unchanged magnitude and directionality across all iterations. Figure 5 presents the detailed results of the sensitivity analyses.

The invariant nature of effect size parameters under varying exclusion conditions substantiates the analytical framework's resilience to individual study influences. These dual validation procedures encompassing bias detection and sensitivity verification collectively affirm the methodological integrity of the meta-analytical findings. The observed stability in effect estimates suggests that pooled results withstand critical appraisal and remain unaffected by potential outlier influences or selective reporting patterns within the included studies.

Table 2 Publication bias.

Outcomes	Egger's test		Begg's test		Publication bias (Yes/No)
	t	P	z	P	
Body weight	−0.63	0.571	0.73	0.462	No
BMI	−0.67	0.550	0.73	0.462	No
TC	0.30	0.782	1.13	0.260	No
TG	−0.22	0.836	0.00	1.000	No
WC	0.43	0.689	0.00	1.000	No
WHR	−0.71	0.528	0.24	0.806	No
LDL-C	−2.52	0.066	0.38	0.707	No
HDL-C	−0.76	0.490	0.38	0.707	No

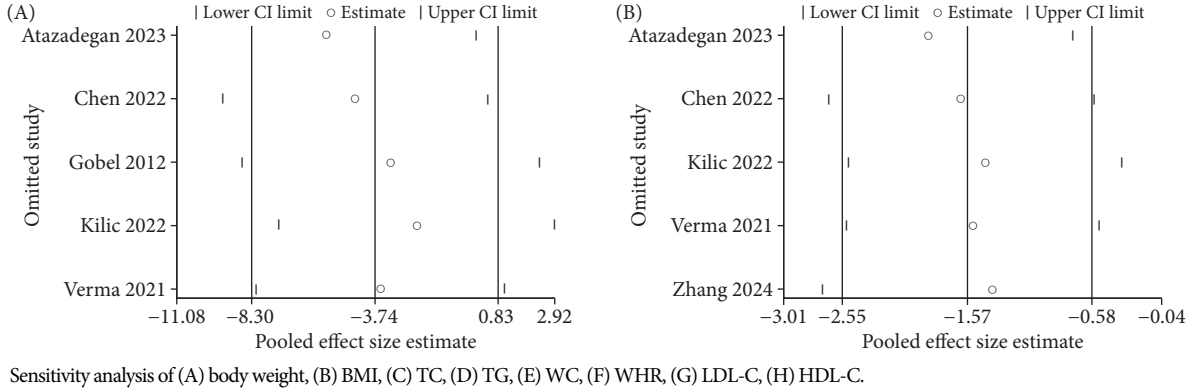


Figure 5 Sensitivity analysis of (A) body weight, (B) BMI, (C) TC, (D) TG, (E) WC, (F) WHR, (G) LDL-C, (H) HDL-C.

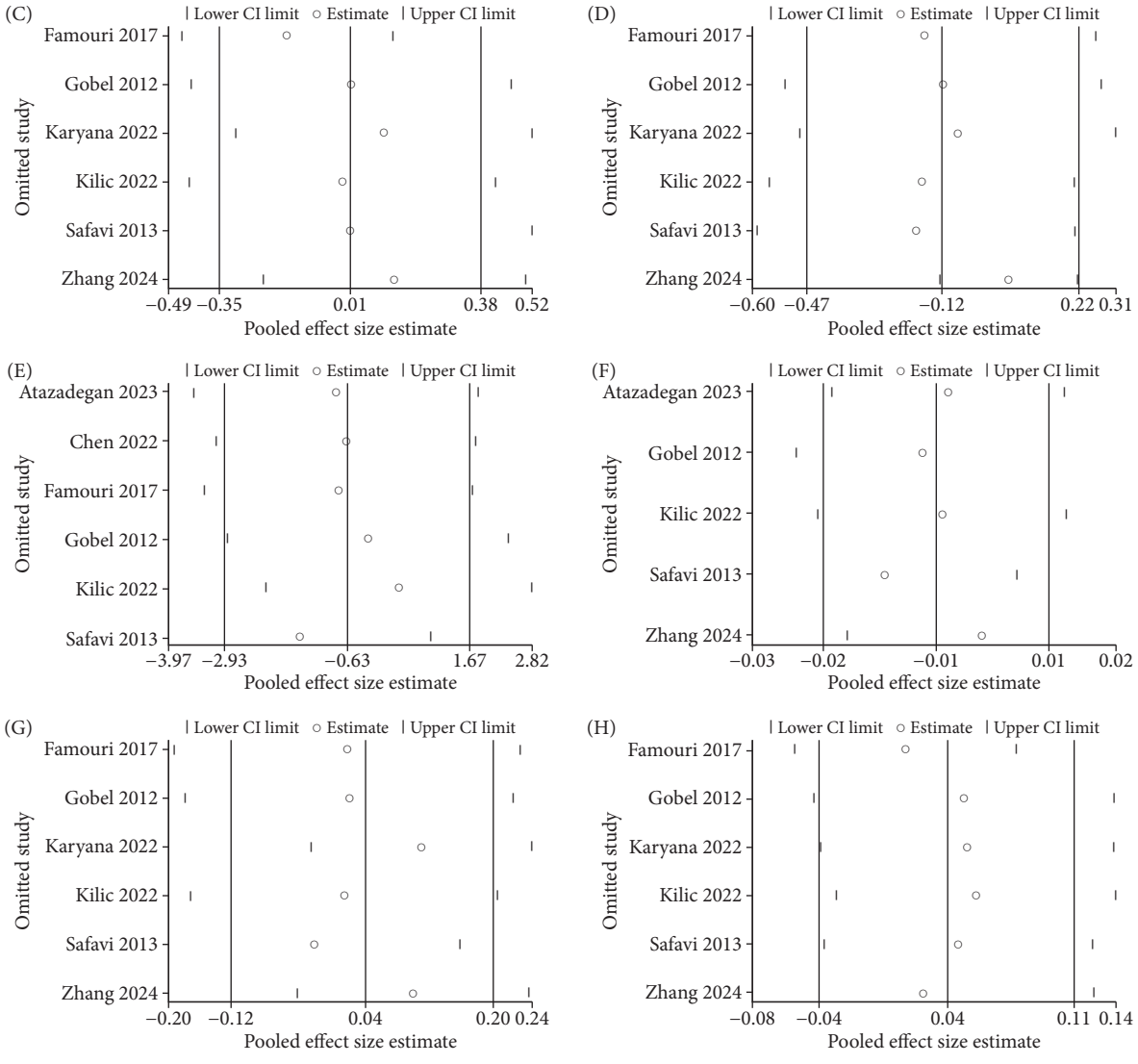


Figure 5 (*Continued*)

4 Discussion

Probiotics, as a novel type of intestinal microbiota modulator in recent years, have attracted considerable attention from researchers regarding their regulatory effects on obesity. This meta-analysis investigated the therapeutic effects of probiotic supplementation on anthropometric indices and serum lipid parameters in pediatric populations with obesity, employing rigorous quantitative synthesis of randomized controlled trials to evaluate clinical intervention outcomes. Our analysis of nine randomized controlled trials involving 464 participants showed that probiotic supplementation significantly reduced BMI and may have potential clinical value in controlling BMI in obese children and adolescents; however, there was no significant effect on other outcomes assessed, including body weight, WC, WHR, and lipid levels, and there was a limited role in improving other obesity-related indicators. The results of the study showed that no significant heterogeneity was observed in the meta-analysis of BMI, suggesting a high degree of reliability of the results; substantial heterogeneity existed in the meta-analysis of some metabolic indicators, such as TC and TG, which may be related to the heterogeneity of the probiotic formulations, intervention durations, participant characteristics, and dietary habits of the different studies. The methodological validity of these conclusions is reinforced by systematic evaluation of publication bias and sensitivity analyses, confirming the analytical stability under varying model specifications.

According to the results of the study, probiotics can significantly reduce BMI in obese children and adolescents, which may be relevant to the regulation of the gut microbiome and the improvement of metabolic function by probiotics. It has been shown that the composition of gut flora and metabolites affects the progression of obesity and obesity-related diseases[39], and probiotics exert their anti-obesity effects by regulating the gut flora, reducing insulin resistance, and making people more satiated[40], thus improving BMI. The significant reduction in BMI observed in our meta-analysis may also be driven by a synergistic modulation of the Microbiota-Gut-Brain axis, involving both metabolic and neuro-immune pathways. As highlighted by Caesar[41], probiotic-derived short-chain fatty acids (SCFAs), particularly acetate and propionate, act as ligands for G-protein-coupled receptors 41/43 on enteroendocrine L-cells, stimulating the secretion of glucagon-like peptide-1 and peptide YY to enhance satiety. Complementing this metabolic signaling, recent evidence reviewed by Lu et al.[42] suggests that gut microbiota dysbiosis is intrinsically linked to

neuroinflammation and hypothalamic-pituitary-adrenal (HPA) axis hyperactivity—mechanisms that drive both metabolic and mood disorders. Probiotics may mitigate obesity-related low-grade inflammation by upregulating tight junction proteins (e.g., zonula occludens-1) and inhibiting the Toll-like receptor 4/nuclear factor κB pathway, thereby reducing the translocation of lipopolysaccharides and subsequent microglial activation in brain regions regulating appetite[42]. Furthermore, the gut microbiota regulates the metabolism of tryptophan and the synthesis of serotonin (5-hydroxytryptamine), a critical neurotransmitter for satiety and mood regulation[42]. Therefore, the observed BMI improvement may result not only from direct metabolic regulation but also from the restoration of serotonergic signaling and the attenuation of stress-induced HPA axis activation, which are often dysregulated in pediatric obesity.

However, this result needs to be interpreted with caution because BMI, as an important indicator of obesity, reflects the overall health status, but it does not directly reveal changes in body fat percentage or muscle mass[43]. But the positive effects of probiotics on BMI may still offer a novel avenue for the nutritional intervention of obese children and adolescents. According to the definition proposed by Ryder et al.[44], a reduction in BMI holds significant clinical relevance for children with severe obesity, and it serves as a pivotal factor in ameliorating comorbidities such as dyslipidemia, hypertension, and non-alcoholic fatty liver disease. Unlike adults, where weight maintenance is often insufficient, effective BMI reduction in youth disrupts the strong "tracking" of obesity into adulthood, thereby mitigating the compounded risks of early mortality and chronic disease accumulation. Furthermore, the practical value of this intervention is amplified when interpreted through the lens of pubertal development. Dorenbos et al.[45] elucidate that puberty is inherently characterized by a state of "transient physiological insulin resistance". However, in the presence of high BMI *Z*-scores, this transient adaptation risks evolving into persistent, pathological insulin resistance and type 2 diabetes. Crucially, BMI *Z*-score is identified as a primary determinant of this metabolic persistence. Therefore, the BMI reduction achieved in our study likely serves a disease-modifying role: by alleviating metabolic burden during this critical window of plasticity, the intervention facilitates the safe resolution of pubertal insulin resistance, preventing the transition to permanent metabolic dysfunction.

WC and WHR can serve as complementary data to BMI for predicting the incidence of cardiovascular risk events in obese populations[46]. Additionally, abdominal obesity often leads to social and psychological issues among children and adolescents[47]. Our research findings indicate that probiotics did not have a significant effect on anthropometric indicators such as body weight, WC, and WHR in obese children and adolescents, which contradicts the results of studies conducted on adult obesity[17,48], and the discrepancy may be due to the specificity of the physiological state of children and adolescents. Pediatric populations represent a distinct developmental cohort undergoing rapid somatic and neurocognitive maturation, and the changes in body weight and height brought about by physical growth and development may mask the intervention effects of probiotics on weight, WC, and WHR. Also, there are large differences in the gut flora groups of children and adolescents[49], which may also contribute to the lack of significant intervention effects of probiotics. The results of this part of the study demonstrate concordance with the results of Duan et al.[24], but there is a contradiction in the assessment of the effect of BMI with the studies of Li et al.[21] and Mohammadi et al.[23], which may be related to the differences in the design of the included studies, the differences in the characteristics of the samples, and the differences in the intervention protocols.

The divergent therapeutic effects of probiotics observed between pediatric and adult populations may be rooted in the fundamental structural and functional differences of their respective gut microbiomes. Unlike the relatively stable and resilient ecosystem found in adults, recent longitudinal evidence confirms that the pediatric gut microbiota is a dynamic, transitional ecosystem that has not yet reached adult-like complexity. Roswall et al.[50] demonstrated that even at 5 years of age, the gut microbiota retains a significantly lower alpha diversity compared to adults and lacks key "late-colonizing" taxa, such as Methanobrevibacter and members of the Christensenellaceae family, which are associated with high community richness and metabolic health in adults. This structural immaturity suggests that the pediatric microbiome possesses higher ecological plasticity, potentially making it more responsive to microbial interventions but also more susceptible to heterogeneity compared to the resistance often seen in the stable adult microbiome. Furthermore, the metabolic "engine" of the pediatric microbiome appears to be functionally distinct from that of adults. Radjabzadeh et al.[51] revealed that children (aged 9–12 years) exhibit a specific functional profile enriched in pathways related to glycan degradation and the biosynthesis of vitamins essential for development (e.g., folate, riboflavin), whereas adults are enriched in pathways for carbohydrate metabolism and the biosynthesis of thiamine and pantothenate. Children showed a predominance of catabolic pathways (e.g., amino acid degradation), contrasting with the biosynthetic dominance observed in adults. The pediatric microbiome is enriched in colonization-related functions, such as flagellar assembly and biofilm formation. These findings imply that probiotic strains administered to children enter an ecosystem prioritized for growth and colonization rather than metabolic maintenance. Consequently, the lack of significant lipid-lowering effects in our analysis might be attributed to the absence of specific adult-associated functional guilds (e.g., *Blautia* and Christensenellaceae) that mediate lipid metabolism, or because the metabolic output of probiotics in a "catabolic" pediatric environment differs from that in a "biosynthetic" adult host.

Dyslipidemia is one of the key risk factors for cardiovascular diseases, and metabolic disturbances caused by obesity are a major contributing factor to the development of dyslipidemia[52]. Moreover, obesity during childhood or adolescence is positively correlated with dyslipidemia in adulthood[53]. Probiotics have been shown to ameliorate obesity-related lipid metabolism indices in obese adults[54], whereas our study found that probiotics did not produce significant improvements in lipid metabolism indices in obese pediatric populations. The finding of this part of the study are consistent with those of Mohammadi et al.[23], but contradictory to the studies of Zhang et al.[22] and Li et al.[21]. This discrepancy may be attributed to several factors: firstly, it has been shown that probiotics significantly increase the abundance of intestinal flora after 3 months of intervention[55], whereas in most of our included studies, the relatively short duration of the intervention (8–12 weeks) may not have been sufficient to observe meaningful changes in lipid metabolism indices; and, secondly, the effect of probiotics on lipid metabolism indices may also be related to the dietary composition. A study by Wastyk et al.[56] found that differences in the effect of probiotics on patients were consistent with dietary differences, and

differences in diet between the children and adolescents included in the study may also contribute to the greater heterogeneity of the findings. The substantial heterogeneity in lipid outcomes (TC and TG) and secondary anthropometric measures across the included trials likely reflects the complex interplay between specific probiotic strains and host environmental factors. Caesar[41] emphasizes that probiotic efficacy is highly strain-dependent, with different strains exhibiting distinct capabilities in SCFA production and immune modulation. Moreover, diet acts as a dominant modulator, accounting for over 20% of inter-individual microbiome variability[41], which may confound intervention outcomes across different cultural dietary patterns. Additionally, Lu et al.[42] highlight that the baseline inflammatory status and "leaky gut" severity can vary significantly among individuals, influencing their responsiveness to microbial interventions. Factors such as genetic variations, medication use, and lifestyle habits further complicate the host-microbiota interaction[42]. For instance, the capacity of the microbiota to metabolize dietary fibers into bioactive metabolites is often reduced in dysbiotic states[42]. Consequently, the lack of significant lipid improvements in some groups may be attributed to insufficient intervention durations to reverse established metabolic dysregulation or the use of strains that lack specific lipid-lowering functional traits. This supports our qualitative assessment that standardized interventions considering both strain specificity and dietary background are essential for future pediatric trials.

4.1 Strengths and limitations

This investigation employed rigorous meta-analytical protocols to systematically evaluate probiotic interventions across anthropometric and metabolic parameters in pediatric obesity. The methodological framework was strengthened through comprehensive sensitivity evaluations, including iterative exclusion testing and publication bias assessments, which confirmed result stability across analytical models.

Notable constraints emerged from critical appraisal of the evidence base. Significant heterogeneity persisted in lipid metabolism outcomes (I^2 = 63%–90%), potentially reflecting variability in probiotic formulations, dietary covariates, or metabolic assessment protocols. Temporal limitations were evident across included trials, with maximum intervention durations of three months precluding longitudinal outcome assessments. The evidence pool remains constrained by both quantitative and qualitative parameters: only nine methodologically rigorous randomized controlled trials met inclusion criteria, reflecting scarcity of pediatric-specific probiotic research meeting standards. The rigorous exclusion of studies with mixed adult-pediatric data or low methodological quality resulted in a limited number of included trials (n = 9). This scarcity underscores a critical gap in the current literature. Consequently, there is an urgent need for future research to prioritize large-scale, multi-center, randomized controlled trials specifically designed for pediatric populations. These future studies should rigorously standardize probiotic strains and intervention durations to minimize heterogeneity, thereby providing more robust evidence for clinical guidelines. Furthermore, due to the limited sample size (n = 464), the study may lack the power to detect smaller effect sizes in secondary outcomes, and thus the negative results should be interpreted with caution (potential type II error).

These constraints collectively necessitate cautious interpretation of null findings while underscoring urgent requirements for extended-duration trials incorporating standardized probiotic regimens, harmonized metabolic monitoring protocols, and stratified analyses of microbial strain-specific effects. Future investigations should prioritize protocol standardization to enable robust evaluation of sustained metabolic impacts and dose-response relationships in children and adolescents.

4.2 Implications for practice

The synthesized evidence positions probiotic interventions as a viable adjunctive therapy for BMI management in pediatric obesity, demonstrating statistically significant reductions in comparison with placebo controls (WMD: −1.57; 95% CI: −2.55 to −0.58). These findings suggest potential integration of probiotic regimens into multimodal obesity treatment frameworks, ideally synergized with evidence-based lifestyle modification protocols addressing dietary habits and physical activity.

Clinical implementation requires judicious risk-benefit evaluation, particularly given documented safety concerns regarding probiotic administration in immunocompromised populations[57]. The therapeutic complexity of probiotics encompassing strain-specific effects, dose-response variability, and host-microbiome interactions necessitates rigorous pretreatment assessment of patient comorbidities and immune status.

4.3 Implications for research

There are relatively few high-quality studies on the effects of probiotics in obese children and adolescents, which may also contribute to the fact that most of the effects that have been demonstrated in adult obesity do not have a corresponding effect in children and adolescents, and therefore larger randomized controlled trials are needed to confirm the effects of probiotics. Also, the influence of different factors on probiotic efficacy needs to be further refined to delineate evidence-based administration protocols for optimizing therapeutic outcomes in pediatric obesity management.

5 Conclusion

This systematic review employed rigorous meta-analytical methods to evaluate probiotic interventions in pediatric obesity management. Pooled evidence from randomized controlled trials demonstrated statistically significant reductions in BMI among probiotic-treated cohorts in comparison with placebo controls (WMD: −1.57; 95% CI: −2.55 to −0.58), establishing BMI improvement as the principal therapeutic benefit. However, comprehensive anthropometric and metabolic analyses revealed non-significant intervention effects across multiple secondary endpoints: body weight regulation (WMD: −3.74; P = 0.11), central adiposity markers (WC: WMD: −0.63, P = 0.59; WHR: WMD: −0.01, P = 0.34), and lipid profile parameters (TC: WMD: 0.01, P = 0.94; TG: WMD: −0.12, P = 0.48; LDL-C: WMD: 0.04, P = 0.63; HDL-C: WMD: 0.04, P = 0.32).

The current evidence base remains constrained by limited pediatric-focused trials (n = 9 studies meeting inclusion criteria) and substantial heterogeneity in lipid outcome measurements (I^2 = 63%–90%). While these findings suggest the metabolic effects of probiotics on BMI modulation, the null results across other endpoints underscore the necessity for expanded clinical investigations. Future research should prioritize standardized protocols for probiotic strain selection, intervention duration, and metabolic outcome assessment to elucidate potential dose-dependent effects or strain-specific therapeutic mechanisms in children and adolescents.

Conflict of interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Data availability

All data analyzed during this study are extracted from the original publications included in this systematic review, which are fully cited in the references.

Author contributions

Guodong Yu: Data curation, formal analysis, writing-original draft. Lifei Tang: Data curation, investigation. Zeyuan Kang: Data curation, investigation. Xiaojiao Zheng: Software, formal analysis. Xiaoqun Zeng: Writing-review & editing. Yangying Sun: Writing-review & editing. Daodong Pan: Software, formal analysis. Maolin Tu: Validation, resources, funding acquisition.

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