BMC Cardiovascular Disorders

SYSTEMATIC REVIEW

Open Access

The effect of glucomannan supplementation on lipid profile in adults: a GRADE-assessed systematic review and meta-analysis

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Abstract

Background Glucomannan has been studied for various health benefits, but its effects on lipid profile in adults are not well understood. This meta-analysis aims to evaluate the impact of glucomannan supplementation on serum/plasma levels of total cholesterol (TC), low-density lipoprotein cholesterol (LDL-C), triglyceride (TG), high-density lipoprotein cholesterol (HDL-C), Apo B1, Apo A1, APO-B/ A1 ratio, and LDL-C/ HDL-C in adults.

Methods A comprehensive search was conducted across Scopus, PubMed, Embase, and Web of Science from inception to June 2024 to identify randomized controlled trials (RCTs) assessing glucomannan supplementation on lipid profile in adults. Data were extracted and analyzed using random effects model to determine the standardized mean differences (SMDs) and 95% confidence intervals (CIs) for each biomarker.

Results Glucomannan supplementation significantly decreased TC (SMD: -3.299; 95% CI: -4.955, -1.664, $P < 0.001$; $I^2 = 95.41\%$, P-heterogeneity < 0.001), LDL-C (SMD: -2.993; 95% CI: -4.958, -1.028; $P = 0.006$; $I^2 = 95.49\%$, P-heterogeneity < 0.001), and Apo B1 (SMD: -2.2; 95% CI: -3.58, -0.82; $P = 0.01$). However, glucomannan did not alter the levels of TG (SMD: -0.119; 95% CI: -1.076, 0.837, $P = 0.789$; $I^2 = 91.63\%$, P-heterogeneity < 0.001), Apo A1 (SMD: -0.48; 95% CI: -6.27, 5.32; $P = 0.76$), APO-B/ A1 ratio (SMD: -1.15; 95% CI: -2.91, 0.61; $P = 0.11$), and LDL-C/ HDL-C ratio (SMD: -2.2; 95% CI: -7.28, 2.87; $P = 0.2$).

Conclusions Glucomannan supplementation has a beneficial effect on the level of TC and LDL-C.

Keywords Glucomannan, Lipid profile, Lipoprotein, Dyslipidemia, Meta-analysis, Systematic review

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Introduction

According to the World Health Organization, cardiovascular diseases (CVDs) are the leading cause of death worldwide, accounting for 17.9 million deaths annually, which constitutes over 40% of all global deaths [1, 2]. Consequently, the prevention of CVD is a critical global health challenge with significant implications for both healthcare systems and the economy. Dyslipidemia, a clinical condition characterized by abnormal levels of lipids in the blood, is a key contributor to the development of CVD. This condition includes imbalances in high-density lipoprotein cholesterol (HDL-C), low-density lipoprotein cholesterol (LDL-C), total cholesterol (TC), and triglycerides (TG) [3]. Herbal medicines and dietary fibers have gained attention as promising nutritional strategies for managing dyslipidemia, with a growing number of individuals seeking natural and safe treatments for CVD [4, 5].

Glucomannan [6], an important dietary fiber, has recently garnered interest for its potential role in CVD management. Known for its exceptionally high viscosity—five times that of β-glucan and guar gum—glucomannan is considered one of the most viscous dietary fibers available. It is primarily sourced from the root of the *Amorphophallus konjac* tuber (konjac)[6]. The potential mechanisms by which glucomannan may help treat hyperlipidemia include inhibiting the absorption of bile acids and cholesterol in the intestine and reducing lipid synthesis [7].

Despite the increasing number of studies on konjac glucomannan, there is inconsistency in the reported effects on lipid profiles. While some studies indicate positive effects [8–10], while others do not [11–13]. Previous meta-analyses conducted in 2008 [14] and 2017 [6] evaluated the impact of glucomannan on lipid profiles. However, these analyses were not exclusively focused on adults and omitted some relevant trials.

Given the conflicting findings from previous clinical trial studies and the absence of a comprehensive meta-analysis, this meta-analysis aims to provide a comprehensive synthesis of the current evidence from randomized controlled trials (RCTs) on the effects of glucomannan supplementation on lipid profile in adults. By critically assessing the pooled data, this article seeks to elucidate the clinical efficacy of glucomannan as an adjunct therapy in management of dyslipidemia.

Methods

The current study was designed, performed, and reported in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses guideline (PRISMA) [15].

Search strategy

A comprehensive search was conducted across the Scopus, PubMed, Embase, and Web of Science databases, covering the period from their inception to June 2024. The search strategy employed a combination of Medical Subject Headings (MeSH), keywords, and subject terms, including the following: ((1–6)-alpha-glucomannan OR glucomannan OR Amorphophallus OR konjac OR konjac mannan) AND (randomized controlled trial OR controlled clinical trial OR random OR placebo OR assignment OR controlled trial OR Clinical Trial OR trial OR crossover procedure OR double blinded). The complete search strategy is detailed in Supplementary Table 1. No restrictions were placed on publication date, language, or other filters. In addition to searching for published studies, we implemented a comprehensive strategy to identify unpublished studies and grey literature. In order to find further articles, the reference lists of included research and available reviews were examined. We searched specialized databases and platforms that archive grey literature, including ClinicalTrials.gov and WHO International Clinical Trials Registry Platform. We also explored preprint servers like arXiv, bioRxiv, and medRxiv for early versions of studies that had not yet undergone peer review or been formally published.

Study selection

Endnote version 20 was used to import and deduplicate all citations identified through the database searches. The titles and abstracts of the articles were then independently reviewed by two researchers (AHM and VM). Following this, the full text of each relevant study was retrieved and assessed according to the inclusion and exclusion criteria based on PICOS format. The inclusion criteria were: (1) randomized controlled trials (RCTs) with either parallel or crossover designs; (2) studies investigating the effect of glucomannan on lipid levels in adults; and (3) studies with a design ensuring that the only difference between the glucomannan and control groups was the intervention. The exclusion criteria included duplicate publications, in vivo studies, trials in which glucomannan was administered in combination with other ingredients, studies with a follow-up period of less than two weeks, research involving children and adolescents, and publications lacking sufficient data for a meta-analysis. Any disagreements regarding article selection were resolved through discussion between the reviewers.

Data extraction

Data extraction was carried out by two independent reviewers (AHM and VM). The extracted information

from the included studies encompassed the following: (1) study characteristics (first author's last name, study location, publication year, study design, and sample size); (2) participant details (mean age, health status, gender, and body mass index (BMI)); (3) specifics of the intervention (study duration, type and dosage of intervention, and control); and (4) main findings. Corresponding authors of the primary studies were contacted when additional information was required. Any disagreements regarding data extraction were resolved through discussion.

Assessment of study quality and grading of the evidence

Two investigators (AHM and VM) independently assessed the risk of bias in the included studies using the Cochrane Risk of Bias tool. This tool evaluates seven domains: (1) random sequence generation, (2) allocation concealment, (3) blinding of participants and personnel, (4) blinding of outcome assessment, (5) incomplete outcome data, (6) other sources of bias, and (7) selective reporting. Each domain was classified as "low risk," "high risk," or "unclear risk" of bias [16]. The certainty of the evidence was assessed using the GRADE approach [17], with detailed criteria outlined in Supplementary Table 2.

Statistical analysis

To assess the effect size for the lipid profile, we calculated the standard deviations (SDs) and mean differences for both the control and intervention groups. Standardized mean differences (SMDs) with 95% confidence intervals (CIs) were calculated using a random-effects model with the restricted maximum likelihood (REML) method. Given the small number of included studies, we applied the Hartung-Knapp adjustment to modify the standard error (SE) of the mean. The SDs of the mean difference were calculated using the formula: SD = √[(SD_pre-treatment)^2 + (SD_post-treatment)^2 − (2R × SD_pre-treatment × SD_post-treatment)], assuming a correlation coefficient (R) of 0.9. For outcome measures reported as medians and ranges, we estimated mean and SD values using the method proposed by Wan et al. [18]. To evaluate between-study heterogeneity, we used the I-square (I^2) statistic and Cochran's Q test. An I^2 value greater than 50.5% or a *p*-value below 0.1 was considered indicative of significant heterogeneity. We conducted subgroup analyses based on glucomannan dose, baseline BMI, intervention duration, health condition, gender, mean age, experimental design, and sample size. We also performed a sensitivity analysis (leave-one-out method) to assess the influence of individual studies on the overall estimate. To evaluate the potential for publication bias, we applied Egger's regression asymmetry test [19]. In cases where significant publication bias was detected, we used the trim-and-fill method to adjust the estimates. A random-effects meta-regression analysis was performed to investigate the influence of study characteristics on SMD. Model fit was assessed using the R^2 statistic, which represents the proportion of between-study variance explained by the model. The tau^2 statistic measured the residual heterogeneity not explained by the covariates. The Knapp–Hartung method was used to adjust the standard errors, and REML was applied to estimate residual heterogeneity (τ^2). There should be at least 10 studies per covariate included in the meta-regression to have enough degrees of freedom to provide stable estimates and reduce the risk of overfitting. Statistical analysis was conducted using STATA version 17 program. *P*-values below 0.05 were statistically significant.

Results

Flow and characteristics of the included studies

The detailed research screening process is shown in Fig. 1. Initially, 144 published studies were identified through searches across multiple databases. After a thorough screening process, 11 RCTs [7–13, 20–23] published between 1984 [22] and 2020 [7] met all inclusion criteria and were deemed eligible for inclusion.

Table 1 summarizes the characteristics of the included trials. A total of 334 participants were divided into either a glucomannan intervention group or a control group. Among the trials, one was conducted exclusively with women [22], two with men [8, 20], and eight with both genders [7, 9–13, 21, 23]. The mean age of participants ranged from 32 to 64 years. The trial durations varied from 3 to 12 weeks: four trials lasted 3 weeks [11, 12, 21, 23], five lasted 4 weeks [7, 9, 10, 13, 20], one lasted 8 weeks [22], and one lasted 12 weeks [8].

The studies targeted diverse populations, including healthy individuals [20, 21], overweight and obese population [8, 22], type 2 diabetes (T2DM) [9, 12, 13, 23], hypercholesterolemia [10], Schizophrenia [7], and insulin resistance syndrome [11].

Risk of bias assessment and meta-evidence

Assessment of risk of bias in included studies using Cochrane criteria is shown in Table 2. The GRADE certainly of evidence was assessed as low for TC and LDL-C, and very low for TG and HDL-C, as indicated in (Supplementary Table 2).

Glucomannan on TC levels

The combined analysis of data from 11 trials (13 arms) revealed a significant reduction in TC levels associated with glucomannan supplementation (SMD: -3.299; $SE_{Hartung-Knapp}$:0.76; 95% CI: -4.955, -1.644, $P=0.001$; $I^2=95.41\%$, P-heterogeneity < 0.001) ((Fig. 2) [7–13, 20–23]. Subgroup analyses showed that glucomannan

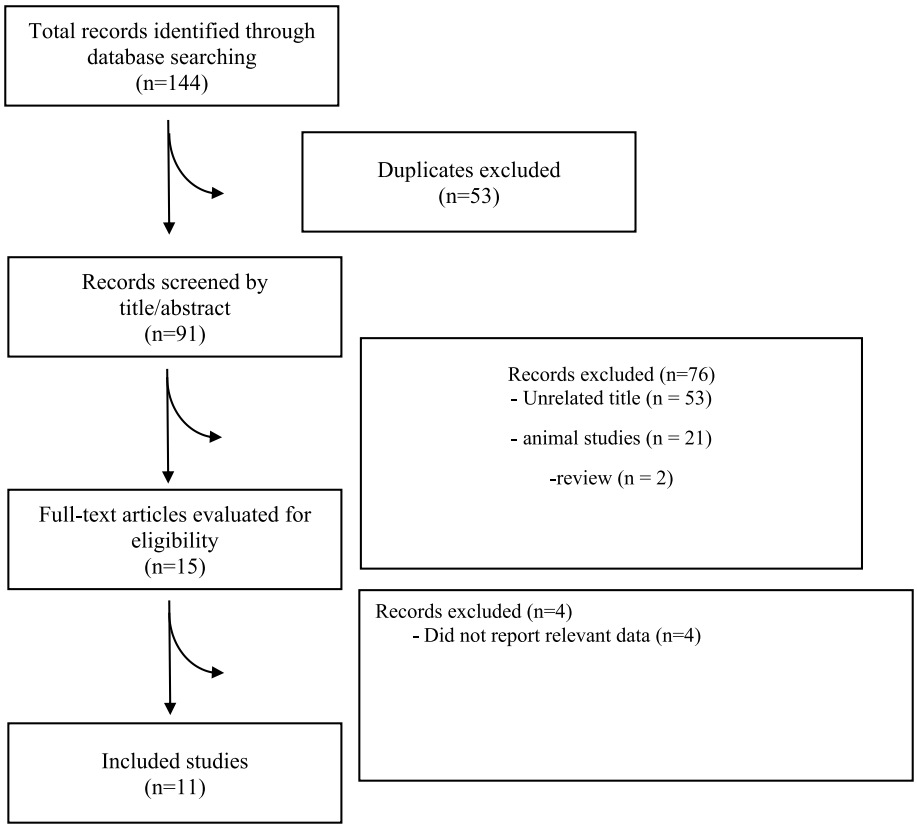

Fig. 1 Flow diagram of study selection

supplementation at doses of ≥5000 mg/day, with an intervention duration of less than 8 weeks, and in participants with an average age of ≥50 years resulted in a more pronounced reduction in TC levels among patients with T2DM (Table 3).

Glucomannan on TG levels

The results showed no significant effect of glucomannan supplementation on TG (SMD: -0.119; SE $_{\text{Hartung–Knapp}}$:0.435; 95% CI: -1.076, 0.837, $P=0.789$; $I^2=91.63\%$, P-heterogeneity < 0.001) (Fig. 3) [7–13, 20, 22, 23].

Glucomannan on HDL-C levels

Combining the data from nine trials with 11 arms intervention revealed a significant effect of glucomannan supplementation on HDL-C levels (SMD: -0.443; SE $_{\text{Hartung–Knapp}}$:0.164; 95% CI: -0.808, -0.078, $P=0.022$; $I^2=41.69\%$, P-heterogeneity = 0.07) (Fig. 4) [7–13, 20, 23].

Glucomannan on LDL-C levels

Glucomannan supplementation showed a considerable decrease in serum LDL-C levels (SMD: -2.993; SE $_{\text{Hartung–Knapp}}$:0.902; 95% CI: -4.958, -1.028; $P=0.006$; $I^2=95.49\%$, P-heterogeneity < 0.001) (Fig. 5) [7–13, 20–23]. Subgroup analysis indicated that glucomannan supplementation resulted in a more substantial reduction in LDL-C levels in trials with an intervention duration of less than 8 weeks, a sample size of 50 or fewer participants, and subjects with T2DM (Table 3).

Glucomannan on other lipid profile parameters

Results did not show any significant effect of glucomannan supplementation on Apo A1 (SMD: -0.476; SE $_{\text{Hartung–Knapp}}$:1.346; 95% CI: -6.269, 5.317; $P=0.757$) (Fig. 6), APO-B/ A1 ratio (SMD: -1.15; SE $_{\text{Hartung–Knapp}}$:0.41; 95% CI: -2.913, 0.614; $P=0.107$) (Fig. 7), and LDL-C/ HDL-C levels (SMD: -2.203; SE $_{\text{Hartung–Knapp}}$:1.18; 95% CI: -7.278, 2.873; $P=0.203$) (Fig. 8). However, the results indicated that glucomannan supplementation had a significant effect on Apo B1 (SMD: -2.201; SE $_{\text{Hartung–Knapp}}$:0.496; 95% CI: -3.579, -0.823; $P=0.011$) (Fig. 9).

Sensitivity analysis and publication bias

Sensitivity analyses for TC, TG, and LDL-C showed no significant changes in effect sizes when any single study was excluded. However, removing individual studies notably impacted the overall effect of glucomannan

Table 1 Study characteristics of included studies

Author, year	Design	Participants, n	Health condition	Age, year	Intervention		Duration (week)
					Treatment group	Control group	
Walsh et al. 1984 [22]	RA/DB/parallel	F: 20 Int: 10, Con: 10	Obesity	NR	3000 mg/day Glucomannan (capsule)	Starch	8
Venter et al. 1987 [10]	RA/DB/crossover	M/F: 10 M/F: 8	Hypercholester-olemia	40.1 42	4500 mg/day Konjac Glucoman-nan (capsule)	Corn Starch	4
Arvill et al. 1995 [20]	RA/DB/crossover	M: 63	Healthy	47	3900 mg/day Glucomannan (capsule)	Corn Starch	4
Vuksan et al. 1999 [12]	RA/DB/crossover	M/F: 11	T2DM	60.5	15000 mg/day Konjac-mannan (biscuit)	Wheat Bran	3
Vuksan et al. 2000 [11]	RA/DB/crossover	M/F: 11	Insulin Resistance Syndrome	55	12900 mg/day Konjac-mannan	Wheat Bran	3
Chen et al. 2003 [9]	RA/DB/crossover	M/F: 22	T2DM	64.2	3600 mg/day Konjac Glucoman-nan (capsule)	Placebo	4
Yoshida et al. 2006 [23]	RA/DB/crossover	M/F: 29 M/F: 29	T2DM Non-diabetic	56.81 55.19	10000 mg/day Glucomannan	Placebo	3
Chearskul et al. 2007 [13]	RA/SB/crossover	M/F: 20	T2DM	51.2	1000 mg/day Glucomannan (capsule)	White Rice Flour	4
Wood et al. 2007 [8]	RA/DB/parallel	M: 29 Int: 14, Con: 15	Overweight and Obese	38.8	3000 mg/day Konjac-mannan (capsule)	Maltodextrin	12
Vuksan et al. 2011 [21]	RA/crossover	M/F: 23	Healthy	35	3900 mg/day Glucomannan	Wheat Bran	3
Zhang et al. 2020 [7]	RA/DB/parallel	M/F: 59 Int: 30, Con: 29	Schizophrenia	Int: 32.57, Con: 31.46	2000 mg/day Konjac Flour	Maltodextrin	4

Abbreviations: *RA* Randomized, *DB* Double-blinded, *M* Male, *F* Female, *Int* Intervention, *Con* Control, *NR* Not reported, *T2DM* Type 2 diabetes mellitus, *SB* Single-blinded

Table 2 Results of risk of bias assessment for randomized clinical trials included in the current meta-analysis on the effects of glucomannan supplementation on lipid profile

Study	Random Sequence Generation	Allocation concealment	Reporting bias	Other sources of bias	Performance bias	Detection bias	Attrition bias
Walsh et al. 1984 [22]	L	L	L	H	L	L	H
Venter et al. 1987 [10]	L	L	L	H	L	L	H
Arvill et al. 1995 [20]	L	H	L	H	L	L	H
Vuksan et al. 1999 [12]	L	U	L	L	L	L	H
Vuksan et al. 2000 [11]	L	U	L	L	L	L	H
Chen et al. 2003 [9]	L	U	L	L	L	L	H
Yoshida et al. 2006 [23]	L	U	L	L	L	L	L
Chearskul et al. 2007 [13]	U	U	L	H	L	H	H
Wood et al. 2007 [8]	L	U	L	H	L	L	H
Vuksan et al. 2011 [21]	L	U	L	H	U	U	H
Zhang et al. 2020 [7]	L	L	L	H	L	L	L

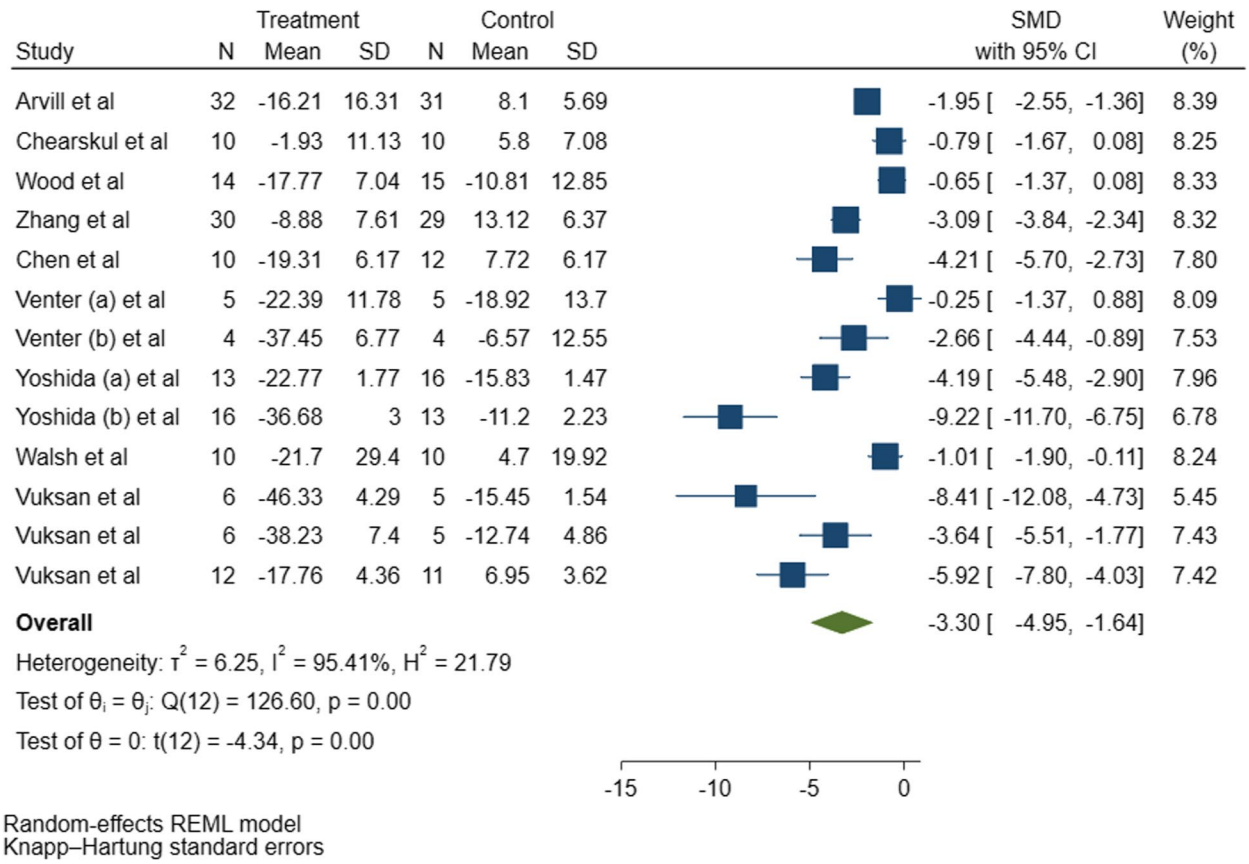

Fig. 2 Forest plot of the effects of glucomannan supplementation on TC levels

supplementation on HDL-C [8, 20] [7, 23], altering the effect to a non-significant level.

Egger's and Begg's tests indicated a significant small study effect for LDL-C and TC ($P < 0.05$), but not for HDL-C or TG. Additionally, publication bias was detected in this meta-analysis, as evidenced by slight asymmetries in the funnel plots (Fig. S1-3). The trim-and-fill test was conducted to address this issue, and the results for TG, with four imputed studies, remained non-significant (SMD: -0.77; 95% CI: -1.66, 0.11, $p > 0.05$) (Fig.S4).

Meta-regression

The meta-regression model included three covariates: sample size, duration of the intervention, and dosage. Other variables such as BMI and mean age were not included in the analysis due to missing data in some studies. Based on R^2 results for TC, LDL-C, HDL-C, and TG, the overall model explained 30.83%, 11.42%, 100%, and 0.0% of the between-study variance, respectively. However, the model was not statistically significant ($p > 0.05$). Except HDL-C. residual heterogeneity remained substantial, as indicated by a τ^2 and I^2 statistics, suggesting that much of the variability in effect sizes was unexplained by the included covariates. Regarding HDL-C, there was negligible residual heterogeneity (τ^2 = 2.0e-07), indicating no unaccounted variability (I^2 = 0%, H^2 = 1.00).

Discussion

Two previous meta-analyses and systematic reviews have examined the effects of glucomannan on lipid profiles [6, 14]. However, these studies had several limitations. Firstly, their subgroup analyses were limited, and the range of biomarkers investigated (LDL-C, non-HDL cholesterol, TG, TC, HDL-C, and apolipoprotein B) was less comprehensive than in our study. Additionally, these reviews combined data from both children and adults, despite differing responses to treatment and supplement dosages between these populations. This makes it inappropriate to report results for these groups together. Furthermore, the study protocol of Sood et al. [14] was not registered in any database. Therefore, there is a need for an updated meta-analysis focusing exclusively on adults and incorporating extensive subgroup analyses to provide a more thorough evaluation.

Table 3 Subgroup analyses for the effects of glucomannan supplementation on lipid profile

	NO	SMD (95% CI)[a]	P-within[b]	I^2 (%)[c]	P-heterogeneity[d]
Glucomannan supplementation on TC					
Overall	13	-3.299 (-4.955, -1.644)	< 0.001	95.41	< 0.001
Age(year)					
< 50	6	-2.315 (-4.388, -0.241)	0.035	93.74	< 0.001
≥ 50	6	-4.834 (-8.112, -1.556)	0.013	93.05	< 0.001
NR	1	-1.007 (-1.902, -0.111)	0.02	-	-
Intervention duration (week)					
< 8	11	-3.791 (-5.634, -1.948)	< 0.001	94.58	< 0.001
≥ 8	2	-0.79 (-3.03, 1.451)	0.14	0.00	0.540
Dosage of Glucomannan (mg/day)					
< 5000	9	-2.171 (-3.573, -0.77)	0.007	92.39	< 0.001
≥ 5000	4	-6.126 (-10.673, -1.578)	0.023	85.89	< 0.001
Study population					
Healthy	2	-3.831 (-28.983, 21.322)	0.304	93.54	< 0.001
T2DM	4	-3.131 (-5.825, -0.437)	0.034	85.31	< 0.001
Hypercholesterolemia	2	-1.352 (-16.658, 13.953)	0.463	80.32	0.024
Non-diabetics	1	-9.223 (-11.701, -6.745)	< 0.001	-	-
Overweight and Obese	2	-0.79 (-3.03, 1.451)	0.014	0.00	0.540
Schizophrenia	1	-3.089 (-3.84, -2.338)	< 0.001	-	-
Insulin Resistance Syndrome	1	-8.407 (-12.084, -4.73)	< 0.001	-	-
Intervention type					
Glucomannan	6	-3.7 (-7.086, -3.14)	0.038	97.09	< 0.001
Konjac	7	-2.923 (-5.171, -0.674)	0.019	92.03	< 0.001
Sample size					
≤ 50	11	-3.498 (-5.517, -1.48)	0.003	94.95	< 0.001
>50	2	-2.497 (-9.705, 4.712)	0.142	81.46	0.020
BMI					
≤ 25	1	-5.918 (-7.801, -4.035)	0.002	-	-
25–30	5	-4.433 (-9.452, 0.587)	0.07	97.28	< 0.001
> 30	1	-4.189 (-5.481, -2.897)	0.001	-	-
NR	6	-2.007 (-3.329, -0.685)	0.011	83.2	< 0.001
Study Design					
RCT	3	-1.586 (-4.874, 1.702)	0.174	90.85	< 0.001
Cross Over	10	-3.895 (-5.973, -1.817)	0.002	94.42	< 0.001
Gender					
Both	10	-4.011 (-6.036, -1.985)	0.002	93.52	< 0.001
Men	2	-1.317 (-9.616, 6.981)	0.293	86.53	0.006
Women	1	-1.007 (-1.902, -0.111)	0.021	-	-
Glucomannan supplementation on LDL					
Overall	13	-2.993 (-4.958, -1.028)	0.006	95.49	< 0.001
Age(year)					
< 50	6	-1.755 (-3.279, -0.231)	0.032	89.06	< 0.001
≥ 50	6	-5.458 (-10.92, 0.004)	0.05	96.73	< 0.001
NR	1	-0.797 (-1.672, 0.078)	0.061	-	-
Intervention duration (week)					
< 8	11	-3.499 (-5.857, -1.141)	0.008	95.52	< 0.001
≥ 8	2	-0.812 (-0.976, -0.648)	0.01	0.00	0.964
Dosage of Glucomannan (mg/day)					
< 5000	9	-1.831 (-2.904, -0.758)	0.004	87.45	7.97

Table 3 (continued)

	NO	SMD (95% CI)[a]	P-within[b]	I^2 (%)[c]	P-heterogeneity[d]
≥ 5000	4	-7.456 (-17.768, 2.855)	0.105	96.84	31.65
Study population					
Healthy	2	-2.496 (-6.557, 1.566)	0.081	12.57	0.285
T2DM	4	-2.941 (-4.653, -1.228)	0.012	66.71	0.023
Hypercholesterolemia	2	-1.929 (-29.788, 25.93)	0.541	90.47	< 0.001
Non-diabetics	1	-8.171 (-10.391, -5.951)	< 0.001	-	-
Overweight and Obese	2	-0.812 (-0.976, -0.648)	0.01	0.00	0.964
Schizophrenia	1	-1.327 (-1.884, -0.769)	0.005	-	-
Insulin Resistance Syndrome	1	-18.339 (-26.079, -10.599)	< 0.001	-	-
Intervention type					
Glucomannan	6	-3.025 (-5.624, -0.425)	0.03	95.29	< 0.001
Konjac	7	-3.465 (-8.002, 1.072)	0.111	97.63	< 0.001
Sample size					
≤ 50	11	-3.379 (-5.916, -0.842)	0.014	95.43	< 0.001
>50	2	-1.805 (-8.031, 4.42)	0.169	80.73	0.023
BMI					
≤ 25	1	-3.038 (-4.218, -1.858)	< 0.001	-	-
25–30	5	-5.767 (-13.907, 2.372)	0.121	98.67	< 0.001
> 30	1	-3.126 (-4.2, -2.052)	< 0.001	-	-
NR	6	-1.805 (-3.453, -0.157)	0.037	89.09	< 0.001
Study Design					
RCT	3	-1.073 (-1.856, -0.289)	0.028	0.00	0.445
Cross Over	10	-3.803 (-6.468, -1.138)	0.01	94.89	< 0.001
Gender					
Both	10	-3.735 (-6.497, -0.973)	0.014	95.62	< 0.001
Men	2	-1.578 (-11.005, 7.85)	0.28	88.82	< 0.001
Women	1	-0.797 (-1.672, 0.078)	0.059	-	-
Glucomannan supplementation on HDL					
Overall	11	-0.443 (-0.808, -0.078)	0.022	41.69	0.07
Age(year)					
< 50	5	-0.476 (-1.414, 0.463)	0.232	69.49	0.032
≥ 50	6	-0.323 (-0.743, 0.097)	0.105	4.06	0.506
Intervention duration (week)					
< 8	10	-0.405 (-0.818, 0.007)	0.053	48.03	0.044
≥ 8	1	-0.635 (-1.361, 0.092)	0.091	-	-
Dosage of Glucomannan (mg/day)					
< 5000	7	-0.455 (-1.034, 0.123)	0.102	54.89	0.042
≥ 5000	4	-0.357 (-1.089, 0.375)	0.219	23.79	0.331
Study population					
Healthy	1	-0.963 (-1.479, -0.447)	0.011	-	-
T2DM	4	-0.191 (-0.637, 0.256)	0.268	0.00	0.731
Hypercholesterolemia	2	0.47 (-7.021, 7.961)	0.572	45.22	0.177
Non-diabetics	1	-0.9 (-1.648, -0.152)	0.002	-	-
Overweight and Obese	1	-0.635 (-1.361, 0.092)	0.072	-	-
Schizophrenia	1	-0.897 (-1.426, -0.368)	0.006	-	-
Insulin Resistance Syndrome	1	0.00 (-1.085, 1.085)	0.058	-	-
Intervention type					
Glucomannan	4	-0.633 (-1.349, 0.084)	0.067	42.47	0.164
Konjac	7	-0.275 (-0.836, 0.286)	0.275	45.42	0.087

Table 3 (continued)

	NO	SMD (95% CI)[a]	P-within[b]	I^2 (%)[c]	P-heterogeneity[d]
Sample size					
≤ 50	9	-0.267 (-0.661, 0.127)	0.157	16.78	0.262
>50	2	-0.931 (-1.351, -0.51)	0.023	0.00	0.861
BMI					
25–30	5	-0.477 (-0.963, 0.01)	0.053	0.00	0.476
> 30	1	0.00 (-0.711, 0.711)	0.068	-	-
NR	5	-0.417 (-1.386, 0.551)	0.298	69.47	0.030
Study Design					
RCT	2	-0.806 (-2.391, 0.779)	0.098	0.00	0.568
Cross Over	9	-0.318 (-0.766, 0.13)	0.14	45.14	0.062
Gender					
Both	9	-0.312 (-0.747, 0.124)	0.138	40.92	0.169
Men	2	-0.853 (-2.822, 1.117)	0.114	0.00	0.999
Glucomannan supplementation on TG					
Overall	12	-0.119 (-1.076, 0.837)	0.789	91.63	< 0.001
Age(year)					
< 50	5	-0.455 (-1.273, 0.362)	0.197	70.63	0.009
≥ 50	6	0.166 (-2.086, 2.419)	0.857	94.27	< 0.001
NR	1	-0.401 (-1.25, 0.448)	0.064	-	-
Intervention duration (week)					
< 8	10	-0.15 (-1.337, 1.038)	0.782	92.9	< 0.001
≥ 8	2	0.057 (-5.351, 5.465)	0.915	55.87	0.132
Dosage of Glucomannan (mg/day)					
< 5000	8	-0.295 (-0.796, 0.207)	0.207	63.21	0.006
≥ 5000	4	0.138 (-4.289, 4.565)	0.927	95.79	< 0.001
Study population					
Healthy	1	-0.883 (-1.394, -0.371)	0.002	-	-
T2DM	4	0.876 (-1.27, 3.023)	0.285	87.33	< 0.001
Hypercholesterolemia	2	-0.217 (-1.955, 1.521)	0.358	0.00	0.745
Non-diabetics	1	-3.693 (-4.88, -2.506)	< 0.001	-	-
Overweight and Obese	2	0.057 (-5.351, 5.465)	0.915	55.87	0.132
Schizophrenia	1	-1.099 (-1.641, -0.558)	0.002	-	-
Insulin Resistance Syndrome	1	1.059 (-0.113, 2.231)	0.092	-	-
Intervention type				-	-
Glucomannan	5	-0.32 (-3.252, 2.612)	0.777	96.72	< 0.001
Konjac	7	-0.034 (-0.683, 0.615)	0.903	63.16	0.004
Sample size					
≤ 50	10	0.07 (-1.082, 1.222)	0.894	90.35	< 0.001
>50	2	-0.985 (-2.36, 0.39)	0.07	0.00	0.568
BMI					
25–30	5	-0.329 (-2.659, 2.001)	0.715	93.68	< 0.001
> 30	1	2.915 (1.881, 3.949)	0.05	-	-
NR	6	-0.61 (-1.128, -0.092)	0.029	36.41	0.198
Study Design					
RCT	3	-0.372 (-2.337, 1.593)	0.501	80.69	< 0.001
Cross Over	9	-0.036 (-1.366, 1.293)	0.951	92.38	< 0.001
Gender					
Both	9	-0.062 (-1.404, 1.28)	0.918	92.38	< 0.001
Men	2	-0.24 (-8.716, 8.237)	0.78	88.66	< 0.001

Table 3 (continued)

	NO	SMD (95% CI)[a]	P-within[b]	I^2 (%)[c]	P-heterogeneity[d]
Women	1	-0.401 (-1.25, 0.448)	0.081	-	-
Glucomannan supplementation on LDL: HDL					
Overall	3	-2.2 (-7.28, 2.87)	0.2	92.07	< 0.001
Glucomannan supplementation on APO-B: A1					
Overall	3	-1.15 (-2.91, 0.61)	0.11	34.26	0.16
Glucomannan supplementation on APO-B					
Overall	5	-2.2 (-3.58, -0.82)	0.01	68.01	0.03
Glucomannan supplementation on APO-A1					
Overall	3	-0.48 (-6.27, 5.32)	0.76	94.11	< 0.001

Abbreviation: *SMD* Standard mean differences, *CI* Confidence interval, *T2DM* Type 2 diabetes mellitus, *NAFLD* Non-alcoholic fatty liver disease

[a] Obtained from the Random-effects model

[b] Refers to the mean (95% CI)

[c] Inconsistency, percentage of variation across studies due to heterogeneity

[d] Obtained from the Q-test

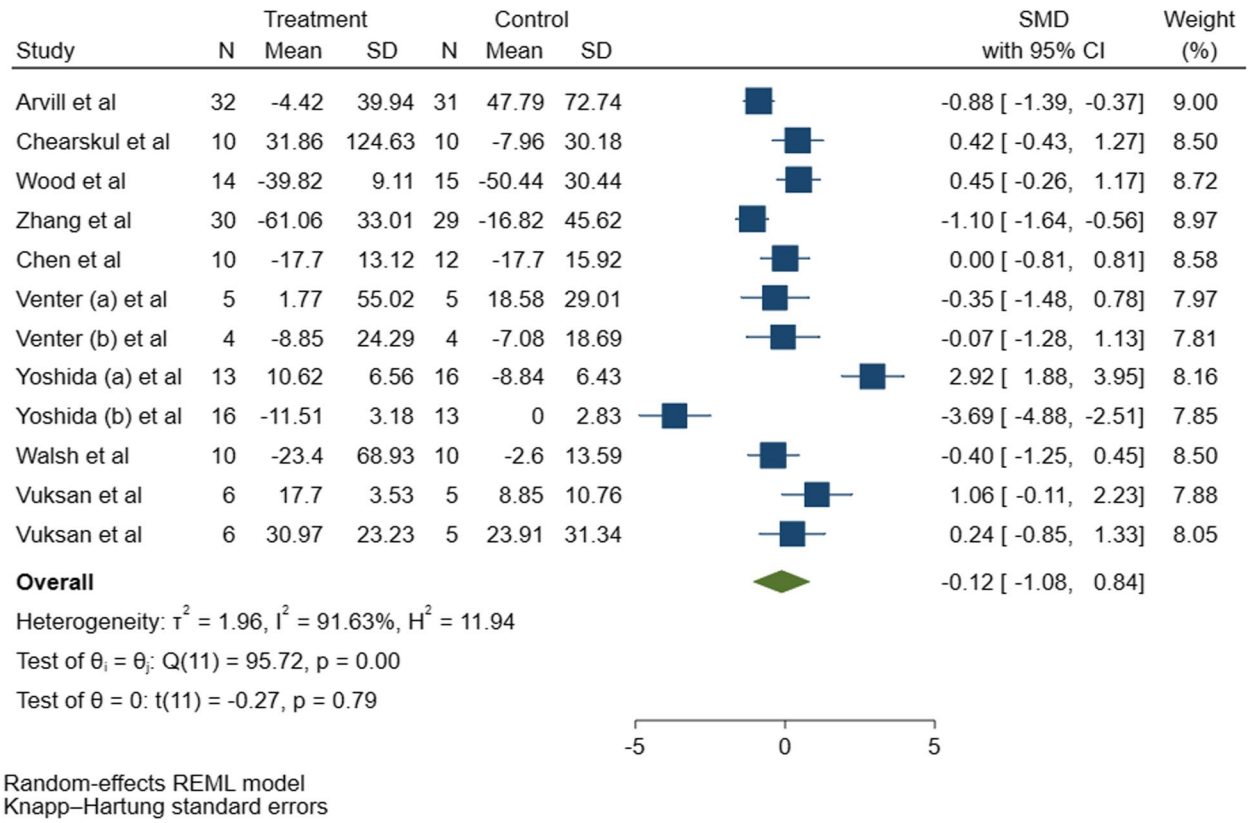

Fig. 3 Forest plot of the effects of glucomannan supplementation on TG levels

Our pooled analysis showed that glucomannan supplementation decreased TC, LDL-C, and Apo-B, while it had no significant effect on TG, Apo-B/A1, LDL-C/HDL-C, and APO-A1. Moreover, it significantly decreased HDL-C levels. This effect is likely related to the weight reduction of participants during the study. Previously, a meta-analysis found that participants actively losing weight experienced a 0.007 mmol/L decrease in HDL-C for each kilogram of body weight lost [24]. However, sensitivity analysis showed that the HDL-C findings could

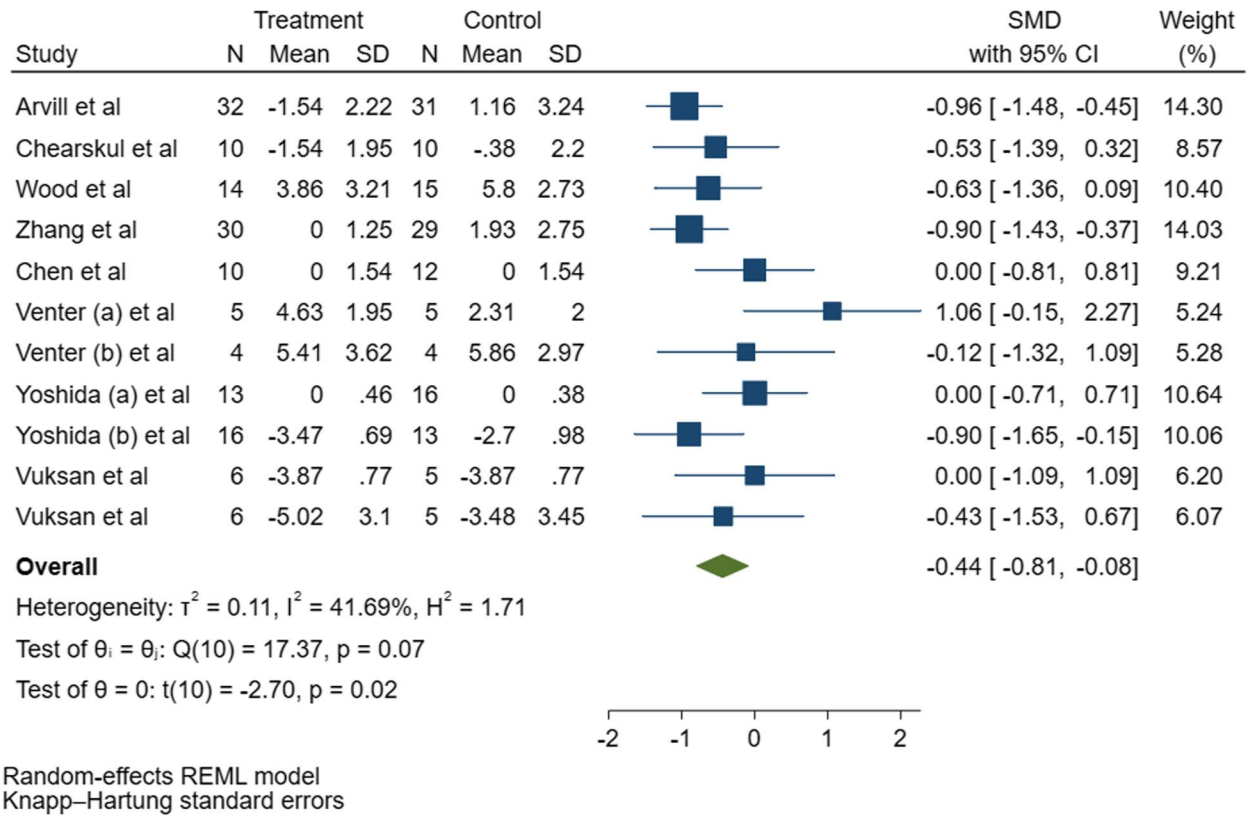

Fig. 4 Forest plot of the effects of glucomannan supplementation on HDL-C levels

not be considered robust, as excluding some studies one by one could change the significance of the results. Due to the high heterogeneity on findings of TC, LDL-C, TG, APO-A, Apo-B, and LDL-C/HDL-C, these findings should be interpreted with caution. According to the minimally clinically important difference defined for LDL-C and TC (± 1 mmol/L) [25], our effect size indicates that the anti-hyperlipidemic effect of glucomannan is not clinically significant. Therefore, glucomannan can only be considered as an adjuvant therapeutic approach in managing hyperlipidemia for healthcare providers.

Based on subgroup analysis, glucomannan, as a viscous soluble fiber, has shown a favorable effect on TC and LDL-C levels across genders. Younger patients (< 50 years of age) appear to benefit more from this supplementation, which may be related to the age-related trends of lipid biomarkers observed by Feng et al., where age was positively associated with LDL-C and TC levels in younger adults. On the other hand, age was negatively associated with LDL-C and TC levels in ≥ 61 years adults [26]. Moreover, lower doses of glucomannan (< 5000 mg/day) and shorter durations of supplementation (< 8 weeks) have more positive effects on TC and LDL-C compared to higher doses (≥ 5000 mg/day) and longer durations (≥ 8 weeks). However, other factors such as the study population, study design, and sample size can be influential in this finding, and this finding cannot necessarily be related to glucomannan. Due to the small sample size in the higher dose and longer duration subgroup (only 2 studies compared to 11 studies), no definitive conclusions can be drawn in this regard. Therefore, more studies with larger sample sizes, longer supplement periods, and higher doses are needed to provide conclusive evidence in this area. Several studies have reported that glucomannan is well tolerated and has a favorable safety profile [27, 28]. Additionally, the viscosity of dietary fiber appears to be more important than quantity in reducing cholesterol levels [21]. Regarding body mass index (BMI), due to the limited number of studies in some subgroups, particularly obese subjects, it is not possible to definitively interpret the effect of glucomannan on lipid profiles in different BMI categories. Further research, especially in obese populations, is warranted. In terms of plant source, glucomannan can be derived from various plants, konjac glucomannan specifically refers to the glucomannan extracted from the *Amorphophallus konjac*. Both serve similar functions and offer similar health benefits, but the source plant is what distinguishes konjac glucomannan from the broader category of glucomannan. The chemical composition of glucomannan,

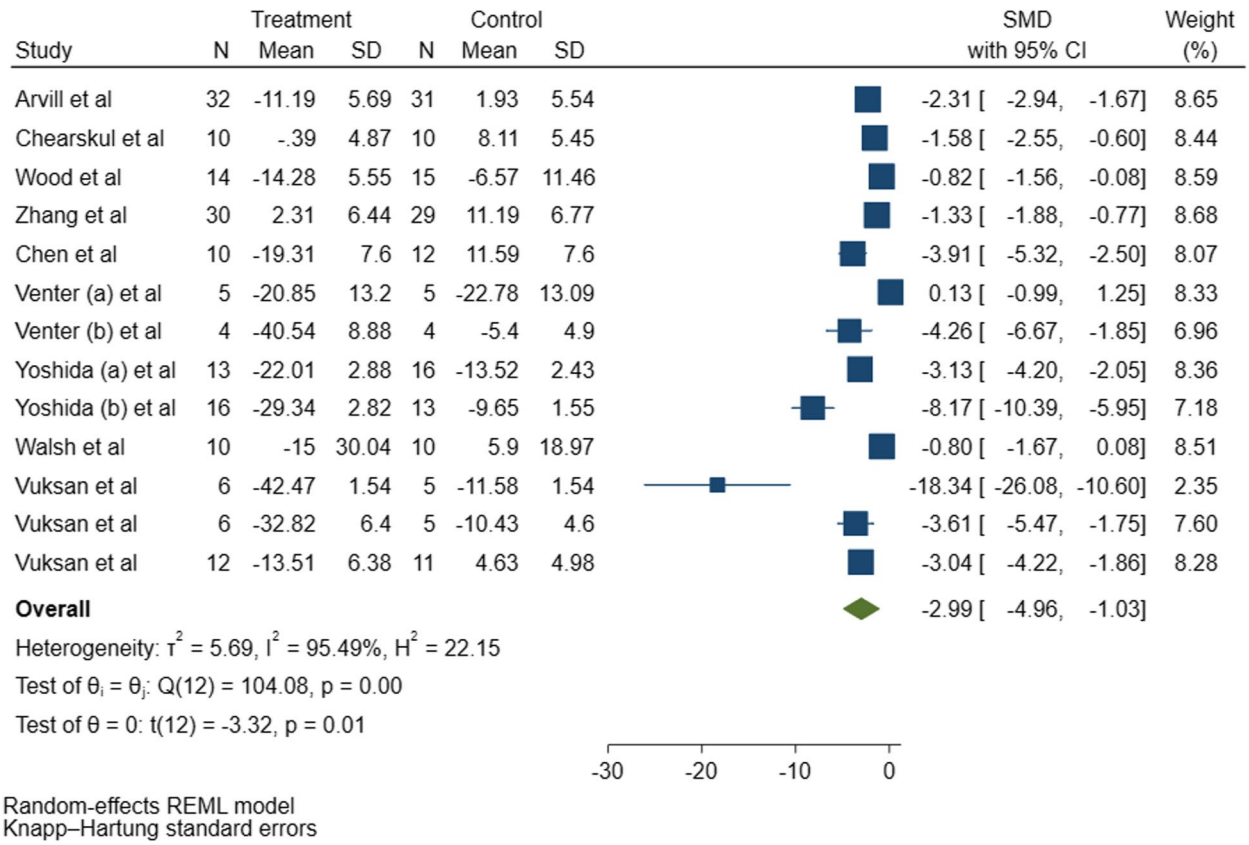

Fig. 5 Forest plot of the effects of glucomannan supplementation on LDL-C levels

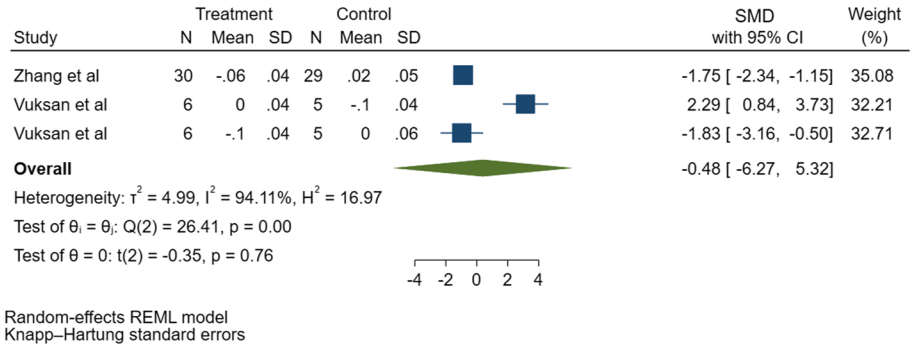

Fig. 6 Forest plot of the effects of glucomannan supplementation on Apo A1

whether derived from konjac or other sources, is essentially the same. Both are composed of the same type of polysaccharide [29]. There are several possible reasons why the effect of glucomannan on lipid profile may be more pronounced in cross-over design. Cross-over studies involve each participant serving as their own control, which can help reduce variability in the results. This can lead to a clearer demonstration of the effects of glucomannan on lipid profile. Moreover, cross-over studies typically require fewer participants compared to parallel group studies, which can result in increased statistical power to detect significant differences in lipid profile outcomes [30].

Glucomannan's ability to form a viscous gel in the gastrointestinal tract allows it to bind bile acids. This binding prevents the reabsorption of bile acids back into the bloodstream, leading to their increased excretion in feces [31]. Bile acids are synthesized from cholesterol in

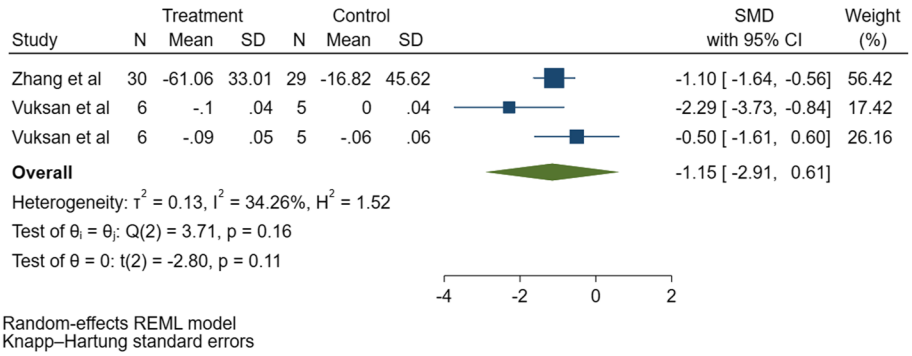

Fig. 7 Forest plot of the effects of glucomannan supplementation on APO-B/ A1 levels

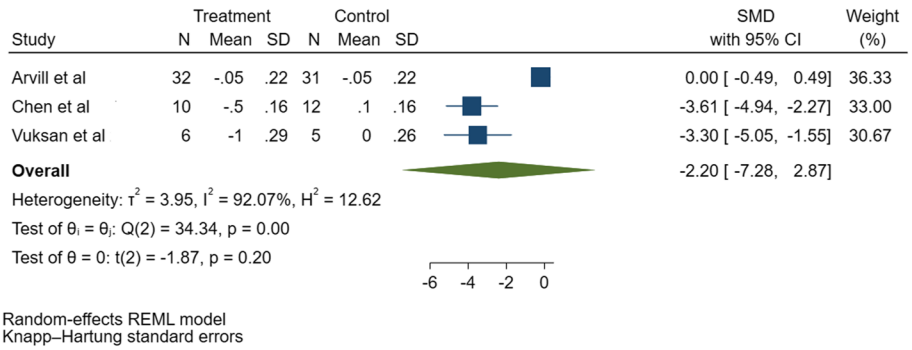

Fig. 8 Forest plot of the effects of glucomannan supplementation on LDL-C/ HDL-C levels

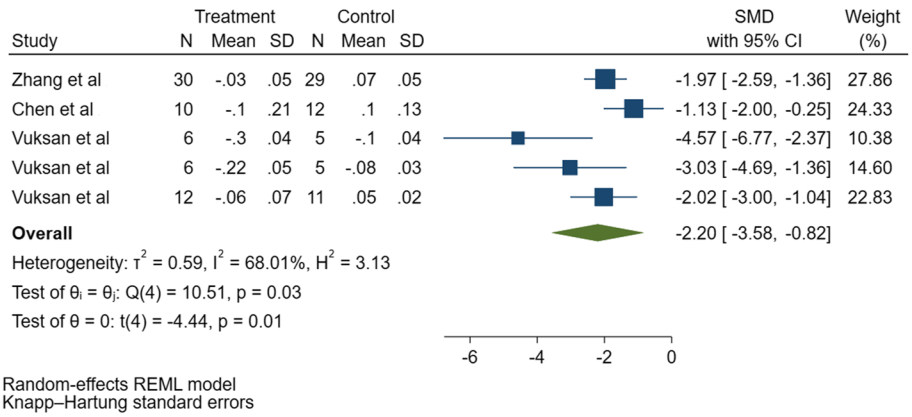

Fig. 9 Forest plot of the effects of glucomannan supplementation on Apo B1 levels

the liver; thus, their increased excretion forces the liver to use more cholesterol to synthesize new bile acids. This process reduces the pool of cholesterol available for other functions, including the formation of LDL-C [9]. The gel-forming property of glucomannan can also reduce the absorption of dietary cholesterol in the intestines. By increasing the viscosity of the intestinal contents, glucomannan slows down the mixing of cholesterol with bile acids and its subsequent micelle formation, which is crucial for cholesterol absorption. Reduced micelle formation leads to decreased cholesterol uptake by enterocytes and thus lowers the amount of cholesterol entering the bloodstream [32]. Glucomannan may influence the metabolism of lipoproteins, particularly

LDL-C. By reducing cholesterol absorption and increasing bile acid excretion, the liver upregulates the expression of LDL receptors to compensate for the decreased cholesterol availability. This upregulation enhances the clearance of LDL-C from the blood, further lowering LDL-C levels [33]. Moreover, through interaction with mannose receptor, glucomannan can stimulate macrophages in vivo in order to effectively remove circulating atherogenic lipoproteins [34]. In addition, glucomannan fermentation in the colon by gut microbiota produces short-chain fatty acids (SCFAs), such as acetate, propionate, and butyrate [35]. Propionate, in particular, has been shown to inhibit hepatic cholesterol synthesis [36]. G protein-coupled receptors (GPCRs), such as GPR41 and GPR43, have been reported as SCFA receptors. These GPCRs can bind to SCFAs in the gut and lead to improved insulin signaling and inhibition of lipid synthesis gene expression [37, 38]. In addition, AMP-activated protein kinase (AMPK) can be activated by SCFAs, leading to increased fatty acid oxidation and decreased fat deposition [39].

There are limitations in the meta-analysis of the present systematic review. Firstly, there is significant heterogeneity among the included studies, which necessitates cautious interpretation of the results. However, we identified major sources of this heterogeneity through subgroup analyses. Additionally, sensitivity analyses were performed to assess the robustness of our findings and to determine the influence of individual studies. Sensitivity analyses for TC, TG, and LDL-C showed no significant changes in effect sizes. However, removing individual studies notably altered the effect of glucomannan on HDL-C to a non-significant level. Future studies must focus on homogeneous study populations and standardized dosages to reach a conclusive finding. Secondly, due to the limited number of studies for some biomarkers, subgroup analyses could not be conducted in these cases. Moreover, detailed interpretations of these subgroups are challenging due to the inclusion of only one study in some instances, highlighting the need for more research. Thirdly, most of the studies included in our analysis were of low quality according to the Cochrane tool, which impacts the certainty of the evidence. Therefore, additional high-quality studies are essential to establish definitive conclusions. Despite these limitations, our study has several strengths. Firstly, our updated systematic review and meta-analysis comprehensively addressed all sources of heterogeneity and conducted thorough subgroup analyses. Secondly, our study protocol was registered in PROSPERO, enhancing transparency and methodological rigor. These strengths contribute to the reliability and validity of our findings, despite the challenges posed by heterogeneity and study quality issues.

Conclusion

Present updated systematic review meta-analysis showed that glucomannan supplementation has a beneficial effect on the level of TC and LDL-C. Based on GRADE, certainly of evidence is low for TC and LDL-C, and very low for TG and HDL-C. Therefore, additional high-quality studies are essential to establish definitive conclusions.

Supplementary Information

The online version contains supplementary material available at https://doi.org/10.1186/s12872-024-04223-0.

Supplementary Material 1.

Supplementary Material 2.

Acknowledgements
None.

Clinical trial number
Not applicable.

Authors' contributions
AHF and VM contributed in the systematic search and data extraction. RYR, and AHM contributed in the statistical analyses and data interpretation. ZBH, MF and ON contributes in manuscript drafting and data interpretation. AHF critically evaluated the analysis and edited the MS. All authors approved the final manuscript for submission.

Funding
Not applicable.

Availability of data and materials
The original data used during the current study can be obtained by contacting the corresponding author.

Declarations

Ethics approval and consent to participate
Not applicable.

Consent for publication
Not applicable.

Competing interests
The authors declare no competing interests.

Received: 16 March 2024 Accepted: 30 September 2024
Published online: 09 October 2024

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