The effect of Glucomannan on fasting and postprandial blood glucose in adults: a systematic review and meta-analysis of randomized controlled trials

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Abstract

Objective Glucomannan is a dietary fiber that slows the absorption of carbohydrates and suppresses appetite, thereby reducing blood glucose. This meta-analysis sought to examine the effect of glucomannan supplementation on Fasting Blood Glucose (FBG) and Postprandial Glucose (PPG) in adults.

Method We searched PubMed, and SCOPUS databases, and Google Scholar from inception to May 2020, using relevant keywords. All randomized controlled clinical trials (RCTs) that examined the effect of glucomannan supplementation on FBG and PPG in adults were included. Weighted mean differences (WMD) and their 95% confidence interval (CI) were calculated using Stata. Subgroup analysis was used to discern possible sources of heterogeneity.

Results Overall, 6 trials were included, consisting of 124 participants. We found that glucomannan supplementation significantly reduced FBG (WMD): -0.60 mmol/L, 95% CI: -1.16, -0.05; P=0.03, but not PPG (WMD: -2.07 mmol/L ; 95% CI: -5.09, 0.95; P=0.18), compared with controls group. We conducted subgroup analysis based on dosage and duration of intervention and health status of the population. Findings from subgroup analysis revealed a significant effect of glucomannan supplementation on FBG in diabetic patients (WMD: -1.28 mmol/L, 95% CI: -2.54, -0.02; P=0.04).

Conclusion Glucomannan supplementation can elicit significant reductions in FBG, but has no significant impact on PPG, in adults. More RCTs may find the exact effect of glucomannan on FBG and PPG.

Keywords Glucomannan · Blood glucose · Diabetes mellitus · Postprandial glucose

Introduction

According to the International Diabetes Federation) IDF(, about 463 million people worldwide are afflicted by diabetes, and it is estimated to increase up to 700 million people by the year 2045 [1]. About 90% of people with diabetes have Type 2 Diabetes Mellitus (T2DM) [2, 3]. Diabetes mellitus (DM) is defined by elevated blood glucose levels as a result of impaired insulin secretion or dysfunctional hormone response [4–6]. Genetics, obesity, low levels of physical activity, low socioeconomic status, and psychological stress are common risk factors for DM [7–11]. Prolonged hyperglycemia can lead to a wide variety of macro and microvascular complications in people with diabetes, including coronary heart disease, stroke, dialysis, retinopathy, and neuropathy, in addition to Lower-Extremity Amputations (LEA) [4, 12–14]. Medical Nutrition Therapy (MNT) and physical activity are considered vital in

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the prevention and treatment of DM and its associated complications and comorbidities [3, 15].

One of the most prevalent dietary recommendations is the consumption of dietary fibers [16]. Glucomannan is a fermentable water-soluble dietary fiber, that is emanate from konjac root (*Amorphophallus konjac*) [17]. It is used as an additive in traditional Asian cooking and also as a fiber supplement in the purified form [18]. The possible effects of glucomannan have been investigated in many studies, where it has been shown to reduce Total Cholesterol (TC) and Low-Density Lipoprotein Cholesterol (LDL-C) [19–21], induce body weight loss [22], and effectively reduce blood glucose level [18, 21, 23]. The underlying mechanism of glycemic parameter improvement is posited to be the slowed absorption of carbohydrates and suppression of appetite [24–26]. The high viscosity of glucomannan increases the gastrointestinal content viscosity as well; in fact, by extending the time for gastric emptying, absorption of nutrients in the small intestine decreases. Thus, the lowering rate of glucose absorption leads to an improved glycemic response [18, 27, 28].

However, several clinical studies dispute the advantages of glucomannan. One study displayed that insulin level was not different between glucomannan and placebo-treated groups [18]. Wood et al. concluded that adding Konjac-Mannan (KM) to a carbohydrate-restricted diet would not alter insulin level [29]; whilst Vuksan et al. reported that glucose and insulin level were not significantly changed by glucomannan in T2DM patients [25]. In 2008, Sood et al. conducted a systematic review and meta-analysis on the impact of glucomannan on plasma lipids, Fasting Blood Glucose (FBG), body weight, and blood pressure [26]. As reported by that meta-analysis, glucomannan favorably affected FBG, but this effect was not observe in people with diabetes. Moreover, a clinical study in 2013 reported that glucomannan did not significantly alter glucose parameters [30]. Therefore, given the inconsistent data, we conducted a systematic review and meta-analysis to evaluate the existing evidence for the effect of glucomannan supplementation on glycemic responses in people with and without diabetes.

Methods

The present study was conducted in accordance with the Preferred Reporting Items for Systematic review and Meta-Analysis Protocols (PRISMA-P) [31]. We registered the protocol of this study in PROSPRO and the registration code is ID=CRD42021291876. We removed this sentences from methods section and insert that we restricted the search until May 2020 and also excluded if not published in English

Eligibility criteria

We included studies that met the following inclusion criteria: 1) RCTs 2) study participants were adults (≥18 years old) 3) Oral glucomannan supplements or studies that used glucomannan in conjunction with another diet if consumed in both treatment and control groups 4) FBG or/and PPG were outcome measures. Studies were excluded if they were literature reviews, observational studies, case reports, republished data, animal studies, and we searched grey literature for relevant studies.

Study selection

Titles and abstracts of all reached articles in the initial search were evaluated independently by 2 investigators (MR-A and R-Z). With utilizing a screen form with a hierarchical approach, based on study design, population or exposure, and outcome, articles that did not meet the eligibility criteria were excluded. Then, full-texts of eligible articles were retrieved and subjected to a second evaluation by the same investigators. Each discordances were argued and resolved by consensus.

Data extraction

Two independent investigators (R-Z and A-M) separately extracted data from the included publications using a standard data extraction form. The following data were collected from each study: first author's name, study location, publication year, study sample size, number of participants in each group, health status of the population, study design (parallel/cross-over), gender of participants, mean or range of age, mean body mass index (BMI), study duration, type of diet, type and dose of glucomannan supplements, and mean and Standard Deviation (SD) of FBG and PPG at baseline and end of the study (Tables 1 and 2).

Quality assessment of studies

Two investigators (A-M and R-Z) assessed the quality of each selected study using the Cochrane risk of bias tools for RCTs. The risk of bias for the contained studies was assessed utilizing the Cochrane quality assessment tool for RCTs. The quality of studies was assessed using the following seven criteria: 1) random sequence generation 2) allocation concealment 3) blinding of participants and personnel 4) blinding of outcome assessment 5) incomplete outcome data 6) selective reporting and 7) other probable sources of biases. To evaluate the quality of studies, each study was judged as low risk, high risk, or unclear risk

Table 1 General characteristics of included studies

Author (year)	Subjects and gender	Age range (y)	Design	Medication dosage (g/day)	Duration (week)	Outcomes	Outcome — Intervention Mean ±SD	Outcome — Control Mean ± SD	Any other intervention	Notes about subjects	type of treatment of control group
Wood et al. [29]	30 Men20-69	20-69	parallel	3	12	FBS	Before mmol/L): 5.09 ±0.83 After (mmol/L):4.8 ±0.64	Before (mmol/L): 5.15±0.63 After (mmol/L): 5.01±0.56	carbohydraterestricted diet	30 overweight and obese men	carbohydraterestricted diet
Vuksan et al. [23]	11men/women	45–65	crossover8	8-13	8	FBS	Before (mmol/L):6.8±1.65 After (mmol/L):5.9±0.99	Before (mmol/L):6.6± 0.99 After (mmol/L):5.9±1.32	-	11 subjects with impaired glucose tolerance	wheat bran
Chen et al. [21]	22men/women	52–77	crossover	3.6	8	FBS PPG	(FBS) Before(mmol/L):9.4±2.3 After (mmol/L): 8±1.8 (PPG) Before (mmol/L):13.8±4,6 After (mmol/L):11.5±3.2	(FBS) Before(mmol/L):8.7±1.8 After (mmol/L):9.6±2.4 (PPG) Before (mmol/L):13±3.7 After (mmol/L):14.3±4.3	-	22 subjects with hyperlipide mia and type 2 diabetes	Placebo
Vuksan et al. [25]	11 men/women	45–65	crossover	15	8	FBS	Before (mmol/L): 9.63±2.95 After (mmol/L):8.62±3.15	Before (mmol/L):9.29±2.45 After (mmol/L): 8.99±2.58	-	11 subjects with hyperlipidemia and type 2 diabetes	Wheat bran
Chearskul et al. [18]	20 Men/women	30-70	crossover	3	10	FBS PPG	(FBS) Before (mmol/L):9.1±3.3 After (mmol/L):8.83±3.08PPG Before(mmol/L):17.36±4.02 After (mmol/L):17.13±4.42	8.8±0.26 After (mmol/L):9.05±2.41 (PPG) Before (mmol/L): 16.89±3.89 After (mmol/L):17.18±4.29	-		
Zhang et al [34]	30 Men/women	19-65	crossover	6	4	FBS	(FBS) Before (mmol/L): 5.1±0.79 After (mmol/L):5.11±1.14	(FBS) Before (mmol/L):5.08±0.81 After (mmol/L):5.27±0.87	-	30 subjects with schizophrenia and dyslipidemia	Placebo

Abbreviations: FBS, fasting blood glucose; PPG, post parandial glucose; SD, standard deviation

Table 2 Subgroup analysis of included RCTs in meta-analysis of the effect of glucomannan supplementation on FBG

Subgroup	WMD (95% Cl)'a	*P* value'*	P-heterogeneity	$I^{2\ b}$ (%)	P for between subgroup heterogeneity	studies foreach subgroup
Glucomannan dosage					<0.001	
3 g	-017 (-0.49, 0.14)	0.28	0.592	0.0		2
>3 g	-.82 (-1.83, 0.19)	0.11	<0.0001	86.5		4
Duration (week)					<0.001	
>8	-0.17 (-0.49, 0.14)	0.28	0.592	0.0		3
8	-.82 (-1.83, 0.19)	0.11	<0.0001	86.5		3
Health status					<0.001	
Obese or overweight	-0.17 (-1.16, -0.05)	0.12	0.98	0.0		2
Diabetic	-1.28 (-.2.54, -0.02)	0.04	0.36	70.0		4

Table 3 Riskof bias summary for included studies

Zhang et al 2020	Chearskul et al. 2007	VUKSAN et al. 1999	Chen et al. 2003	VUKSAN et al. 2000	Wood et al. (2007)	
+	-	+	+	+	+	Random sequence generation
+	?	?	?	?	?	Allocation concealment
+	-	+	+	+	+	Blinding of participants and personnel
+	-	?	?	?	+	Blinding of outcome assessment
+	+	?	?	+	+	Incomplete outcome data
-	+	-	+	-	-	Selective reporting
+	+	+	+	+	+	Other probable sources of biases

of bias. The quality assessment results of the articles are shown in Table 3.

Statistical analysis

To analyze the effect size of FBG and PPG the mean change and its SD for both the intervention and control groups were extracted.

The random-effects model (DerSimonian and Laird method) was used to find the relationships [32]. we calculated any within-group changes that were not reported by subtracting the baseline mean from the final mean value in each group. The SDs for mean differences were computed utilizing the following formula [33]:

$$\text{SD change} = \sqrt{[(\text{SD baseline})\ 2 + (\text{SD final})\ 2 - (2 \times 0.9 \times \text{SD baseline} \times \text{SD final})]}$$

For trials which only reported the Standard Error of the Mean (SEM), SD was calculated using the following formula: SD = $SEM\ x\ \sqrt{n}$, where "n" is the number of participants in each group. Serum concentrations of glucose were converted to the units that most frequently used (mmol/L). Heterogeneity between studies was assessed by Cochrane's Q test (significance point at *P*<0.1) and I^2 index.

Subgroup analysis based on the dose of supplementation, duration of the study, and health status of participants was performed to explore the potential sources of heterogeneity. Between subgroup heterogeneity was assessed using a fixed-effect model. Sensitivity analysis was conducted by removing each study, one by one, and recalculating the pooled evaluations. Begg's rank correlation test and Egger's regression asymmetry test were performed for detecting potential publication bias. Statistical analysis was conducted using STATA, Version 11.2 (Stata Corp,

College Station, TX). The threshold for statistical significance value was, *a priori*, defined as $P < 0.05$.

Results

Study selection

Of the 109 relevant publications that were initially identified in our search, 35 duplicate publications were removed. The remaining 74 records were screened, of which 61 irrelevant articles were omitted based on title and abstract screening. Then, 13 publications were selected for further evaluation of full texts. An additional 7 records were excluded for the following reasons: irrelevant studies ($n = 1$), studies without a suitable control group ($n = 3$), did not report adequate data of outcomes ($n = 2$), and complex intervention ($n=1$). Finally, a total of 6 eligible clinical trials were included in the meta-analysis. Out of 6 studies, 6 investigated the effect of glucomannan on FBG [18, 21, 23, 25, 29, 34], and 2 provided data on PPG [18, 21]. The flow diagram of the study selection process is presented in Fig. 1.

Study characteristics

Overall, 6 studies were included in the current meta-analysis. The general characteristics of the 6 eligible RCTs are shown in Table 1. All studies were published between 1999 and 2020 and conducted in Thailand [18], Taiwan [21], Canada [23, 25], the USA [29], and China [34]. Sample sizes varied from 11 to 30 people, totaling 124 subjects enrolled in the studies. The age of participants ranged from 19 to 77 years, and mean baseline BMIs varied from 25 to 30 kg/m^2 across studies. All trials were performed in men and women, except for the study by Wood et al. [29] which was just performed in men. Glucomannan was used as the intervention in the included studies, the dosage of glucomannan supplements ranged from 3 to 15 g/day, and the duration of glucomannan administration was from 4 to 12 weeks. Three trials [23, 25, 29] recommended a special diet concomitant to the glucomannan supplementation. Five trials employed a crossover design [18, 21, 23, 25, 34], while Wood et al. [29] was a parallel design. Two studies included overweight or obese people [23, 29] and three studies included people with T2DM [18, 21, 25] and one study included people with schizophrenia and dyslipidemia [34]. All included studies measured serum concentrations of FBG as the outcome and two trials also considered PPG as the outcome [18, 21].

Effects of glucomannan on FBG

The effect of glucomannan supplementation on FBG and PPG was assessed in 6 studies, including 124 participants. We observed that glucomannan supplementation significantly reduced FBG (weighted mean differences (WMD): -0.60 mmol/L, 95% CI: -1.16, -0.05; $P = 0.03$) compared with controls (Fig. 2). Because of a high between-study heterogeneity ($I^2 = 79.0\%$, $P < 0.0001$), we conducted subgroup analysis based on the dosage of intervention (3g/d and >3g/d), duration of the intervention (8 weeks and >8 weeks), and health status of the population (overweight or obese people and person with type 2 diabetes), which considerably removed the heterogeneity (Table 2). These analyses indicated that glucomannan supplementation significantly reduced FBG in people with type 2 diabetes (WMD: -1.28 mmol/L, 95% CI: -2.54, -0.02; $P=0.04$). No other subgroup yielded significant changes.

Effects of glucomannan on PPG

The effect of glucomannan supplementation on PPG was assessed in 2 studies, including 42 participants. We observed that glucomannan supplementation significantly reduced PPG (weighted mean differences (WMD): -3.60 mmol/L, 95% CI: -5.20, -2.00) compared with controls in study of chen et al. However We did not observe that glucomannan supplementation significantly reduced PPG (weighted mean differences (WMD): -0.52 mmol/L, 95% CI: -5.09, 0.95) compared with controls in study of chearskul et al.

Sensitivity analysis

To identify the influence of every trial on the total effect size, we excluded each study and repeated the analysis of the effect sizes for the influence of glucomannan on FBG (Fig. 3).

Publication bias

Begg's test and Egger's linear regression were implemented for the assessment of publication bias in the meta-analyses of glucomannan supplementation effects on FBG and PPG. These tests did not provide any evidence of publication bias; Begg's test ($P= 0.091$) and Egger's linear regression ($P=0.274$). Moreover, visual inspection of the funnel plots approved the result of Egger's linear regression.

Discussion

The systematic review and meta-analysis of current available RCTs, that investigated the effects of glucomannan supplementation on blood glucose, indicated a significant reduction
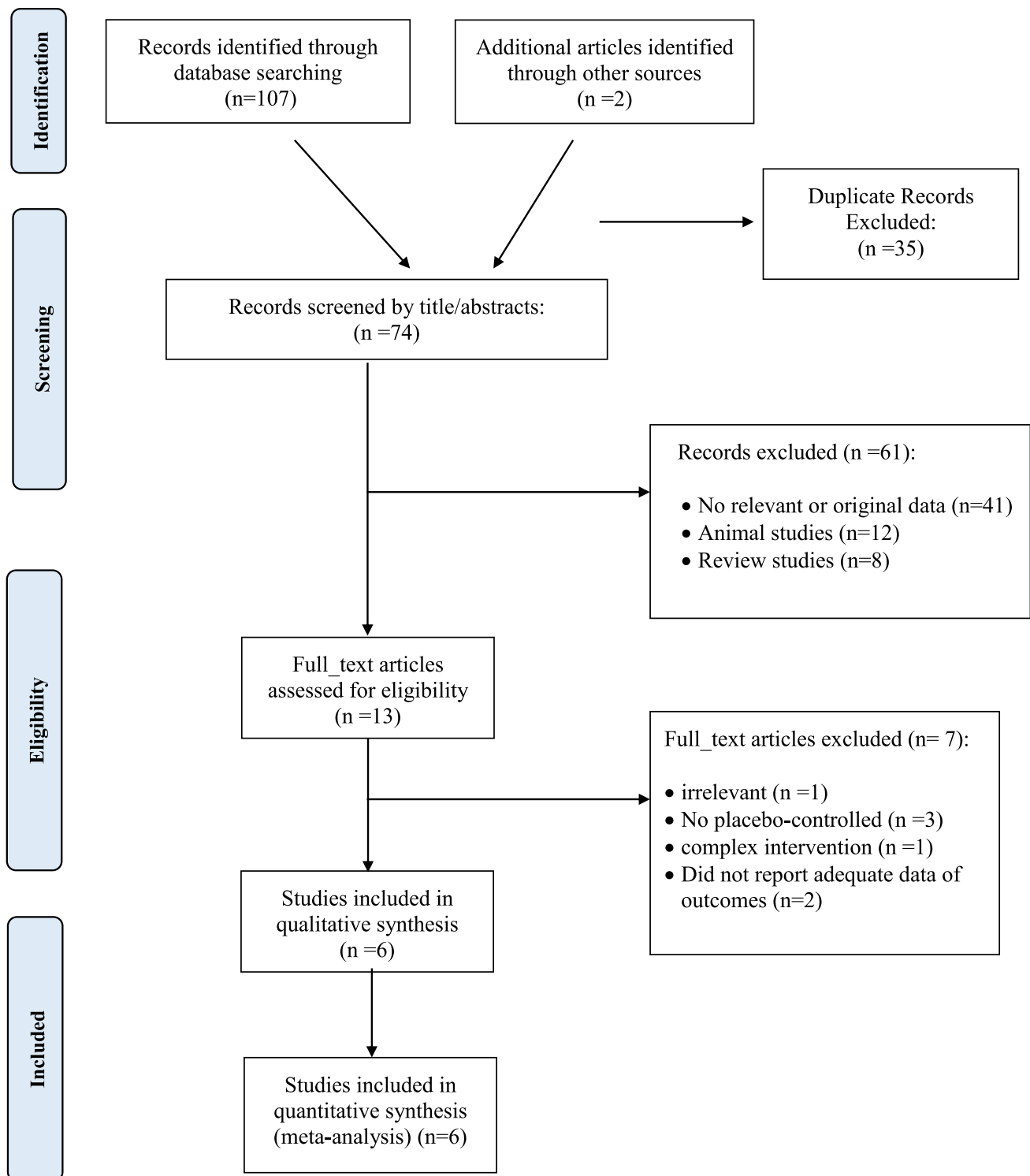
Fig. 1 Flow chart of the number of studies identified and selected into the meta-analysis

in FBG, but not in PPG, in response to glucomannan supplementation in adults. In addition, subgroup analysis showed glucomannan supplementation significantly decreased FBG in people with diabetes.

Concordant with our results, several previous clinical trials indicated no significant effects of glucomannan on blood glucose [17, 23, 29, 30, 34, 35]. However, a previous clinical trial reported that glucomannan supplementation

Fig. 2 Forest plot detailing weighted mean difference and 95% confidence intervals for the effect of glucomannan supplementation on FBG
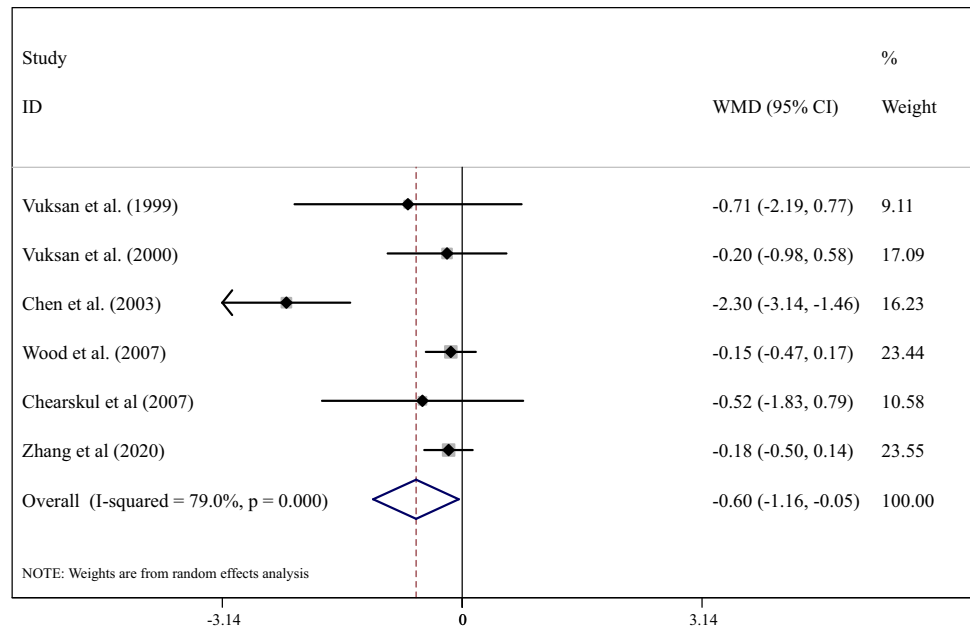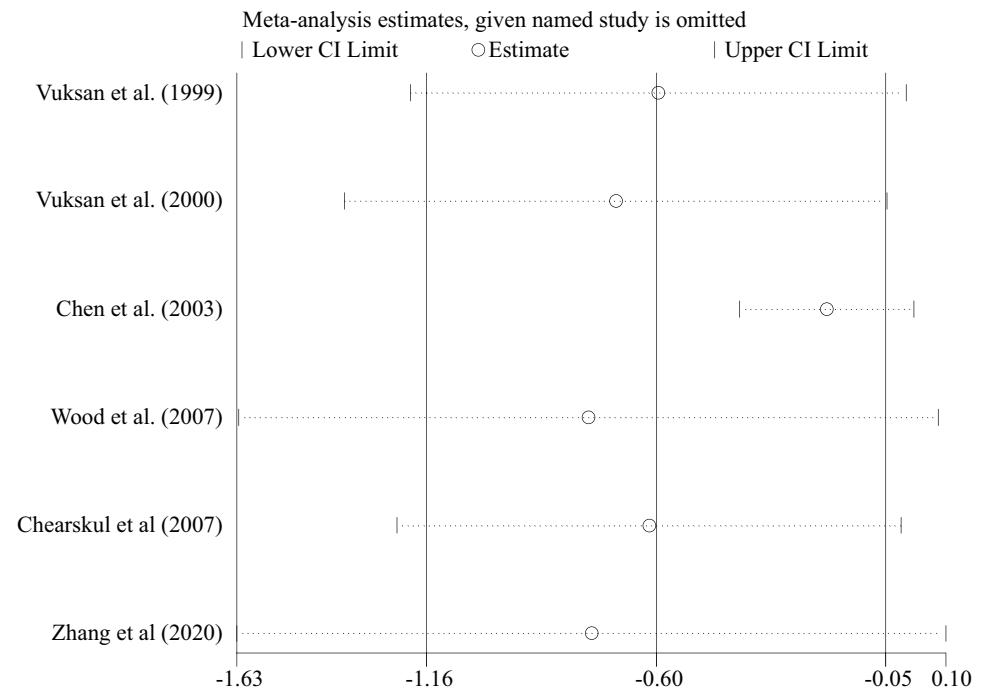

Fig. 3 Sensitivity analysis for the effect of glucomannan supplementation on FBG

significantly reduced FBG in people with diabetes [21], and in an animal study, the administration of Konjac Glucomannan (KGM) significantly decreased the levels of FBG in T2DM rats, compared with the control group [36]. Moreover, Devaraj et al., in a critical review, reported that KGM supplementation could reduce blood glucose in people with diabetes who ingested a KGM-rich diet daily (0.7 g KGM/100 kcal intake) [28].

However, in some previous clinical trials, supplementation with glucomannan did not elicit any significant effects on FBG in people with diabetes [18, 25, 26] and reported that glucomannan significantly reduced PPG in people with diabetes [18] and healthy adults [37], respectively. In Boers et al., the effect of high fiber flour flatbreads containing 2/4 g KM on PPG in normal-weight adults was assessed. Accordingly, flatbreads with 4 g KM decreased PPG ≥30

% ($p < 0.01$), while consumption of 2 g KM did not significantly alter in PPG vs. the control group [37]. In this present review, the effect of glucomannan on PPG was only assessed in doses <4 g/d, in addition to the limited number of studies (2 RCTs), which are plausible reasons for no significant effects of glucomannan supplementation on PPG being found. Further, one previous meta-analysis of RCTs showed a significant reduction of FBG in adults, but this effect was not indicated in people with diabetes [26].

T2DM is a chronic metabolic disease that is typically caused by Insulin Resistance (IR), leading to increases in FBG and PPG. Genetic factors, weight gain caused by overeating, and reduced physical activity are common causes of, or contributors to, diabetes. Overeating leads to unnecessary insulin secretion, which negatively affects the capacity of insulin secretory pancreatic β-cells, leading to obesity and IR [28]. Unfortunately, most medication used in the treatment of diabetes concurrently increase body weight, contributing to dysfunctional glycemic control [18]. Glucomannan is a soluble dietary fiber that absorbs high amounts of water and increases the volume of content in the stomach, accordingly, this can lead to delays in stomach emptying, increases in satiety, and prevention of overeating [26, 28]. Glucomannan is a low-calorie fiber that decelerates the absorption of dietary sugar and it may reduce hepatic glucose production [28, 29]. Indeed, in vitro analysis has indicated that glucomannan can improve glycemic control and can be a beneficial dietary supplement for people with diabetes. Other health benefits previously described for glucomannan include lowering of triglycerides, cholesterol, blood pressure, and body weight, in addition to promoting intestinal activity and preventing constipation. On the basis of the available data, glucomannan appears to be well tolerated, although it has been associated with various gastrointestinal complaints, including abdominal discomfort, diarrhea, loose stools, and flatulence [26, 28].

Strengths and limitations

In the present review, we assessed the effect of glucomannan supplementation on FBG and PPG in people with and without diabetes, which is novel. In addition, a previous meta-analysis only assessed the effects of glucomannan on FBG, but not PPG, while in this meta-analysis, we evaluated the effects of glucomannan supplementation on both FBG and PPG. However, despite the novelty of the present study, some limitations should be considered, including the limited number of RCTs. In addition, between included studies dosage of glucomannan, duration of studies, and types of intervention were different, however, we tried to consider them in the subgroup analysis. Based on the present study, it is clear that further RCTs, of varying duration and glucomannan dose, be conducted, particularly in relation to PPG.

Conclusion

The current study revealed that glucomannan supplementation can elicit a significant reduction in FBG in adults, but not in PPG. Moreover, subgroup analysis indicated that glucomannan supplementation may be particularly useful in people with diabetes.

Abbreviations CI: Confidence Intervals; DM: Diabetes mellitus; FBG: Fasting Blood Glucose; IDF: International Diabetes Federation; IR: Insulin Resistance; KGM: konjac Glucomannan; KM: Konjac-Mannan; LDL-C: Low-Density Lipoprotein Cholesterol; LEA: Lower-Extremity Amputations; MeSH: Medical Subject Headings; MNT: Medical Nutrition Therapy; PPG: Postprandial Glucose; PRISMA-P: Preferred Reporting Items for Systematic Review and Meta-Analysis Protocols; RCTs: Randomized Controlled Trials; SEM: Standard Error of the Mean; TC: Total Cholesterol; T2DM: Type 2 Diabetes Mellitus; WMD: Weighted Mean Differences

Declarations

Ethical approval and consent to participate Not applicable.

Consent for publication Not applicable.

Competing interests There is no competing interest in this study.

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