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Review

The effect of glucomannan on body weight in overweight or obese children and adults: A systematic review of randomized controlled trials

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ABSTRACT

Objective: Glucomannan (GM), a soluble fiber derived from the plant *Amorphophallus konjac*, is marketed as being helpful in reducing body weight. However, the data supporting this claim are scarce. The aim of this review was to systematically evaluate the effects of GM on body weight (BW) and body mass index (BMI) in otherwise healthy obese or overweight children and adults.
Methods: MEDLINE, EMBASE, CENTRAL, and Google Scholar databases were systematically searched up to June 2014 for randomized controlled trials (RCTs) assessing the effectiveness of GM versus placebo. The primary outcome measures were BW and BMI.
Results: Six eligible RCTs, only one of which performed in children, were included. In adults, three RCTs reported a significant reduction in BW in the GM group compared with the control group at the following different points during the intervention: At week 2 (mean difference [MD], 0.21 kg; 95% confidence interval [CI], 0.13–0.29); at week 4 (MD, 2.04; 95% CI, 0.52–3.56); at week 5 (MD, 1.3; 95% CI, 0.89–1.71); and at week 8 (MD, 3.17; 95% CI, 1.29–5.05). Only one RCT reported a beneficial effect at more than one point. None of the RCTs reported a favorable effect of GM on BMI.
Conclusions: In otherwise healthy overweight or obese adults, there is some evidence that in the short term GM may help to reduce BW, but not BMI. Data in children are too limited to draw any conclusions.

Introduction

The prevalence of obesity is reaching epidemic proportions. The first-line treatment (i.e., promoting a healthy diet and regular physical activity) is difficult to follow. There is continuous research into the modalities complementing lifestyle modifications that are helpful in the reduction of body weight (BW). Due to the prevalent use of over-the-counter products advertised as being effective in promoting weight loss, evidence is needed to confirm the claimed effects. One of the dietary supplements used is glucomannan (GM), a soluble fiber derived from the plant *Amorphophallus konjac*. Its potential mechanism of action is to increase satiety due to a delay in gastric empting caused by the "mass effect" of the viscous, gel-like mass forming in the stomach and slowing down gastrointestinal transit time [1]. In 2010, the European Food Safety Authority (EFSA) confirmed the positive effect of GM on the reduction of BW in overweight adults. The effect was achieved when ≥3 g of GM was supplemented daily in three doses of 1 g together with one to two glasses of water before a meal. However, at that time, the EFSA did not confirm the effect in children [2]. Similarly, an expert panel sponsored by the U.S. National Heart, Lung, and Blood Institute stated that "glucomannan does not significantly improve weight loss" in children [3].

In many European countries, GM is being widely marketed for the management of overweight or obesity. Given the scarcity of data and somewhat conflicting recommendations, we decided to conduct a randomized controlled trial (RCT) assessing the weight loss effect of GM supplementation in children. However, as previously noted, a systematic review of the literature should precede the conducting of new research [4].

This study was fully funded by the Medical University of Warsaw. HS was responsible for the study concept and supervision. BMZ and AC were responsible for acquisition and analysis and interpretation of data, as well as for drafting of the first draft of the manuscript. All the authors took part in the study design and critical revision of the manuscript for important intellectual content. The authors have no conflicts of interest to report.

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A systematic search has been conducted to evaluate the clinical effectiveness of supplementation with GM; the researchers concluded that it beneficially affected total cholesterol, low-density lipoprotein cholesterol, triglycerides and BW, but not high-density lipoprotein cholesterol or blood pressure [5]. As more than 5 y have passed since that review was published, we aimed to systematically evaluate the current evidence on the effect of GM supplementation on BW reduction as our main point of interest. We aimed to exclude studies referring to patients with diabetes mellitus, hypertension, or other chronic conditions requiring drug treatment. This decision was made to better define the effect of GM supplementation in the population we aimed to address in the planned interventional trial (i.e., obese or overweight, otherwise healthy individuals).

Materials and methods

Review protocol

The methods planned at each stage of this review were specified in advance and documented. However, formally, the review protocol was not registered.

Criteria for eligibility

Participants had to be children or adults who were overweight/obese (according to our definition). To increase the homogeneity, we decided not to include studies performed in populations with predefined diabetes mellitus or hyperglycemia, as well as in other populations with diagnosed diseases requiring drug treatment. All studies that assessed the effectiveness of GM versus placebo were considered for inclusion. GM administration of capsules, sachets, and bars or any other form was allowed. Studies that assessed GM as part of a complex drug or supplement, as well as those that compared GM with no intervention, were excluded.

The primary outcome measures were BW or BW change, as well as body mass index (BMI) or BMI change. The secondary outcomes (measured and calculated by the investigators) were percentage of BMI; body composition (body fat mass or fat-free mass); appetite assessed using visual analog scales or other tools, as defined by the investigators; and energy intake assessed using food records, or others, as defined by the investigators. Adverse effects were also an outcome of interest. All relevant RCTs, including those of crossover design, were considered for inclusion.

Search methods for identification of studies

A systematic literature search up to the end of January 2014 (and updated in June 2014, with no additional studies added), included search of the following databases: MEDLINE through PubMed, EMBASE (OVID), The Cochrane Central Register of Controlled Trials (CENTRAL, the Cochrane Library, Issue 5 of 12, May 2014), as well as Google Scholar. We also searched trial registries of ongoing trials. We used the "NCBI's Clinical Queries for Therapy" validated filter for identifying RCTs [6] (high sensitivity of 99%) combined with a search strategy using the Medical Subject Headings (MeSH) and text keywords of konjac OR mannan OR konjac-mannan OR konjac flour OR glucomannan OR glucoman* OR konjaku OR konnyaku OR konjac OR devils tongue OR voodoo lily OR snake palm OR amorphophallus konjac OR amorphophallus rivieri OR araceae (based on a previous review) [5]. No language restrictions or other filters were imposed. Letters to the editor, abstracts, and proceedings from scientific conferences were excluded from the analysis. Two authors (AC, BMZ) independently searched the databases and browsed the reference lists of identified papers and recent review articles.

Selection of studies

The authors independently screened the titles and abstracts of identified studies, and the full texts of potentially eligible studies were retrieved. Disagreements concerning the eligibility for inclusion were resolved by discussion.

Data collection and risk for bias assessment

All of the included studies were assessed independently by the authors (AC, BMZ) for methodological quality, using the Cochrane Collaboration tool for assessing risk of bias. The following were assessed: adequacy of sequence generation, allocation concealment, and the blinding of investigators, participants, and outcome assessors; whether incomplete outcome data were adequately addressed; the type of analysis; and the extent of loss to follow-up. The risk for bias was indicated as low by an answer of "yes," and as high, by an answer of "no" [7].

Data extraction

Standard data-extraction forms were used to obtain patient characteristics. If feasible for analysis, data were entered (AC, BMZ) into Review Manager (RevMan) (Version 5.2. Copenhagen: The Nordic Cochrane Centre, The Cochrane Collaboration, 2012).

Statistical methods, data synthesis

For dichotomous outcomes, the total number of participants and the number of participants who experienced the end point were extracted. For continuous outcomes, the mean difference (MD) with 95% confidence interval (CI) between the study groups was selected to show the difference. If data were presented as means ± SE, SDs were obtained by multiplying SEM by the square root of the sample size [$SD = SE * \sqrt{(n)}$], where "n" is the number of participants in a trial. If means without SD were reported, we contacted the authors for raw data. Weight was expressed in kg, and BMI was expressed in kg/m^2.

Heterogeneity was quantified by χ^2 and I^2, interpreted as the percentage of the total variation between studies, to assess whether there was more variation rather than would be expected by chance. The lower the percentage value, the lower the degree of heterogeneity. For pooling studies in a meta-analysis, if heterogeneity was not revealed, the fixed-effects model was used. If there was substantial heterogeneity (>50%), the analysis was based on the random-effects model.

We planned subgroup analyses based on factors that could potentially influence the treatment effect (i.e., administration of GM to children versus adults; administration of GM to otherwise healthy overweight/obese versus overweight/obese individuals with dyslipidemia). However, the aforementioned analyses were not performed due to the limited data available.

Results

Search results and characteristics of studies

Figure 1 presents a flowchart documenting the study selection process. Two registered studies were identified in clinicaltrials.gov (one ongoing and one of unknown status); for their characteristics, see Supplementary Table 1. The Table summarizes the key characteristics of the six included trials (RCTs). Five studies are of parallel design [8–12] and one is a crossover design [13]. The participants included were children (one RCT, N = 60) [10] and adults (five RCTs, N = 233). Data of n = 60 and n = 211 participants, respectively, were available for analysis. The studies were heterogeneous in regard to GM dose (from 1.24 to 3.99 g/d) and the duration of the intervention; the latter ranged from 5 [8] to 12 wk [11] in the parallel studies, and the duration of the intervention was 4 wk in the crossover RCT, with a 2-wk washout period [13]. The type of GM administration was consistent across the studies (capsules or tablets). Most of the studies included concurrent interventions, such as the following different types of diets: balanced [8], normocaloric [10], or carbohydrate-restricted [11]. In two trials, multivitamin supplementation was provided [8,11]. The authors of three studies encouraged maintaining current dietary intake [9,12,13]. In four trials, the authors advised no changes in physical activity [9,11–13]. The authors of three of the included RCTs reported on funding through industry [10–12], one by trust fund [9], and the remaining RCTs did not state the source of financial support [8, 13]. All potentially relevant studies not included in the systematic review are listed in Supplementary Table 2 with the reasons for exclusion given.

Methodological quality

An adequate description of the intention-to-treat analysis was provided in four studies [8–11]. Three of them were

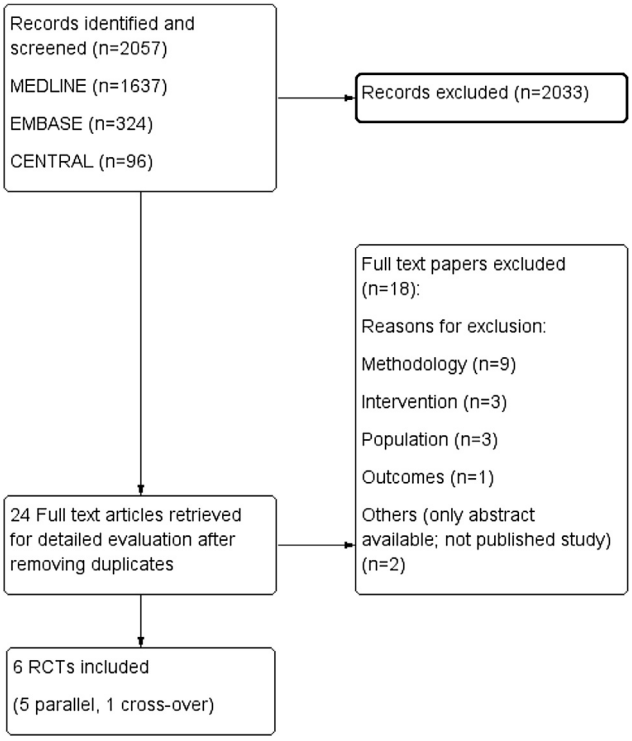

Fig. 1. Flowchart of study identification process.

classified as available case analyses. Only three trials used an adequate method for sequence generation [9,11,12], and only one adequately described the method to conceal allocation [9]. In the remaining trials, the methods used were evaluated as unclear. Only two trials reported satisfactorily on use of a double-blind design [11,12]; the blinding of three trials was partially described [8–10] and in one case, the description was unclear [13]. No additional sources of bias were identified. Apart from one study not reporting on a follow-up period [12], all authors included an adequate number of participants for the final analysis (≥80%). The authors of five studies [8–11,13] reported on the methods of compliance assessment, three [9–11] consistently stating that there were no differences between groups. The methodological quality assessment is summarized in Table 2 and in Supplementary Figure 1.

Effect of interventions

Body weight

Five RCTs, all conducted in adults, assessed the effect of GM supplementation on BW reduction at different points [8,9,11–13]. A graphic summary of the data is presented in Figure 2.

Body weight at week 2

One study (n = 47; GM 3.99 g/d) showed a small, but significant difference between groups in BW at week 2 in favor of the GM group (MD, 0.21; 95% CI, 0.13–0.29) [9]. Of note, the study did not report the CI or *P*-value, stating that the effect was insignificant. The CI value given here was self-calculated from data received by email communication with the author of the original publication and indicated statistical significance of the results at this time point [9].

Body weight at week 4

Two studies reported this outcome. One (n = 20; GM 3 g/d) found a significant difference between the study groups in BW at week 4 (MD, 2.04; 95% CI, 0.52–3.56) [12]. The other (n = 63, GM 3.9 g/d) presented mean BW at baseline and at week 4 without giving the mean difference between the study

Table 1
Characteristics of included trials

Reference (country)	Participants (age at enrollment)	Intervention	Concurrent intervention	Comparison	Duration of intervention (Follow-up)
Studies in adults					
Keithley 2013 (US)[9]	n/N = 47/53; 18–65 y (Mean 40.6), (BMI 25 to 35 kg/m^2)	GM 3.99 g/d (capsules) (n/N = 23/26)	Water (236 mL/8 oz) before meals; encouraged to maintain current diet	Placebo (inactive cellulose) (n/N = 24/27)	8 wk (8 wk)
Wood 2007 (US)[11]	n/N = 29/30; males, mean age 38.8 (14.4) y, BMI 25–35 kg/m^2	GM 3 g/d (capsules) (n/N = 14/15)	Water (236 mL/8 oz) before main meals; CHO- restricted diet (10% CHO, 60% fat)	Placebo (maltodextrin) (n/N = 15/15)	12 wk (12 wk)
Birketvedt 2005 (US, Norway)[8]	n/N = 52/60; women 30–60 y, BMI 25–30 kg/m^2 (mean 27.7)	GM 1.24 g/d (tablets) (n/N = 23/30)	Water (250 mL) 15 min before main meals; Balanced 1200-kcal diet	Placebo (not defined) (n/N = 29/30)	5 wk (5 wk)
Arvill 1995 (Sweden)[13]	n/N = 63/70; normo-/ hypercholesterolemic men, 15%–30% overweight, mean age 47 (8.2) y	GM 3.9 g/d (capsules) (n/N = 63/70)*	Glass of water 30 min before meals; Encouraged to maintain current dietary intake and physical activity	Placebo (cornstarch) (n/N = 63/70)*	4 wk
Walsh 1984 (US)[12]	n/N = 20/20; women ≥ 20% ideal weight	GM 3 g/d (capsules) (n = 10)	Water (236 mL/8 oz) 1 h before meals; Encouraged to maintain current dietary intake	Placebo (starch) (n = 10)	8 wk (8 wk)
Studies in children					
Vido 1993 (Italy)[10]	n/N = 60/60; Children with primary obesity (8–14 y)	GM 2 g/d (capsules) (n/N = 30/30)	2 glasses of water with GM; Normocaloric diet	Placebo (not defined) (n/N = 30/30)	2 mo (2 mo)

BMI, body mass index; CHO, carbohydrate; GM, glucomannan
* Cross-sectional study, groups combined.

Table 2
Methodological quality of included trials

Reference	Adequate sequence generation	Allocation concealment	Blinding of participants and personnel	Blinding of outcomes assessment	Incomplete outcome data addressed	Compliance assessed	Type of analysis	Follow-up (n/N)
Keithley 2013 [9]	Yes	Yes	Yes	Unclear	Yes	Yes	ACA	47/53 (89%)
Wood 2007 [11]	Yes (minimization)	Unclear	Yes	Yes	Yes	Yes	ACA	29/30 (97%)
Birketvedt 2005 [8]	Unclear	Unclear	Yes	Unclear	Unclear*	Yes	ACA	52/60 (87%)
Arvill 1995 [13]	Unclear	Unclear	Unclear	Unclear	Yes	Yes	PP	63/70 (90%)
Walsh 1984 [12]	Yes	Unclear	Yes	Yes	Unclear	No	Not stated	Not stated
Vido 1993 [10]	Unclear	Unclear	Yes	Unclear	Yes (not applicable)	Yes	ITT	60/60 (100%)

ACA, available case analysis; ITT, intention to treat; PP, per protocol. An answer of "yes" indicates a low risk for bias, and an answer of "no" indicates a high risk for bias
* Eight patients were excluded after randomization (before the intervention) because of weight loss; their data were not analyzed.

groups. Because an attempt to contact the authors to retrieve raw data failed, we were not able to calculate the effect as a mean difference, as reported in the other studies included. Thus, BW at week 4 as reported in the study is not presented in Figure 2 [13].

Body weight at week 5

One RCT (n = 52, GM 1.24 g/d) demonstrated a significant difference between groups for this outcome measure. Weight loss in the GM group was greater than that in the placebo group at week 5 (MD, 1.3; 95% CI, 0.89–1.71) [8].

Body weight at week 8

Two RCTs (total n = 67, GM 3.99 g/d and 3 g/d) reported on BW at this time point. One of them found a significant difference between the GM and placebo groups (MD, 3.17; 95% CI, 1.29–5.05) [9], whereas the other found no difference between groups [12]. Due to the very high heterogeneity of the trials ($I^2 = 91\%$), we did not pool the results.

Body weight at week 12

One study (n = 29, GM 3 g/d) assessed body weight after 12 wk of the intervention and observed no effect of GM supplementation (MD, −0.1; 95% CI, −1.96 to 1.76) [11]. Another study (n = 60; GM 2 g/d) conducted in children presented the outcome related to BW as a change in the percentage of overweight individuals after the GM supplementation. No significant effect of the intervention compared with placebo was observed [10].

Body mass index

BMI was reported in two RCTs. One observed a significant BMI reduction over time in both the GM and placebo groups after 12 wk of the intervention, but no difference between the study groups was noted (data not shown by the authors of the original publication) [11]. Participants in the second study [9] had BMI calculated after 2 wk and 8 wk. No difference between groups was noted at either of the time points (MD, 0.00; 95% CI, −1.81 to 1.81 and MD, 0.08; 95% CI, −1.71 to 1.87, respectively).

Body composition

Body composition parameters such as body fat, total fat mass, lean body mass, and waist and hip circumference were reported in two studies [9,11]. The latter additionally evaluated abdominal fat. For all aforementioned outcomes, no differences between the patients receiving GM and those being given placebo were observed.

Appetite

Two RCTs evaluated appetite. In one study, the investigators assessed children's subjective feelings of satiety after meals using a scale of "more, the same, or less satiety" [10]. No significant differences between groups were noted. A lack of a significant effect was also found in the second study, which assessed hunger/fullness with the use of standardized, 100-mm, visual analog scales [9].

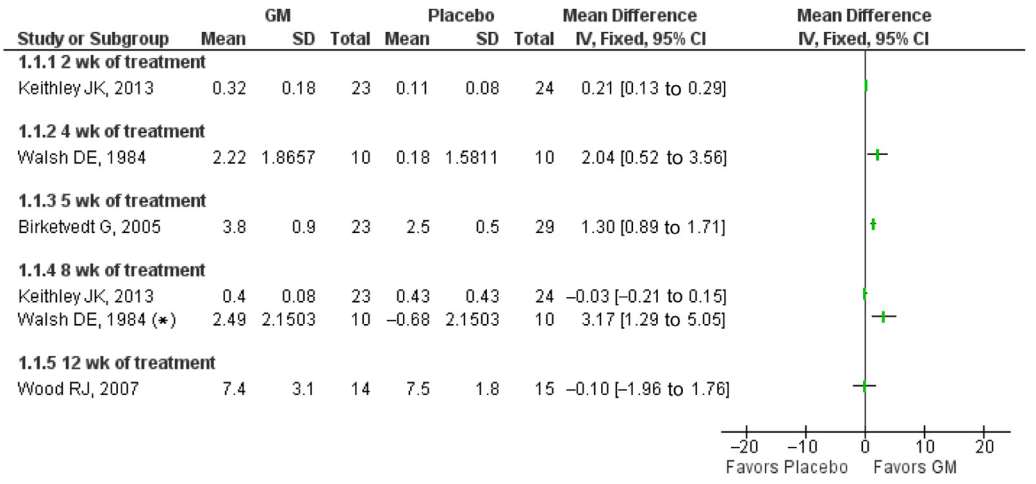

Fig. 2. Summary of results: body weight reduction at different time points.* Weight changes GM versus placebo: after 2, 4, 5, 8, and 12 wk of treatment. Negative number indicates weight increase. GM, glucomannan.

Energy intake

One RCT, with an intervention time of 12 wk, showed no significant reduction in energy intake between the GM and placebo groups, as assessed by 5- and 7-d weighted food records [11].

Adverse events

All included studies monitored participants for adverse events (AEs), and in four of them no AEs were observed [8,10,12,13]. In one RCT, patients in the GM group more frequently reported belching, bloating, and stomach fullness. The authors did not provide specific data to calculate statistical differences between groups, but the listed symptoms occurred approximately three times more often in participants receiving GM [9]. In another trial, participants had decreased appetite and increased thirst with similar frequency in both study groups. Diarrhea was reported significantly more often in the GM group than in the placebo group, whereas the constipation rate was similar between groups [11]. In both the just mentioned RCTs, a comparable dose of GM (3.99 versus 3 g/d) was used in the intervention groups. Of note, in one of the studies with no AEs reported, a significant increase in the triglyceride level was observed in the GM group but not in the placebo group ($P < 0.05$) [10].

Discussion

Principal findings

The objective of this review was to update evidence on the effect of GM supplementation on BW compared with administration of a placebo. A statistically significant reduction in BW was observed in three studies. In the first one, the transient effect was seen after 2 wk, but it did not last until the end of the 8-wk intervention [9]. Another study, on the contrary, reported a significant difference between groups in BW reduction in favor of GM after both 4 and 8 wk of treatment [12]. An effect after 5 wk of GM administration was found in one study. However, the methodology of this study may raise concern because 8 of 60 participants were excluded before the intervention because of weight loss that occurred after randomization, which should make one interpret the results with caution [8]. Moreover, despite the statistical significance reported in the studies showing a positive effect, its clinical relevance may be of doubt. Only one study reported a result that may seem meaningful for an individual patient (>3 kg difference between study groups). Additionally, it was the only study in which the weight reduction effect was consistent at different time points [12]. With regard to the long-term effects of the intervention, in the study with the longest duration of GM administration (12 wk), it did not alter BW significantly compared with placebo [11].

Strengths and weaknesses

This systematic review focused on a well-defined population of overweight or obese, but otherwise healthy, individuals. Unlike previous summaries of data on the effect of GM on BW, this review did not concern patients with hypertension or diabetes mellitus. Excluding individuals with these chronic conditions that often coexist with obesity forms a more homogeneous population and makes the assessment of the intervention's effect more credible. An additional strength of this review is that it targeted only the trials of randomized, placebo-controlled, double-blind design (i.e., those of an optimal methodology for evaluating the effect of an intervention). However, a review can only be as good as the studies included. The overall methodological quality of the trials was moderate, study groups were relatively small, and the time of the intervention was short, reaching 12 wk in one study only.

Comparison with previously published data

Three reviews assessing the efficacy of GM have been previously published, one of which was released at the time this publication was ready for submission. One study was a nonsystematic review and reported that in controlled trials ranging from 3 wk to 4 mo, doses of 2 to 4 g/d resulted in significant weight loss in mostly overweight and obese populations (no data shown by the authors) [1]. A second study was a systematic review and meta-analysis of 14 studies evaluating the effect of GM supplementation in a heterogeneous population (including obese, normal weight, diabetic, hypertensive, and osteoarthritic subjects). A statistically significant effect was shown for BW reduction (weighted MD, −0.79 kg; 95% CI, −1.53 to −0.05) [5]. The third review was a systematic review that was published at the completion of this review [14]. The search had been completed in March 2012, and it did not include the most recent RCT [1]. Furthermore, one RCT was incorrectly excluded because of the assumption that the participating patients were not overweight ("on average the group as a whole [N = 63] was 15 to 30% overweight") [13]. Additionally, a crossover RCT with no washout period was included in the review, which may be inappropriate [15]. A meta-analysis of eight RCTs revealed no statistically significant difference in weight loss between the GM and placebo groups. However, we identified some discrepancies in numeric data between two of the original studies [8,12] and those presented in the review. In one of them, weight gain was presented with an error that altered the statistical significance from significant to insignificant. It might have been due to changing the unit of weight from pounds, as reported in the original paper, to kilograms [12]. In another included study, incorrect numbering resulted in a changed MD (1.3; 95% CI, 0.89–1.71 to MD, 1.1; 95% CI, 0.49–1.71). However, the error in data transfer did not change the total pooled result for BW. Since the review by Sood et al. [5] came out, one additional RCT was published and was not included by Onakpoya et al. [9]. It can be pointed out that the population studied in the latter review was not homogeneous because trials with diabetic and hypertensive individuals were included; also populations of children and adults, as well as different measures of weight reduction (lb, kg, percentage overweight), were mixed together. Additionally, pooling data for BW measured at different time points (3 to 12 wk) might have been inappropriate, as well as combining data from crossover and parallel RCTs without using specific statistical tools [16]. In contrast to the aforementioned reviews, we did not conduct a meta-analysis, as we found combining results measured at different time points to be incorrect.

The intervention in most of the studies lasted <2 mo, which is a relatively short period for a drug trial [17]. We noted a small, but significant effect after 2 wk of the intervention, which diminished after another 6 wk in one of the RCTs [9], showing the importance of a longer intervention. In one of the Cochrane Collaboration reviews on treating obesity [17], the authors included only studies with drug therapy lasting a minimum of 3 mo and length of follow-up of another 3 mo. Using the aforementioned criteria, none of the studies included in our review would have been eligible, which underlines the need for better methodology and longer follow-up periods in further studies.

Conclusion

In overweight or obese, otherwise healthy adults, there is limited evidence that GM supplementation may help to reduce BW, but not BMI. Limited data do not allow one to draw any conclusions with regards to the effect of GM supplementation in children.

Studies of proper methodological design, consisting of larger study groups with longer (>12 wk) interventions and follow-up periods, are needed, especially in children, to establish whether the BW-reducing potential of GM is clinically significant.

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Supplementary Table 1. Summary of Ongoing Studies (clinicaltrials.gov) EU Clinical Trials Register (EUCTR) (https://www.clinicaltrialsregister.eu/): No eligible ongoing trial found

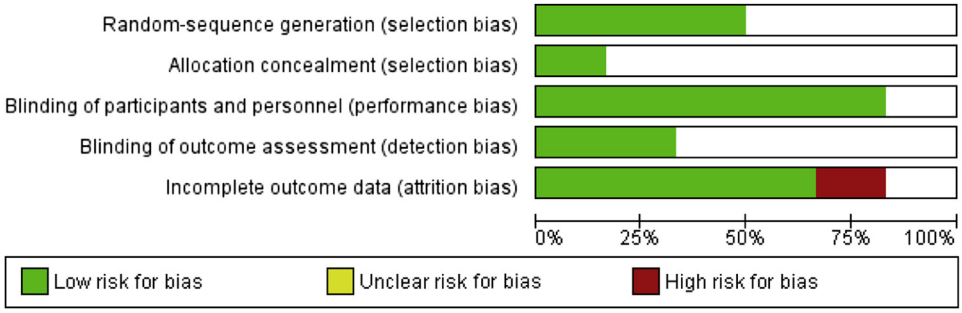
Trial Id	Title	Intervention 1	Intervention 2	Intervention 3	Intervention 4	Intervention 5	Status, June 2014	Completion date	Publication indexed
NCT01485718	Effect of Daily Glucomannanin Overweight Patients	Glucomannan 575 mg; 2 capsules 3 × /d; 30 min before meal	Placebo 2 capsules 3 × /d; 30 min before meal				Unknown	March 2012	No
NCT01657058	Effects of Mode of Administration of Soluble Fibre Blend on Glycemia, Appetite & Sensory Parameters (FI-FORM)	5 g soluble viscous fiber blend premixed into margarine (hydrophobic matrix) + white bread + jello	0 g soluble viscous fiber blend margarine + white bread +5 g Konjac fiber prehydrated in jello	Margarine + white bread (1/2 CHO) +5 g soluble viscous fiber blend hydrated in jello with glucose	Placebo 0 g soluble viscous fiber blend margarine + white bread + jello	Placebo 2 Margarine + white bread (1/2 CHO) +5 g soluble viscous fiber blend hydrated in jello with glucose	Ongoing, not recruiting	October 2014 (primary December 2012)	No

CHO, carbohydrate

Supplementary Table 2. Excluded studies

Excluded study	Reason for exclusion
Vuksan 2009 [1]	Intervention: GM vs. cellulose or novel viscous polysaccharide (no placebo)
Vasquez 2008 [2]	Intervention: A mixture of GM and garcinia cambogia
Salas-Salvadó 2008 [3]	Intervention: A mixture of GM and Plantago ovata husk
Livieri 1992 [4]	Methodology: No placebo control
Salvatoni 1991 [5]	Methodology: Non-RCT
Fanelli 1986 [6]	Methodology: Non-RCT
Teresawa 1979 [7]	Methodology: Non-RCT
Vita 1992 [8]	Methodology: No placebo control
Zhang 1990 [9]	Methodology: No placebo control
Martino 2005 [10]	Methodology: No placebo control
Stefanutti 1995 [11]	Methodology: Retrospective study
Doi 1981 [12]	Methodology: No placebo control; no outcomes of interest
Yoshida 2006 [13]	Population: Patients with chronic diseases requiring drug treatment
Cairella 1995 [14]	Population: Hyperglycemia
Guardamagna 2013 [15]	Population: Overweight/obese excluded
Martino 2013 [16]	Outcomes: No primary outcome of interest
Vido 1987 [17]	Abstract available only
Kaats 2004 [18]	Proceedings, data not published

GM, glucomannan; RCT, randomized controlled trial



Supplementary Figure 1. Graphic illustration of methodological quality of included studies.