Effect of glucomannan on plasma lipid and glucose concentrations, body weight, and blood pressure: systematic review and meta-analysis¹,²

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ABSTRACT

Background: Several clinical trials have investigated the impact of glucomannan on plasma lipids, body weight, fasting blood glucose (FBG), and blood pressure (BP), but have yielded conflicting results and had only modest sample sizes.

Objective: The objective was to perform a meta-analysis of randomized controlled trials of glucomannan to better characterize its impact on plasma lipids, FBG, body weight, and BP.

Design: A systematic literature search of MEDLINE, EMBASE, CINAHL, Web of Science, the Cochrane Library, and the Natural Medicines Comprehensive Database was conducted from the earliest possible date through November 2007. A random-effects model was used to calculate the weighted mean difference (WMD) and 95% CIs as the difference between the mean for the glucomannan and control groups. Standard methods for assessing statistical heterogeneity and publication bias were used.

Results: Fourteen studies (n = 531) met the inclusion criteria. The use of glucomannan significantly lowered total cholesterol [weighted mean difference (WMD): −19.28 mg/dL; 95% CI: −24.30, −14.26], LDL cholesterol (WMD: −15.99 mg/dL; 95% CI: −21.31, −10.67), triglycerides (WMD: −11.08 mg/dL; 95% CI: −22.07, −0.09), body weight (WMD: −0.79 kg; 95% CI: −1.53, −0.05), and FBG (WMD: −7.44 mg/dL; 95% CI: −14.16, −0.72). The use of glucomannan did not appear to significantly alter any other study endpoints. Pediatric patients, patients receiving dietary modification, and patients with impaired glucose metabolism did not benefit from glucomannan to the same degree.

Conclusions: Glucomannan appears to beneficially affect total cholesterol, LDL cholesterol, triglycerides, body weight, and FBG, but not HDL cholesterol or BP. Am J Clin Nutr 2008;88:1167–75.

INTRODUCTION

More than 50 million Americans are thought to suffer from the metabolic syndrome, which is characterized by a group of metabolic risk factors occurring in a single individual, including but not limited to abdominal obesity, atherogenic dyslipidemia, elevated blood pressure, and insulin resistance or glucose intolerance (1). Patients with the metabolic syndrome are at increased risk of coronary heart disease, stroke, and peripheral vascular disease as well as type 2 diabetes mellitus. According to the American Heart Association, the primary goal for the management of patients with the metabolic syndrome is to reduce their risk of cardiovascular disease and type 2 diabetes through smoking cessation and by reducing LDL cholesterol, blood pressure, body mass index, and glucose to recommended levels (1).

Glucomannan is a soluble fiber derived from Amorphophallus konjac and is available in numerous over-the-counter products such as Lipozene. Like other soluble fiber (oats, guar gum, pectin, and psyllium), glucomannan has been touted for its potential beneficial effects on the risk of coronary heart disease (2). Glucomannan is thought to prolong gastric emptying time, which increases satiety, reduces body weight, decreases the ingestion of foods that increase cholesterol and glucose concentrations, reduces the postprandial rise in plasma glucose, suppresses hepatic cholesterol synthesis, and increases the fecal elimination of cholesterol containing bile acids (2).

Several clinical trials (3–19) have investigated the impact of glucomannan on total cholesterol, LDL cholesterol, HDL cholesterol, triglycerides, body weight, fasting blood glucose (FBG), systolic blood pressure (SBP), or diastolic blood pressure (DBP), but have yielded conflicting results and had only modest sample sizes. Although previous meta-analyses assessing the effects of soluble fibers on these same endpoints have been published, none have evaluated glucomannan. Therefore, we conducted a meta-analysis of randomized controlled trials of glucomannan to better characterize its impact on various characteristics of the metabolic syndrome.

METHODS

Data sources

A systematic literature search of MEDLINE, EMBASE, CINAHL, Web of Science, the Cochrane Library, and the Natural Medicines Comprehensive Database was conducted from the earliest possible date through November 2007. A search strategy

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using the Medical Subject Headings (MeSH) and the text keywords konjac, mannan, konjac-mannan, konjac flour, gluco-
mannan, glucomannano, konjaku, konnyaku, konjac, devil’s tongue, voodoo lily, snake palm, Amorphophallus konjac, Amor-
phophallus rivieri, and Araceae was used. This search was then limited to clinical trials. No language restrictions were imposed.
In addition, a manual search of references from reports of clinical trials or review articles was performed to identify relevant trials. When applicable, efforts were made to contact investigators for clarification or additional data.

Study selection

Only randomized controlled trials of glucomannan that re-
ported efficacy data on at least one of the following components of the metabolic syndrome were included in the analysis: total cholesterol, LDL cholesterol, HDL cholesterol, triglycerides, body weight, FBG, SBP, or DBP. Both parallel and crossover trials were eligible for inclusion; however, crossover trials had to have a washout period of ≥2 wk to be included in the meta-
analysis. Studies using an active control were excluded.

Validity assessment

Because they are inherent controls of bias, randomization and double-blinding were used to assess the methodologic quality of included trials.

Data abstraction

Through use of a standardized data abstraction tool, 2 review-
ers independently collected data; disagreements were resolved through discussion. The following information was obtained from each trial: author identification, year of publication, study design, the abovementioned methodologic quality criteria, source of study funding, study population (including study in-
clusion and exclusion criteria), sample size, duration of patient follow-up, glucomannan dose and formulation used, use of concurrent dietary modification, effects on lipid variables (total cho-

esterol, LDL cholesterol, HDL cholesterol, and triglycerides), body weight, FBG, SBP, and DBP.

Statistical analysis

The mean change in lipid, glucose, body weight, and blood pressure variables from baseline was treated as a continuous variable and the weighted mean difference (WMD) was calcu-
lated as the difference between the mean in the glucomannan and control groups. A DerSimonian and Laird random-effects model (a variation on the inverse variance method, which incorporates an assumption that the different studies are estimating different, yet related, treatment effects) was used in calculating the WMD and its 95% CI. For parallel trials, net changes in each of these study variables were calculated as the difference (glucomannan minus control) in the changes (baseline minus follow-up) in these mean values (also referred to as the change score). For crossover trials, net changes were calculated as the mean difference in values at the end of the glucomannan and control periods. Be-
cause variances for net changes were not reported directly for most studies, they were calculated from CIs, P values, or indi-

vidual variances for intervention and control groups or periods. For parallel trials in which variance for paired differences was reported separately for each group, we calculated a pooled vari-
ance for net change using standard methods. When the variance for paired differences was not reported, we calculated it from variances at baseline and at the end of follow-up. As suggested by Follmann et al (20), we assumed a correlation coefficient of 0.5 between initial and final values. We assumed equal variances during the trial and between intervention and control groups.

The statistical analysis was performed by using StatsDi-
RECT statistical software (version 2.4.6; StatsDirect Ltd, Cheshire, United Kingdom), and MIX statistical software (freely accessible at www.mix-for-meta-analysis.info). A P value <0.05 was considered statistically significant for all analyses, except where otherwise specified.

Statistical heterogeneity was addressed using the Q Statistic, where a P value < 0.10 was considered representative of signif-
ificant statistical heterogeneity. Visual inspection of funnel plots and Egger’s weighted regression statistics were used to assess for the presence of publication bias. To assess the potential effect of any publication bias on the meta-analysis results, the Trim and Fill method was used, which uses funnel plot symmetry to esti-

mate the number of “missing” studies and the magnitudes of their effects. It then re-estimates the overall effect size after imputing any potentially “missing” studies into the meta-analysis to de-
termine whether the results of the original analysis were mark-
edly affected by publication bias.

Studies of poorer methodologic quality, such as open-label or crossover trials, may exhibit inaccurate treatment effects. Ex-
cluding them may result in increased internal validity but could reduce the external validity of the analysis. In addition, the se-
lection of a random-effects rather than a fixed-effects model in a meta-analysis is controversial. The use of a random-effects model in the calculation of CIs results in wider intervals and thus a more conservative estimate of treatment effects when com-
pared with a fixed-effects model. To reconcile these issues, sen-
sitivity analysis was conducted whereby the meta-analysis was reanalyzed excluding studies that were not double-blinded, ex-
cluding crossover studies, and finally using a fixed-effects model (Mantel-Haenszel methodology). We also conducted a sensiti-

vity analysis excluding the study by Venter et al (15), which uses a glucomannan product that may have also contained another pharmacologically active soluble fiber (pectin) and was limited to studies using total daily doses of ≤3 (ie, the minimum recom-

mended dose of soluble fiber by the Food and Drug Administra-
tion) (21) and 10 g glucomannan/d (ie, the maximum practical dose of soluble fiber) (22). Additionally, subgroup analyses were performed whereby the effects of glucomannan on study end-
points were assessed separately in subjects with impaired glu-
ceose metabolism [type 2 diabetes mellitus or impaired glucose tolerance (IGT)], in obese subjects, in adults, and in children and in studies using or not using concurrent dietary modifications.

RESULTS

The initial search yielded 3109 potential literature citations. Of these, only 24 were clinical trials in humans. On review of references from identified studies, an additional 5 studies poten-
tially meeting our inclusion criteria were identified, bringing the total number of studies for full-text review to 29. Fifteen of the 29 studies were excluded for the reasons given in Figure 1. Of note, the study by Vita et al (17) met all the inclusion criteria but could not be included because measures of variation around effect were not provided and could not be estimated from the data provided.
Two other studies (18, 19) did not meet our criteria for inclusion because of the lack of a washout period of adequate duration. Thus, a total of 14 randomized controlled trials (3–16) that evaluated 531 subjects were included in this meta-analysis (Table 1). Two of these studies (5, 15) reported the results of 2 heterogeneous and mutually distinct populations separately in their articles; therefore, we opted to include each of these analyses in our meta-analysis separately. Eight of the studies were conducted using a parallel study design (4, 6, 7, 11–14, 16), whereas the other 6 studies used a crossover design. All the crossover studies used a 2-wk washout period, except for the study by Yoshida et al (5), which used a 4-wk washout period. Each of the studies enrolled a relatively small number of patients (median sample size: 20 subjects; range: 11–110 subjects) and had a short length of follow-up (median duration: 5–8 wk; range: 3–16 wk). All of the studies used placebo as the control, except for 3 studies (6, 13, 14) that used diet only as the control. Each of the included studies evaluated patients having at least one, if not, multiple, constituents of the metabolic syndrome, including type 2 diabetes mellitus or impaired glucose tolerance (3, 5, 8, 9), hyperlipidemia (5, 6, 9–11, 14, 15), hypertension (9), or obesity (4, 7, 11–13, 15). The dosage range of glucomannan used in the included studies ranged from 1.2 to 15.1 g/d and were administered in various forms, such as capsules, tablets, bars, biscuits, and refined konjac meal. Nine of the studies (3, 4, 6–9, 11–13) administered glucomannan along with some type of dietary modification. Of the 14 studies, 4 were not double-blinded (3, 6, 13, 14). More than half (57.1%) of the studies stated they were funded through industry (4–6, 8, 9, 12, 15, 16), and the remainder did not report their funding source (3, 10, 11, 13, 14).

The meta-analysis showed that the use of glucomannan appeared to statistically significantly lower total cholesterol, LDL cholesterol, triglycerides, body weight, and FBG (Figure 2). Of these other endpoints, only DBP displayed statistical heterogeneity ($P = 0.03$).

Visual inspection of funnel plots (data not shown) could not rule out publication bias for many of the analyses. Review of Egger’s weighted regression statistics suggested that publication bias was unlikely for all analyses ($P > 0.19$ for all). We recalculated effect size estimates using the Trim and Fill methodology, only our conclusion regarding glucomannan’s effect on HDL cholesterol was significantly altered. In this case, the Trim and Fill analysis suggests that as many as 3 studies could potentially exist but were masked by publication bias and, when factored in, that glucomannan may have a small but statistically significant detrimental effect on HDL cholesterol (reduction of 2.01 mg/dL).

The results of subgroup and sensitivity analyses are presented in Table 2. Similar benefits in pediatric patients compared with adults (or the base-case analysis) were not seen. In addition, it appears that glucomannan does not have as robust an effect on triglycerides in patients with impaired glucose metabolism or when used in conjunction with dietary modification, the benefits of glucomannan on weight loss are enhanced by dietary modification, and patients with impaired glucose metabolism do not reap the hypoglycemic benefits of glucomannan. After the exclusion of studies that used a double-blind methodology, the analyses for triglycerides and FBG went from being of borderline statistical significance to borderline nonsignificance; however, because the effect size estimates for both analyses remained relatively constant, these changes were likely a result of decreased statistical power. No other subgroup or sensitivity analyses resulted in changes in overall study conclusions.

**DISCUSSION**

In this meta-analysis of 14 randomized controlled trials, patients receiving glucomannan had statistically significantly lower total cholesterol, LDL cholesterol, triglycerides, body weight, and FBG (Figure 2). Statistical heterogeneity was observed only in the body weight endpoint ($Q$ statistic $P = 0.04$). The use of glucomannan did not appear to significantly alter any of the other study endpoints (Table 2). Of these other endpoints, only DBP displayed statistical heterogeneity ($P = 0.03$).
<table>
<thead>
<tr>
<th>Study design</th>
<th>Patient population</th>
<th>Baseline lipids</th>
<th>Baseline weight</th>
<th>Baseline FBG</th>
<th>Glucomannan dosing and dosage form</th>
<th>Concurrent diet modification</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chearskul et al (3), 2007 (n = 20)</td>
<td>Type 2 DM</td>
<td>TC = 191, 193; LDL-C = 92, 96; HDL-C = 53, 53; TG = 267, 243</td>
<td>70</td>
<td>69</td>
<td>163</td>
<td>159</td>
</tr>
<tr>
<td>Wood et al (4), 2007 (n = 29)</td>
<td>Obese men</td>
<td>TC = 179, 177; LDL-C = 115, 112; HDL-C = 41, 42; TG = 116, 119</td>
<td>94</td>
<td>93</td>
<td>92</td>
<td>93</td>
</tr>
<tr>
<td>Yoshida et al (5), 2006 (n = 29)</td>
<td>Hyperlipidemic with or without DM</td>
<td>TC = 221, 225; LDL-C = 149, 152; HDL-C = 40, 41; TG = 147, 165</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td>Martino et al (6), 2005 (n = 40)</td>
<td>Hyperlipidemic children</td>
<td>TC = 243, 254; LDL-C = 172, 176; HDL-C = 54, 59; TG = 96, 90</td>
<td>30</td>
<td>30</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td>Birkeveth et al (7), 2005 (n = 52)</td>
<td>Overweight women</td>
<td>TC = NA; LDL-C = NA; HDL-C = NA; TG = NA</td>
<td>79</td>
<td>77</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td>Vuksan et al (8), 2000 (n = 11)</td>
<td>IGT</td>
<td>TC = 240, 232; LDL-C = 151, 147; HDL-C = 39, 39; TG = 248, 257</td>
<td>81</td>
<td>81</td>
<td>122</td>
<td>119</td>
</tr>
<tr>
<td>Vuksan et al (9), 1999 (n = 11)</td>
<td>Hypertensive, hyperlipidemic type 2 DM</td>
<td>TC = 236, 225; LDL-C = 150, 138; HDL-C = 41, 40; TG = 224, 238</td>
<td>86</td>
<td>86</td>
<td>173</td>
<td>167</td>
</tr>
<tr>
<td>Arvill and Bodin (10), 1995 (n = 63)</td>
<td>Hyperlipidemic men</td>
<td>TC = 268, 260; LDL-C = 177, 176; HDL-C = 46, 48; TG = 210, 255</td>
<td>90</td>
<td>90</td>
<td>NA</td>
<td>NA</td>
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</tbody>
</table>
### TABLE 1 (Continued)

<table>
<thead>
<tr>
<th>Study design</th>
<th>Patient population</th>
<th>Baseline lipids&lt;sup&gt;2&lt;/sup&gt;</th>
<th>Baseline weight</th>
<th>Baseline FBG</th>
<th>Glucomannan dosing and dosage form</th>
<th>Control group</th>
<th>Concurrent diet modification</th>
</tr>
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<tbody>
<tr>
<td>Cairella and Marchini (11), 1995 &lt;i&gt;(n = 15)&lt;/i&gt;</td>
<td>Hyperlipidemic obese women</td>
<td>TC = 249, 246; LDL-C = NA; HDL-C = NA; TG = NA</td>
<td>NA</td>
<td>NA</td>
<td>3.87 g/d as capsules</td>
<td>Placebo (inactive contents)</td>
<td>Low calorie (1200 kcal, 29% fat, 25% protein, 46% CHO)</td>
</tr>
<tr>
<td>Vido et al (12), 1993 &lt;i&gt;(n = 60)&lt;/i&gt;</td>
<td>Obese children</td>
<td>TC = 194, 175; LDL-C = NA; HDL-C = NA; TG = 73, 107</td>
<td>NA</td>
<td>NA</td>
<td>2 g/d as capsules</td>
<td>Placebo (contents NA)</td>
<td>Well balanced normocaloric diet</td>
</tr>
<tr>
<td>Livieri et al (13), 1992 &lt;i&gt;(n = 53)&lt;/i&gt;</td>
<td>Obese children</td>
<td>TC = 187, 184; LDL-C = NA; HDL-C = NA; TG = 82, 83</td>
<td>NA</td>
<td>NA</td>
<td>2-3 g/d as capsules</td>
<td>Diet only</td>
<td>Balanced diet (30% fat, 15% protein, 55% CHO)</td>
</tr>
<tr>
<td>Zhang, et al (14), 1990 &lt;i&gt;(n = 110)&lt;/i&gt;</td>
<td>Hyperlipidemic adults</td>
<td>TC = 205, 199; LDL-C = 56, 109; HDL-C = 47, 118; TG = 222, 253</td>
<td>NA</td>
<td>NA</td>
<td>5-10 g/d as refined foods</td>
<td>Diet only</td>
<td>Ordinary diet</td>
</tr>
<tr>
<td>Venter et al (15), 1987 &lt;i&gt;(n = 18)&lt;/i&gt;</td>
<td>Hyperlipidemic adults</td>
<td>TC = 274; LDL-C = NA; HDL-C = NA; TG = NA</td>
<td>74</td>
<td>74</td>
<td>4.5 g/d as capsules</td>
<td>Placebo (cornstarch, quantity NA)</td>
<td>Normal diet</td>
</tr>
<tr>
<td>Walsh et al (16), 1984 &lt;i&gt;(n = 20)&lt;/i&gt;</td>
<td>Obese women</td>
<td>TC = 198; LDL-C = 125; HDL-C = NA; TG = NA</td>
<td>84</td>
<td>83</td>
<td>3 g/d as capsules</td>
<td>Placebo (500 mg starch)</td>
<td>Previously established eating patterns</td>
</tr>
</tbody>
</table>

<sup>1</sup> CHO, carbohydrate; NA, not available; TC, total cholesterol; TG, triglycerides; C, cholesterol; DM, diabetes mellitus; IGT, impaired glucose tolerance; NCEP, National Cholesterol Education Program; FBG, fasting blood glucose. To convert values for cholesterol from mg/dL to mmol/L, multiply by 0.0286; to convert values for TG from mg/dL to mmol/L, multiply by 0.01129.

<sup>2</sup> First value given is for the treatment group at baseline; second value is for the control group at baseline. Venter et al (15) and Walsh et al (16) reported only means for entire study cohort.

<sup>3</sup> Glucomannan bar composition: 360 kcal total energy, 58.4 g CHO, 6.3 g protein, 11.2 g fat, 2.3 g saturated fat, 274.9 mg Na, 14.9 g fiber, 768.6 IU vitamin A, 4 mg vitamin C, 2.3 mg Fe, and 42.6 mg Ca.

<sup>4</sup> Placebo bar composition: 399 kcal total energy, 66.7 g CHO, 8.3 g protein, 11.2 g fat, 2.0 g saturated fat, 308.6 mg Na, 5 g fiber, 852.7 IU vitamin A, 5.3 mg vitamin C, 2.6 mg Fe, and 48.2 mg Ca.

<sup>5</sup> Glucomannan biscuit composition (g/100 g): 6.2 g protein, 13.9 g fat, 61.2 g CHO, 1.3 g ash, 2.3 g dietary fiber, 944 kJ/100 g energy; placebo biscuit composition (g/100 g): 6.8 g protein, 14.4 g fat, 66.5 g CHO, 1.4 g ash, 2.8 g dietary fiber, and 1011 kJ/100 g energy.
weight, and FBG after treatment than did control patients; however, the use of glucomannan did not appear to alter HDL cholesterol or either systolic or diastolic blood pressure. Our analysis could only show trends toward benefits with glucomannan in children. We also found that glucomannan does not have as robust an effect on triglycerides in patients with impaired glucose metabolism or when used in conjunction with dietary modification, that the benefits of glucomannan on weight loss are enhanced by dietary modification, and that patients with impaired glucose metabolism do not reap the hypoglycemic benefits of glucomannan. Because of the smaller number of studies included in the abovementioned subgroup analyses, they should be interpreted with caution and considered hypothesis-generating only.

It would appear that the greatest potential cardiovascular benefits from glucomannan are due to its effect on lipids. Studies have shown that for each 1-mg/dL reduction in a patient’s LDL-cholesterol concentration, their relative risk of having a coronary heart disease event is decreased by 1%. Thus, the 16-mg/dL reduction in LDL cholesterol seen in our meta-analysis with glucomannan is not only statistically significant, but likely is also clinically significant (23).

Two previous meta-analyses (22, 24) evaluated the hypocholesterolemic effects of pectin, psyllium, oats, and guar gum with reductions in total and LDL cholesterol ranging from 7% to 15% and 7% to 10%, respectively, along with a detrimental but small effect on HDL cholesterol. These effects on total cholesterol, LDL cholesterol, and HDL cholesterol are similar to those observed in our meta-analysis, which suggests that this may be a class effect of soluble fibers. Interestingly, whereas previous meta-analyses did not demonstrate reductions in triglycerides with soluble fibers other than glucomannan, our meta-analysis showed a statistically significant 11-mg/dL reduction. The reason behind glucomannan’s ability to preferentially lower triglycerides compared with other soluble fibers is not known, but may be related to its higher viscosity and thus its greater ability to alter the metabolic pathways of hepatic cholesterol and lipoprotein metabolism (2).

Glucomannan is commonly touted in the United States as an effective over-the-counter weight-loss supplement (25, 26). In studies lasting a mean of 5.2 wk, our meta-analysis found that there was a statistically significant but small reduction in weight of 0.79 kg (~1%) with glucomannan. While studied over a slightly longer period of time, orlistat (Alli) the only over-the-counter weight-loss treatment approved by the Food and Drug

**FIGURE 2.** Effect of glucomannan on characteristics of the metabolic syndrome. A: Total cholesterol (n = 286 glucomannan, n = 273 control); B: LDL cholesterol (n = 146 glucomannan, n = 148 control); C: HDL cholesterol (n = 138 glucomannan, n = 140 control); D: Triglycerides (n = 301 glucomannan, n = 287 control); E: Body weight (n = 186 glucomannan, n = 193 control); F: Fasting blood glucose (n = 61 glucomannan, n = 62 control). A DerSimonian and Laird random-effects model was used in calculating the weighted mean difference and its 95% CI. To convert values for cholesterol from mg/dL to mmol/L, multiply by 0.0286; to convert values for triglycerides from mg/dL to mmol/L, multiply by 0.01129. *Study reported separate analyses of 2 heterogeneous and mutually exclusive patient populations; thus, each analysis was treated as a separate study in this meta-analysis.
TABLE 2
Results of the meta-analysis of randomized controlled trials that evaluated glucomannan

<table>
<thead>
<tr>
<th>TC</th>
<th>LDL-C</th>
<th>HDL-C</th>
<th>TG</th>
<th>Body weight</th>
<th>FBG</th>
<th>SBP</th>
<th>DBP</th>
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<tbody>
<tr>
<td>mg/dL</td>
<td>mg/dL</td>
<td>mg/dL</td>
<td>mg/dL</td>
<td>kg</td>
<td>mg/dL</td>
<td>mm Hg</td>
<td>mm Hg</td>
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<tr>
<td>All studies (all doses)</td>
<td>(24.30, 14.26)</td>
<td>(18.89, 10.07)</td>
<td>(3.98, 2.39)</td>
<td>(11.98, 12.10)</td>
<td>(1.53, 0.05)</td>
<td>(1.41, 0.81)</td>
<td>(2.77, 12.83)</td>
</tr>
<tr>
<td>(n = 13 studies)</td>
<td>(n = 15 studies)</td>
<td>(n = 7 studies)</td>
<td>(n = 6 studies)</td>
<td>(n = 12 studies)</td>
<td>(n = 4 studies)</td>
<td>(n = 3 studies)</td>
<td>(n = 4 studies)</td>
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<tr>
<td>Fixed-effects model</td>
<td>(24.30, 14.26)</td>
<td>(18.89, 10.07)</td>
<td>(3.98, 2.39)</td>
<td>(11.98, 12.10)</td>
<td>(1.53, 0.05)</td>
<td>(1.41, 0.81)</td>
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<td>(n = 12 studies)</td>
<td>(n = 4 studies)</td>
<td>(n = 3 studies)</td>
<td>(n = 4 studies)</td>
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<tr>
<td>Trim and fill</td>
<td>(24.30, 14.26)</td>
<td>(18.89, 10.07)</td>
<td>(3.98, 2.39)</td>
<td>(11.98, 12.10)</td>
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<td>(n = 6 studies)</td>
<td>(n = 12 studies)</td>
<td>(n = 4 studies)</td>
<td>(n = 3 studies)</td>
<td>(n = 4 studies)</td>
</tr>
<tr>
<td>Excluding crossover studies</td>
<td>(24.30, 14.26)</td>
<td>(18.89, 10.07)</td>
<td>(3.98, 2.39)</td>
<td>(11.98, 12.10)</td>
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<td>(n = 12 studies)</td>
<td>(n = 4 studies)</td>
<td>(n = 3 studies)</td>
<td>(n = 4 studies)</td>
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<tr>
<td>Excluding studies not double-blinded</td>
<td>(24.30, 14.26)</td>
<td>(18.89, 10.07)</td>
<td>(3.98, 2.39)</td>
<td>(11.98, 12.10)</td>
<td>(1.53, 0.05)</td>
<td>(1.41, 0.81)</td>
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(Continued)
### Table 2 (Continued)

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<th>TC (mmol/L)</th>
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<th>HDL-C (mmol/L)</th>
<th>TG (mmol/L)</th>
<th>Body weight (kg)</th>
<th>FBG (mmol/L)</th>
<th>SBP (mmHg)</th>
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</table>

A DerSimonian and Laird random-effects model was used to calculate weighted mean differences and their 95% CIs (in parentheses). To convert values for cholesterol from mg/dL to mmol/L, multiply by 0.0286; to convert values for TG from mg/dL to mmol/L, multiply by 0.01129. DBP, diastolic blood pressure; FBG, fasting blood glucose; TC, total cholesterol; TG, triglycerides.

### CONCLUSIONS

Glucomannan appears to beneficially affect total cholesterol, LDL cholesterol, triglycerides, body weight, and FBG, but not HDL cholesterol or blood pressure. Larger individual studies following patients for longer periods of time and evaluating both safety and efficacy are warranted and needed.

The authors’ responsibilities were as follows—NS, WLB, and CIC: responsible for analyzing the data, interpreting the data and results, and writing the manuscript; and WLB and CIC: responsible for formulating the research question, conducting the literature search, interpreting the data and results, and writing the manuscript. We certify that none of the material in this manuscript was previously published, and we have no conflicts to declare germane to this manuscript.

### REFERENCES

1. Grundy SM, Cleeman JI, Daniels SR, et al. Diagnosis and management of the metabolic syndrome: an American Heart Association/National...


