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Effects of Low Glycemic Index Diets on Gestational Diabetes Mellitus

A Meta-Analysis of Randomized Controlled Clinical Trials

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Abstract: Studies of the effects of low glycemic index (LGI) diets on gestational diabetes mellitus (GDM) have reported conflicting findings.

The aim of the study was to evaluate the results of randomized controlled trials (RCTs) that investigated the effects of LGI diets with and without added dietary fiber (DF) on maternal and neonatal outcomes in GDM patients.

We searched the MEDLINE, EMBASE, EBSCO, Springer, Ovid, and Cochrane Library databases for studies of the effects of LGI diets in GDM patients. We performed a meta-analysis of the effects of the LGI diets with and without added dietary fiber (DF) on GDM outcomes. Risk ratios (RR) and 95% confidence intervals (CIs) were calculated using random- and fixed-effects models.

Five RCTs involving 302 participants were included in our meta-analysis. No statistically significant differences in the risks of cesarean section delivery, large for gestational age, and small for gestational age were observed. The risk of macrosomia in the LGI groups was significantly lower (RR = 0.27; 95% CI: 0.10–0.71; $P = 0.008$) than that in the control groups. Our subgroup analysis of the effects of DF showed that LGI diets with an increased level of DF, relative to the control diet, reduced the risk of macrosomia beyond that of the LGI diets alone (RR: 0.17 vs 0.47, respectively). The subgroup analysis also showed that LGI diets in which the level of DF was approximately equivalent to that in the control diets significantly reduced the risk of insulin usage (RR = 0.69; 95% CI: 0.52–0.92; $P = 0.01$).

The LGI diets reduced the risk of macrosomia in GDM patients, and LGI diets with added DF reduced the risk of macrosomia further. The LGI diets with levels of DF approximately equivalent to that in the control diets reduced the risk of insulin usage in GDM patients.

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Abbreviations: CIs = confidence intervals, CSD = cesarean section delivery, DF = dietary fiber, GDM = gestational diabetes mellitus, GI = glycemic index, LGA = large for gestational age, LGI = low glycemic index, RCTs = randomized controlled trials, RR = risk ratios, SGA = small for gestational age, T2DM = Type 2 diabetes mellitus.

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INTRODUCTION

The diagnosis of gestational diabetes mellitus (GDM) is currently given to pregnant women with blood glucose levels in the 90th percentile of the population distribution for women.[1] These criteria were established to identify women with a $\geq$ 50% risk of developing type 2 diabetes mellitus (T2DM) after pregnancy.[2] Although the severity of hyperglycemia in some GDM patients would not warrant a diagnosis of T2DM outside of pregnancy, a 7-fold increase in the risk of T2DM following pregnancy is associated with GDM, with the majority of GDM patients developing T2DM within 3 years of childbirth.[3]

The incidence of GDM is closely associated with the frequency of T2DM in the general population,[4] and the incidence of T2DM is increasing on a global scale.[5] Trends toward diets high in fat, the increased prevalence of obesity, an increase in more sedentary lifestyles, and trends toward increasing maternal age have also contributed to increases in the incidence of GDM worldwide, especially in developing countries.[6,7] A variety of adverse outcomes, both for the mother and the newborn, are associated with GDM. Adverse maternal outcomes include cesarean section delivery and postpartum T2DM, and adverse neonatal outcomes include fetal mortality,[1] premature birth, and a diagnosis of macrosomia or large for gestational age.[8,9]

Treatments for GDM include diet control, hypoglycemic drugs, and insulin therapy. The use of hypoglycemic drugs and insulin therapy have certain limitations in the clinical care of pregnant women,[10–12] whereas no significant side effects have been reported for diet control. The aim of diet control is to maintain blood glucose levels within the normal range by optimizing the carbohydrate composition of the diet while avoiding hypoglycemia or ketosis due to an excessive reduction in carbohydrate intake.[13] A healthy diet and exercise are key factors in the management of GDM. Clinical studies have shown that diet control can be effective for controlling blood glucose levels in pregnant woman, and the use of diet control for GDM patients is associated with improved maternal and neonatal outcomes,[1,13,14] which has lead to an increasing interest in dietary interventions aimed at reducing the risk of GDM.

The glycemic index (GI) is used to estimate the in vivo blood glucose response to the intake of a food item, relative to that of a carbohydrate reference.[15] The GI ranks food items on a scale of 0 to 100, with food items with higher GI values contributing to a greater increase in blood glucose. The ability to evaluate the effects of patients' diets on their blood glucose levels based on GI values is crucial for improving the clinical

outcomes of GDM patients undergoing diet control. Although various factors influence the relationship between the GI of food and the postprandial glucose level, the current consensus among international experts is that GI is a valid and reproducible method for estimating glycemic response to carbohydrate intake.[16]

Food items with a GI value < 55 are considered low-glycemic index (LGI) foods.[17,18] Previous investigations have shown that LGI diets can reduce the risk of T2DM among women.[16,19] Previous studies have also shown that LGI diets can reduce the level of glycated hemoglobin in diabetic patients[20] and are effective for controlling the level of blood glucose in pregnant diabetic women.[21] The adjustments for other carbohydrate-related nutritional factors, such as the dietary fiber (DF) content and the types of polysaccharides in foods, have also been shown to have beneficial effects on insulin sensitivity in GDM patients.[22]

The findings of various recent clinical trials investigating the effects of LGI diets on postprandial glucose levels and the risk of T2DM have, however, been inconsistent.[23,24] Similar inconsistencies have been observed in effects of diet control on maternal and neonatal outcomes in GDM patients.[25–28] We conducted a meta-analysis of recent randomized clinical trials to systematically evaluate the therapeutic effects of diet control in GDM patients in an effort to clarify whether LGI diets with and without DF have beneficial effects on maternal and neonatal outcomes.

METHODS

Data Sources and Searches

Our meta-analysis was performed according to the recommendations of the Cochrane Handbook,[29] and this report was prepared according to the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines.[30] Ethics approval dose not required for meta-analysis. We searched for the MEDLINE, EMBASE, EBSCO, Springer, Ovid, and Cochrane Library databases for published reports of randomized controlled trials (RCTs) published in English that evaluated the effects of LGI diets on GDM, glucose intolerance, or hyperglycemia in pregnant women. The following keywords were used for our search: ''Glycemic Index'' [MeSH], ''Diet'' [MeSH], ''Pregnancy'' [MeSH], ''Gestational Diabetes'' [MeSH], ''Gestational Diabetes Mellitus'' [MeSH], and ''randomized controlled trial'' [MeSH]. We used a date range ending in 2015. The References section of retrieved articles was also searched manually to identify other relevant RCTs. Ethics approval was not required for this meta-analysis.

Study Selection

Two reviewers (JW and WH) independently reviewed the full-text versions of all the articles retrieved in the literature search to identify eligible studies. Studies that met the following criteria were included in our analysis: (1) Included patients $\geq$18 years of age only; (2) provided a detailed description of randomization; (3) implemented allocation concealment; (5) blinding of patients and personnel; (6) appropriately handled withdrawals; (7) evaluated the effects of an LGI diet; and (8) reported both maternal and neonatal outcomes. Studies that met the following criteria or did not meet the inclusion criteria were excluded from our analysis: (1) Did not implement randomization; (2) did not describe the dietary interventions clearly; (3) did not describe the delivery outcomes; (4) included patients who were diagnosed with any form of diabetes mellitus before their pregnancy; or (5) included subjects who developed a malignancy, heart failure, or renal failure during the study period.

Conflicts in study selection were resolved by a third reviewer (JG). The maternal endpoints used for our analysis included insulin usage and caesarean section delivery, and the neonatal endpoints included macrosomia, small for gestational age (SGA), and large for gestational age (LGA). Macrosomia was defined as birth weight > 4 kg. The SGA and LGA outcomes were defined as $<$ 10th percentile body weight and $>$ 90th percentile body weight, respectively, based on sex and gestational age. We extracted the following types of data from the studies selected: (1) general Information, such as the title of the RCT article, author names, and location of the study; (2) study features, such as study design, approaches, randomization method, bias prevention; and (3) outcome-related data, which included the follow-up duration, number of withdrawals, maternal and neonatal data, number of subjects in each study group, and the incidence of each outcome.

Data Extraction and Quality Assessment

Two reviewers (JW and WH) used a standardized data extraction form to independently extract the data from the selected studies. The following items comprised the extracted data: (a) publication year; name of first author; number of participants; (b) details of the study design, including the trial duration and descriptions of the blinding, allocation concealment, and randomization methods; (c) maternal characteristics, including age, ethnicity (white or nonwhite), and body mass index; (d) gestational age of neonate at the time of maternal GDM diagnosis; (e) intervention and control diet characteristics, including total caloric intake, macronutrient content, and DF content; and (f) a description of the criteria by which dietary compliance was evaluated.

Statistical Analysis

The data analysis was performed using the Review Manager, version 5.2, software which is provided via The Cochrane Collaboration website (http://tech.cochrane.org/revman). Random- or fixed-effects models were used to calculate the overall RR and 95% CI of the maternal and neonatal outcomes for the meta-analysis. Heterogeneity in the clinical outcomes was evaluated using the I^2 statistic. A fixed-effects model was used to estimate risk when significant heterogeneity was not detected ($I^2 \leq 50\%$), whereas a random-effects model was used when significant heterogeneity was detected ($I^2 > 50\%$). The overall effect size was evaluated using the Z test, and a P-value for $Z < 0.05$ was considered to indicate a statistically significant difference. A subgroup analysis was performed to compare the effects of LGI diets on insulin usage and macrosomia in studies in which DF intake was increased in the intervention group with those of studies in which DF intake was not increased in the intervention group. The results of the risk analysis are presented using forest plots.

RESULTS

Study Characteristics

The literature search retrieved 86 records, of which 53 were deemed ineligible based on title and abstract screening. Twenty-one basic research studies and 5 meta-analyses and reviews were excluded because they were not clinical trials.

TABLE 1. Characteristics of the Randomized Controlled Trials Included in the Meta-Analysis

Source	Control Diet	Added DF in Diet
Moses 2009	Low sugar with high DF	No
Grant 2011	Moderate and high GI diets	Yes
Louie 2011	Moderate GI with high DF	No
Perichart-Perera 2012	Low, moderate, and high GI diets	No
Asemi 2014	Low-moderate GI	Yes

DF = dietary fiber, GI = glycemic index.

Two additional studies were excluded after examining the full-length articles because the content did not fit the aim of our study. Five studies that enrolled a total of 302 participants who were 18 to 45 years of age were included in our analysis (Table 1). The selected studies were published between 2009 and 2014, and were conducted in the United States, Europe, and Australia.[25,31–34] Methods of randomization and allocation concealment were adequately described in all of the selected studies, whereas blinding was performed in only 1 of the studies.[32]

Effects of LGI Diets on Maternal Outcomes

All 5 of the selected studies compared the incidence of insulin usage between the LGI and control groups.[25,31–34] Heterogeneity in insulin usage existed between the various studies ($I^2 = 60\%$). The overall RR of insulin usage was 0.67 (95% CI: 0.44–1.00), indicating that the risk of insulin usage was reduced by the LGI diets (Figure 1). However, the upper boundary of the 95% CI (1.00) indicates that the reduction might have been marginal in at least one of the LGI groups, and the difference in the risk of insulin usage between the LGI and control groups was not statistically significant ($Z = 1.97$, $P = 0.05$). Therefore, whether the LGI diets reduced insulin usage was unclear.

The incidence of cesarean section delivery (CSD) was analyzed in 3 of the selected studies.[31–33] Heterogeneity in CSD existed between the various studies ($I^2 = 52\%$). The overall RR of CSD was 0.78 (95% CI: 0.40–1.51), indicating that the risk of cesarean delivery was reduced by the LGI diets (Figure 2). However, the difference between the risk of CSD between the LGI and control groups was not statistically significant ($Z = 0.73$, $P = 0.46$). Therefore, whether the LGI diets reduced the risk of CSD was unclear.

Effects of DF on the Benefit of LGI Diets for Preventing Insulin Usage

In 2 of the selected studies,[25,31] DF intake in the LGI diet groups was significantly greater than that in the control diet groups. We investigated the role of DF content in the effect of LGI diets on the risk of insulin usage. Significant heterogeneity in insulin usage was observed in these 2 studies ($I^2 = 87\%$), and the overall RR of insulin usage was 0.61 (95% CI: 0.17–2.22), which indicated that the relative risk of insulin usage was reduced by the LGI diets with increased DF content (Figure 3). However, the differences in insulin usage between the LGI and control groups were not statistically significant ($Z = 0.75$, $P = 0.46$).

No significant heterogeneity in insulin usage ($I^2 = 9\%$) was detected among the remaining 3 studies in which the amount of DF in the LGI diet was not significantly different than that in the control diet.[32–34] The overall relative risk of insulin usage in the LGI groups in these 3 studies was significantly lower (RR = 0.69; 95% CI: 0.52–0.92; $Z = 2.50$; $P = 0.01$) than that in the control groups (Figure 4). Therefore, our subgroup analysis showed that LGI diets with DF levels approximately equivalent to that in the control diets reduced the risk of insulin usage in GDM patients, whereas no significant difference in the risk of insulin usage was associated with an increased level of DF in the LGI diets, relative to the level of DF in the control diet.

Effects of LGI Diets on Neonatal Outcomes

All 5 of the selected studies compared the incidence of macrosomia between the LGI and control groups.[25,31–34] Significant heterogeneity in macrosomia was not detected ($I^2 = 0\%$). The overall relative risk of macrosomia in the LGI groups was significantly lower (RR = 0.27; 95% CI: 0.10–0.71; $Z = 2.65$; $P = 0.008$) than that in the control groups (Figure 5).

Three of the selected studies analyzed the incidence of LGA.[25,32,33] Significant heterogeneity in LGA was not detected ($I^2 = 0\%$). The RR of LGA was 1.38 (95% CI: 0.58–3.32), indicating that the relative risk of LGA was increased by the LGI diets (Figure 6). However, the lower boundary of the 95% CI (0.58) indicates that the risk of LGA may have actually been lower in at least one of the LGI groups, and the difference in risk of LGA between the LGI groups and control groups was not statistically significant ($Z = 0.72$, $P = 0.47$). Therefore, whether the LGI diets affected the risk of LGA was unclear.

Four of the studies analyzed the incidence of SGA.[25,32–34] Significant heterogeneity in SGA was not detected ($I^2 = 0\%$). The overall RR of SGA was 1.59 (95% CI: 0.60–4.18), indicating that the LGI diets increased the relative risk of SGA (Figure 7). However, the lower boundary of the 95%

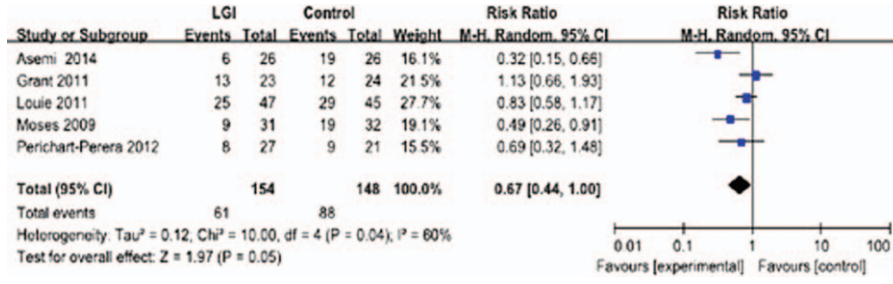

FIGURE 1. Effects of low glycemic index diets on insulin usage.

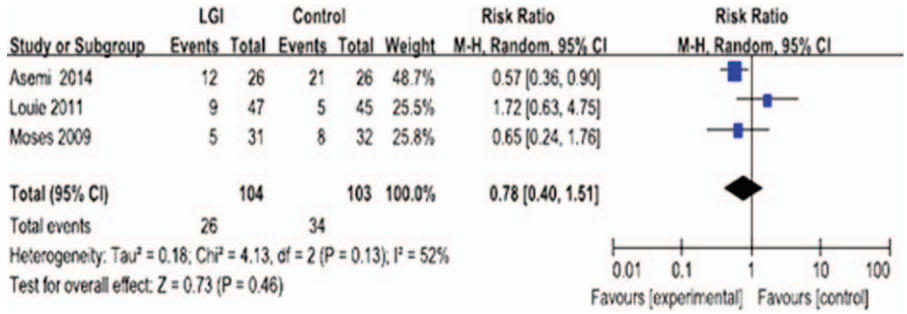

FIGURE 2. Effects of low glycemic index diets on cesarean section deliveries .

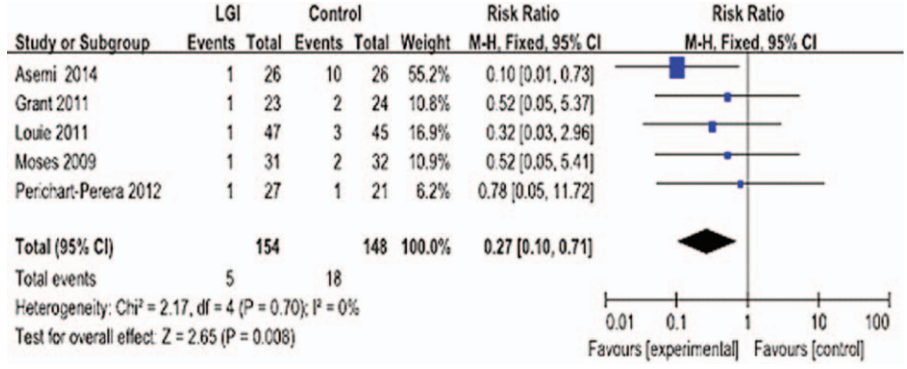

FIGURE 3. Effects of low glycemic index diets with added dietary fiber on insulin usage.

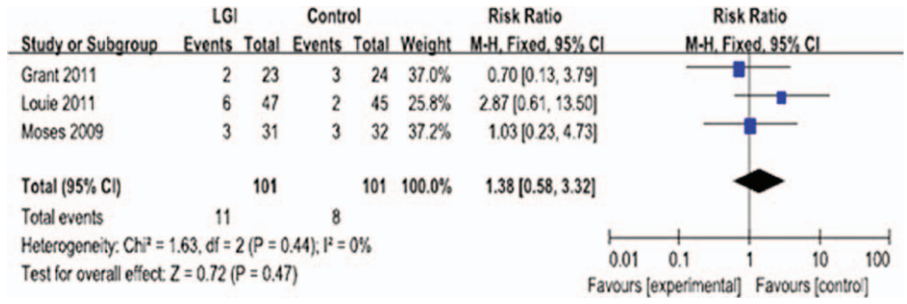

FIGURE 4. Effects of low glycemic index diets without added dietary fiber on insulin usage.

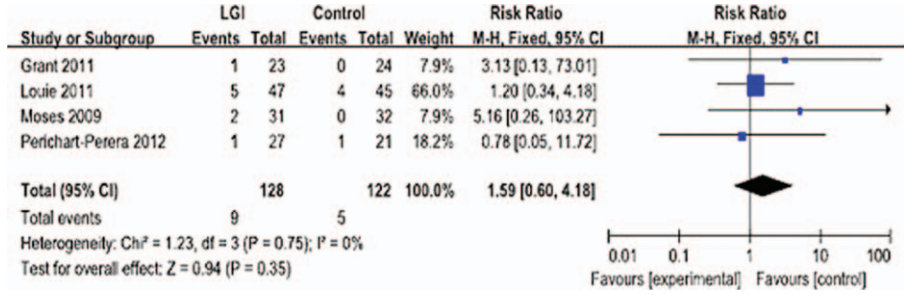

FIGURE 5. Effects of low glycemic index diets on macrosomia.

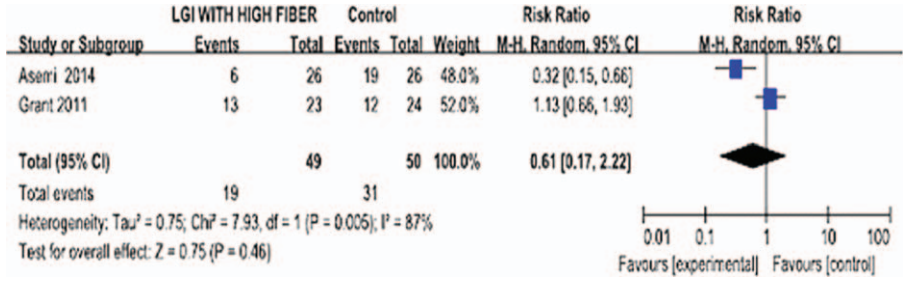

FIGURE 6. Effects of low glycemic index diets on large for gestational age.

CI (0.60) indicates that the risk of LGA may have actually been lower in at least one of the LGI groups, and the difference in the risk of SGA between the LGI groups and the control groups was not statistically significant ($Z = 0.94$, $P = 0.35$). Therefore, whether the LGI diets influenced the risk of SGA was unclear.

Effects of DF on the Benefit of LGI Diets for Preventing Macrosomia

We also investigated the role of DF content in the effect of LGI diets on the risk of macrosomia. No significant heterogeneity in macrosomia was detected in the 2 studies that used LGI diets with increased DF content ($I^2 = 14\%$), and the overall relative risk of macrosomia in the LGI groups was significantly lower (RR = 0.17; 95% CI: 0.04–0.71; $Z = 2.42$, $P = 0.02$) than that in the control groups (Figure 8). In the remaining 3 studies in which the amount of DF in the LGI diet was not greater than that in the control diet, no significant heterogeneity in macrosomia was observed ($I^2 = 0\%$), and the overall relative risk of macrosomia in the LGI groups was significantly lower (RR = 0.47; 95% CI: 0.12–1.83; $Z = 1.09$; $P = 0.27$) than that in the control groups (Figure 9). Therefore, our subgroup analysis showed that LGI diets significantly reduced the risk of macrosomia and that LGI diets with higher DF content reduced the risk of macrosomia further.

DISCUSSION

The results of RCTs investigating the effects of LGI diets on GDM-related outcomes have yielded conflicting findings.[25–28] We performed our meta-analysis to clarify whether LGI diets reduced the risk of adverse maternal and neonatal outcomes, and we also investigated the contribution of DF to the effects of LGI diets in pregnant women. We performed a meta-analysis of 5 RCTs that investigated the effects of LGI diets on GDM-related maternal and neonatal outcomes. We found that LGI diets reduced the risk of macrosomia. In addition, our subgroup analysis showed that LGI diets, with levels of DF approximately equivalent to that in the control diets, reduced the risk of insulin usage, and that LGI diets with increased DF reduced the risk of macrosomia beyond that of an LGI diet alone. Our investigation focused on improvements in clinically tangible endpoints, rather than blood glucose markers, thereby providing information directly relevant to the prevention of adverse GDM-related maternal and neonatal outcomes.

The most recent Cochrane systematic review and meta-analysis of the effects of dietary interventions on GDM-related outcomes did not identify any significant benefit associated with LGI diets.[35] However, Han et al[35] evaluated the effects of low-moderate GI foods on the risk of macrosomia based on a meta-analysis of data from only 2 RCTs involving 88 participants. Our investigation of the effect of LGI diets on the risk of macrosomia analyzed data from 5 RCTs in which 302 participants were enrolled. Although this might suggest that our results are more reliable, future large-scale studies are needed to obtain the level of statistical power required to conclusively assess the effect of LGI diets on GDM-related outcomes. In addition, the comparison of low-moderate GI diets to moderate-high GI diets by Han et al[35] might have diminished the differences in macrosomia rates between the RCTs that they analyzed, thereby contributing to the differences between their results and those of our current meta-analysis.

We also investigated the contribution of dietary fiber to the effects of LGI diets. The indirect blood glucose lowering effects of DF are mainly due to slowing food absorption and promoting gastrointestinal peristalsis and defecation.[22] The findings of previous studies of the role of DF in lowering postprandial glucose levels in pregnant women, like those of the effects of LGI diets, have been inconsistent.[35,36] One previous study did show that an LGI diet with added DF reduced the incidence of

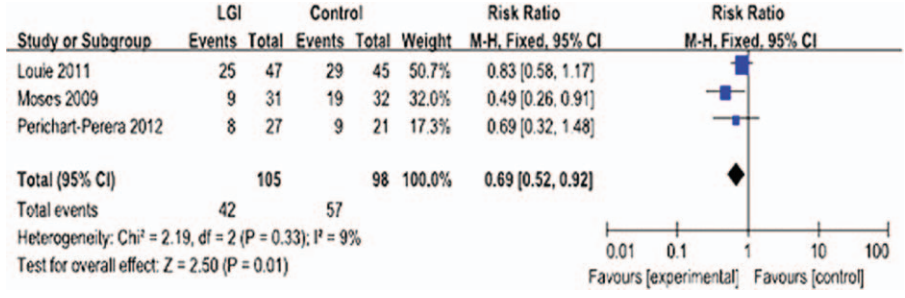

FIGURE 7. Effects of low glycemic index diets on small for gestational age.

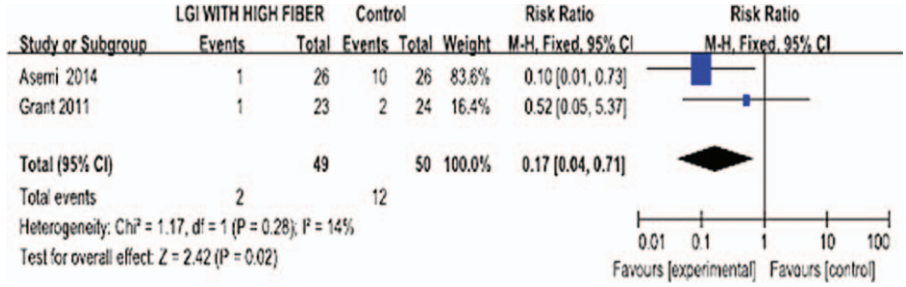

FIGURE 8. Effects of low glycemic index diets with added dietary fiber on macrosomia.

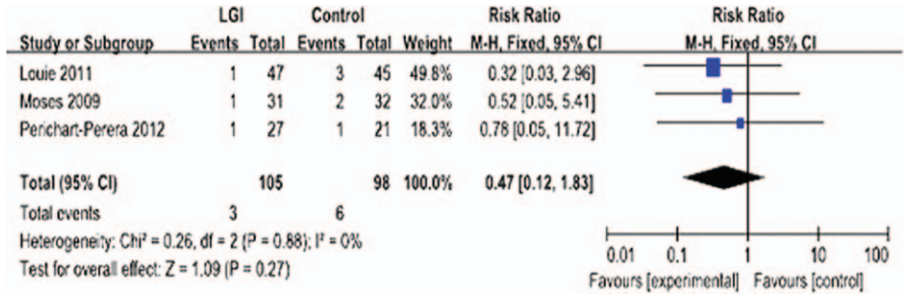

FIGURE 9. Effects of low glycemic index diets without added dietary fiber on macrosomia.

insulin usage in GDM patients, relative to that of GDM patients receiving an LGI diet alone.[36] Our results showed that an LGI diet with increased DF was associated with a reduced risk of insulin usage. However, the differences between the rates of insulin usage in the LGI and control groups analyzed in our study were not statistically significant.

Although our subgroup analysis based on DF intake did not provide conclusive evidence of protection against insulin usage, the relative risk of macrosomia among women receiving an LGI diet with added DF was significantly lower than that associated with an LGI diet alone (RR: 0.17 vs 0.47, respectively). Han et al did not identify blood glucose lowering effects associated with increased DF.[35] However, their comparison of LGI diets to moderate GI diets with high DF content may have diminished the differences in macrosomia rates between their study groups, whereas we investigated the effects of added DF on LGI diets alone. Our findings and those of previous investigations suggest the need for future studies of LGI diets with added DF and suggest that more stringent study designs are required to clearly identify the contribution of DF to postprandial glucose levels in pregnant women.

Our subgroup analysis also showed that, in the studies in which the levels of DF in the intervention and control groups were approximately equivalent, the LGI diets significantly reduced the relative risk of insulin usage (RR = 0.69; 95% CI: 0.52–0.92; $P = 0.01$). These findings are consistent with those of Afaghi et al,[36] who used a study design in which the LGI diet with added DF used food items similar to those used in the LGI diet without added DF. Our findings are also supported by those of a previous meta-analysis, which found that LGI diets significantly reduced the level of glycated hemoglobin in patients with T2DM.[20] By contrast, a previous meta-analysis by Oostdam et al did not find that LGI diets significantly reduced fasting glucose.[37] However, Oostdam et al reported that the quality of the data in the studies which they analyzed was low. These findings of previous studies and those of our current meta-analysis further emphasize the need for standardized protocols for evaluating LGI diets and DF supplements for the treatment of GDM patients.

Our findings are subject to certain limitations. We observed substantial variation in the reporting methods of the patient characteristics and compositions of the LGI and control diets, both of which may have served as confounding factors in our overall analysis of the risks of insulin usage, LGA, and SGA. Standardized methods of evaluating LGI diets and reporting patient characteristics and diet composition are critically needed for reducing interstudy variation in future investigations of the effects of LGI diets on GDM. The relatively small number of studies included in our meta-analysis rendered the use of funnel plots impractical. Therefore, we cannot exclude the influence of publication bias on our findings. Additional RCTs with larger samples and standardized protocols are needed to comprehensively evaluate the effects of LGI diets high in DF in GDM patients.

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