Gestational Diabetes Mellitus and Diet: A Systematic Review and Meta-analysis of Randomized Controlled Trials Examining the Impact of Modified Dietary Interventions on Maternal Glucose Control and Neonatal Birth Weight

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OBJECTIVE

Medical nutrition therapy is a mainstay of gestational diabetes mellitus (GDM) treatment. However, data are limited regarding the optimal diet for achieving euglycemia and improved perinatal outcomes. This study aims to investigate whether modified dietary interventions are associated with improved glycemia and/or improved birth weight outcomes in women with GDM when compared with control dietary interventions.

RESEARCH DESIGN AND METHODS

Data from published randomized controlled trials that reported on dietary components, maternal glycemia, and birth weight were gathered from 12 databases. Data were extracted in duplicate using prespecified forms.

RESULTS

From 2,269 records screened, 18 randomized controlled trials involving 1,151 women were included. Pooled analysis demonstrated that for modified dietary interventions when compared with control subjects, there was a larger decrease in fasting and postprandial glucose (−4.07 mg/dL [95% CI −7.58, −0.57]; *P* = 0.02 and −7.78 mg/dL [95% CI −12.27, −3.29]; *P* = 0.0007, respectively) and a lower need for medication treatment (relative risk 0.65 [95% CI 0.47, 0.88]; *P* = 0.006). For neonatal outcomes, analysis of 16 randomized controlled trials including 841 participants showed that modified dietary interventions were associated with lower infant birth weight (−170.62 g [95% CI −333.64, −7.60]; *P* = 0.04) and less macrosomia (relative risk 0.49 [95% CI 0.27, 0.88]; *P* = 0.02). The quality of evidence for these outcomes was low to very low. Baseline differences between groups in postprandial glucose may have influenced glucose-related outcomes. As well, relatively small numbers of study participants limit between-diet comparison.

CONCLUSIONS

Modified dietary interventions favorably influenced outcomes related to maternal glycemia and birth weight. This indicates that there is room for improvement in usual dietary advice for women with GDM.

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Gestational diabetes mellitus (GDM) is one of the most common medical complications in pregnancy and affects an estimated 14% of pregnancies, or one in every seven births globally (1). Women with GDM and their offspring are at increased risk of both short- and longer-term complications, including, for mothers, later development of type 2 diabetes, and for offspring, increased lifelong risks of developing obesity, type 2 diabetes, and metabolic syndrome (2–6). The adverse intrauterine environment causes epigenetic changes in the fetus that may contribute to metabolic disorders, the so-called vicious cycle of diabetes (7).

The mainstay of GDM treatment is dietary and lifestyle advice, which includes medical nutrition therapy, weight management, and physical activity (8). Women monitor their fasting and postmeal glucose levels and adjust their individual diet and lifestyle to meet their glycemic targets. This pragmatic approach achieves the glycemic targets in approximately two-thirds of women with GDM (8). However, despite the importance of medical nutrition therapy and its widespread recommendation in clinical practice, there are limited data regarding the optimal diet for achieving maternal euglycemia (8–11). It is also unknown whether the dietary interventions for achieving maternal glycemia are also effective for reducing excessive fetal growth and adiposity (12).

Different dietary strategies have been reported including low glycemic index (GI), energy restriction, increase or decrease in carbohydrates, and modifications of fat or protein quality or quantity (12–14). Three recent systematic reviews have been performed examining specific diets and pregnancy outcomes (15–17). Viana et al. (16) and Wei et al. (15) concluded that low-GI diets were associated with a decreased risk of infant macrosomia. However, the most recent systematic review from Cochrane, including 19 trials randomizing 1,398 women, found no clear difference in large for gestational age or other primary neonatal outcomes with the low-GI diet (17). The primary maternal outcomes were hypertension (gestational and/or preeclampsia), delivery by cesarean section, and type 2 diabetes, outcomes for which most trials lacked statistical power, even when dietary subgroups were combined. Remarkably, no systematic reviews examined the impact of modified dietary interventions on the detailed maternal glycemic parameters, including change in glucose-related variables, the outcomes that are most directly influenced by diet.

To address this knowledge gap, we performed a systematic review and meta-analysis of randomized controlled trials to investigate whether modified dietary interventions (defined as a dietary intervention different from the usual one used in the control group) in women with GDM offer improved glycemic control and/or improved neonatal outcomes when compared with standard diets.

RESEARCH DESIGN AND METHODS

In accordance with a published protocol (PROSPERO CRD42016042391), we performed a systematic review and meta-analysis. Reporting is in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines (18). An international panel of experts was formed by the International Life Sciences Institute Europe. This panel determined the review protocol and carried out all aspects of the review.

Data Sources and Search Strategy

The following databases were searched for all available dates using the search terms detailed in Supplementary Table 1: PubMed, MEDLINE, Cochrane Central Register of Controlled Trials (CENTRAL), Embase, Cumulative Index to Nursing and Allied Health Literature (CINAHL), Web of Science Core Collection, Applied Social Sciences Index and Abstracts, ProQuest, ProQuest Dissertations & Theses—A&I and UK & Ireland, National Institute for Health and Care Excellence evidence search, Scopus, UK Clinical Trials Gateway, ISRCTN, and ClinicalTrials.gov. The initial search was performed in July 2016. An updated search of MEDLINE, Embase, CENTRAL, and CINAHL was performed on 3 October 2017 using the same search terms.

A hand-search of relevant reviews and all included articles was conducted to identify studies for potential inclusion. As well, experts on the panel were consulted for the inclusion of additional articles. Reference management was carried out using EndNote.

Study Selection

All titles and abstracts were assessed independently and in duplicate to identify articles requiring full-text review. Published studies fulfilling the following criteria were included: randomized controlled trials, evaluated modified dietary interventions on women with GDM, glucose intolerance or hyperglycemia during pregnancy, reported-on primary maternal and neonatal outcomes, included women aged 18–45 years, had a duration of 2 weeks or more, and were published in English, French, Spanish, Portuguese, Italian, Dutch, German, or Chinese. We excluded studies that included participants with type 1 or type 2 diabetes if data for participants with GDM were not presented independently, if dietary characteristics were not available, if the study was in animals, or if the study did not report outcomes of interest. We did not include studies of nutritional supplements such as vitamin D or probiotics as recent reviews have addressed these topics (19,20).

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See accompanying commentary, p. 1343. See accompanying articles, pp. 1337, 1339, 1362, 1370, 1378, 1385, 1391, and e111.

All citations identified after title and abstract assessment were full-text reviewed in duplicate. Reasons for exclusion at the full-text review stage were recorded. Any disagreements between reviewers were resolved by consensus and with consultation with the expert group when required.

Data Extraction

Data from included studies were extracted in duplicate using prespecified data extraction forms. Extracted data elements included study and participant demographics, study design, diagnostic criteria for GDM, glucose intolerance or hyperglycemia, funding source, description of modified dietary intervention and comparator, and maternal and neonatal outcomes. For studies with missing data, inconsistencies, or other queries, authors were contacted. Record management was carried out using Microsoft Excel and RevMan.

For articles providing information on maternal weight, fasting glucose, postprandial glucose, HbA_{1c}, or HOMA insulin resistance index (HOMA-IR) at baseline and postintervention but not their change, change was calculated as the difference between postintervention and baseline. Standard deviations were imputed using the correlation coefficient observed in articles reporting full information on the variable at baseline and postintervention and its change or a correlation coefficient of 0.5 when this information was not available (21). As studies differed in postprandial glucose at baseline, glycemic control at study entry was not considered to be equivalent in both arms, and thus continuous glucose-related variables at follow-up are reported as change from baseline.

Data Synthesis

The primary outcomes were maternal glycemic outcomes (mean glucose, fasting glucose, postprandial glucose [after breakfast, lunch, and dinner and combined], hemoglobin A_{1c} [HbA_{1c}], assessment of insulin sensitivity by HOMA-IR, and change in these parameters from baseline to assessment; medication treatment [defined as oral diabetes medications or insulin]) and neonatal birth weight outcomes (birth weight, macrosomia, and large for gestational age).

Data were pooled into relative risks (RRs) or mean differences with 95% CI for dichotomous outcomes and continuous outcomes, respectively. Meta-analysis was performed using random-effects models. A prespecified analysis stratified by type of diet and quality assessment was performed to explore potential reasons for interstudy variation. Heterogeneity was assessed using I^2 statistics. Small study effects were examined for using funnel plots. Analyses were conducted using RevMan version 5.3. Pooled estimation of birth weight in the study and control arms, both overall and according to the specific diet intervention, was performed using Stata 14.0.

Quality Assessment

Methodological quality and bias assessment was completed by two reviewers. Risk of bias was assessed using the Cochrane Collaboration tool, which rates seven items as being high, low, or unclear for risk of bias (21). These items included random sequence generation, allocation concealment, blinding of participants and personnel, blinding of outcome assessment, incomplete outcome data, selective outcome reporting, and other potential sources of bias (21). A sensitivity analysis was performed excluding articles with relevant weaknesses in trial design or execution.

The overall quality of the evidence was also assessed using Grading of Recommendations Assessment, Development and Evaluation (GRADE) working group guidelines (21). GRADE was assessed for all primary and secondary outcomes, both maternal and neonatal, but without subgroup analysis per different dietary intervention for each outcome measure.

RESULTS

We screened 2,269 records for potential inclusion, and 126 articles were reviewed in full (Supplementary Fig. 1). Eighteen studies (12–14,22–36) were included in the meta-analysis with a total of 1,151 pregnant women with GDM.

Study Characteristics

The types of modified dietary intervention included low-GI (n = 4), Dietary Approaches to Stop Hypertension (DASH) (n = 3), low-carbohydrate (n = 3), fat-modification (n = 2), soy protein–enrichment (n = 2), energy-restriction (n = 1), high-fiber (n = 1), and ethnic diets (i.e., foods commonly consumed according to participant's ethnicity) (n = 1) and behavioral intervention (n = 1). Details of the study characteristics are included in Table 1. Most trials were single centered and had small sample sizes (range 12–150). Only two trials (one each from Spain and Australia) included over 100 participants, nine had 50–100 participants, and seven studies had fewer than 50 participants. They were performed in North America, Europe, or Australasia and all had a duration of at least 2 weeks. The ethnicity of participants was reported in seven studies (12,13,26,29,31,32,34).

Most studies assessed individual dietary adherence using food diaries (13, 23–36). Although most studies did report an overall difference in dietary composition between the intervention diet and control diet, few studies reported a detailed assessment of dietary adherence. Only five studies used a formal measure of adherence (24,25,29,33,34), and four of them reported data (25,29,33,34). Adherence ranged from 20% to 76% in the control groups and 60% to 80% in the intervention groups.

Participant Characteristics

When baseline characteristic data were pooled, women in the intervention group were older than women in the control group (pooled mean difference 0.60 years [95% CI 0.06, 1.14]) and had higher postprandial glucose (5.47 [0.86, 10.08]), most influenced by the DASH and ethnic diet studies. There was no overall significant difference between the intervention and control groups for BMI, gestational age at enrollment, fasting glucose, HbA_{1c}, or HOMA-IR.

Maternal Glycemic Outcomes for All Modified Dietary Interventions

Pooled risk ratios in 15 studies involving 1,023 women demonstrated a lower need for medication (RR 0.65 [95% CI 0.47, 0.88]; I^2 = 55) (Table 2). Thirteen studies (n = 662 women) reported fasting glucose levels, nine (n = 475) reported combined postprandial glucose measures, and three (n = 175) reported post-breakfast glucose measures. Pooled analysis demonstrated a larger decrease in fasting, combined postprandial, and post-breakfast glucose levels in modified dietary interventions (mean −4.07 mg/dL [95% CI −7.58, −0.57], I^2=86, P=0.02; −7.78 mg/dL [−12.27, −3.29], I^2 = 63, P = 0.0007; and −4.76 mg/dL [−9.13, −0.38], I^2 = 34, P = 0.03, respectively) compared with

Table 1—Characteristics of studies included

Author, year (ref.)	Country	*n*	Estimated sample size	Definition of GDM	Duration of dietary intervention	Gestational age in weeks at enrollment (mean ± SD)	Baseline BMI, kg/m^2 (mean ± SD)	Mean maternal age, years (mean ± SD)	Dietary intervention	Diet composition (mean ± SD)*
Low-GI diet										
Grant, 2011 (26)	Canada	47	50 to detect a 0.6 mmol/L difference in capillary glucose; *n* not achieved	Canadian Diabetes Association (40)	28 weeks until delivery	Control: 29 ± 2.35 Intervention†: 29 ± 3.21	Control: 26 ± 4.69 Intervention: 27 ± 4.58 (prepregnancy)	Control: 34 ± 0.46 Intervention: 34 ± 5.16	Low GI: Women were provided with a list of starch choices specific to either intervention (low GI) or control	Control: GI 58.0 ± 0.5 Intervention: GI 49.0 ± 0.8
Louie, 2011 (29)	Australia	99	120 to detect a 260-g difference in birth weight (stopped early because of smaller than expected SD)	Australasian Diabetes in Pregnancy Society criteria (41)	Randomization until delivery	Control: 29.7 ± 3.5 Intervention: 29 ± 4.0	Control: 24.1 ± 5.7 Intervention: 23.9 ± 4.4 (prepregnancy)	Control: 32.4 ± 4.5 Intervention: 34 ± 4.1	Low GI: Target GI ≤50 but otherwise similar composition to the control diet	Control: energy 1,934 ± 465; carbohydrate 40.3 ± 8.3; protein 22.2 ± 7.5; fat 35.1 ± 16.9; GI 53.0 ± 6.5 Intervention: energy 1,836 ± 403; carbohydrate 38.7 ± 8.3; protein 23.4 ± 5.8; fat 34.9 ± 11.0; GI 47.0 ± 6.5
Ma, 2015 (30)	China	95	Not reported	Chinese Medical Association and American Diabetes Association (42)	24–26 weeks until delivery	Control: 27.9 ± 1.1 Intervention: 27.5 ± 1.1	Control: 21.15 ± 2.75 Intervention: 21.90 ± 3.14 (prepregnancy)	Control: 30.0 ± 3.5 Intervention: 30.1 ± 3.8	Low GI: Women provided with an exchange list for starch choices specific to either intervention (low GI) or control	Control: energy 2,030 ± 215; carbohydrate 49.8 ± 6.8; protein 18.8 ± 2.5; fat 31.8 ± 3.8; GI 53.8 ± 2.5 Intervention: energy 2,006 ± 215; carbohydrate 48.56 ± 7.0; protein 18.9 ± 2.9; fat 32.1 ± 4.1; GI 50.1 ± 2.2

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Table 1—Continued

Author, year (ref.)	Country	*n*	Estimated sample size	Definition of GDM	Duration of dietary intervention	Gestational age in weeks at enrollment (mean ± SD)	Baseline BMI, kg/m^2 (mean ± SD)	Mean maternal age, years (mean ± SD)	Dietary intervention	Diet composition (mean ± SD)*
Moses, 2009 (13)	Australia	63	Not reported	Australasian Diabetes in Pregnancy Society (41)	28–32 weeks until delivery	Control: 29.9 ± 1.11 Intervention: 30.3 ± 1.11	Control: 32.8 ± 7.92 Intervention: 32.0 ± 6.68 (at enrollment)	Control: 31.3 ± 4.52 Intervention: 30.8 ± 3.90	Low GI: Women asked to avoid specific high-GI foods and were provided with a booklet outlining carbohydrate choices	Control: energy 1,656 ± 433; carbohydrate 36.2 ± 8.2; protein 24.0 ± 4.4; fat 34.3 ± 9.9; GI 52.2 ± 6.0 Intervention: energy 1,713 ± 368; carbohydrate 36.7 ± 6.1; protein 23.9 ± 3.9; fat 33.4 ± 6.12; GI 48.0 ± 5.0
DASH diet										
Asemi, 2013 (22)	Iran	34	32 for "key variable serum HDL"	50-g glucose challenge >140 mg/dL → 100 g OGTT; GDM if two or more of fasting >95 mg/dL, 1-h 180 mg/dL, 2-h 155 mg/dL, or 3-h 140 mg/dL	4 weeks	Not reported	Control: 31.4 ± 5.7 Intervention: 29.0 ± 3.2 (at enrollment)	Control: 29.4 ± 6.2 Intervention: 30.7 ± 6.7	DASH diet: diet rich in fruit, vegetables, whole grains, and low-fat dairy; low in saturated fats, cholesterol, refined grains, and sweets	Control: energy 2,392 ± 161; carbohydrate 54.0 ± 6.9; protein 17.6 ± 2.8; fat 29.3 ± 5.6 Intervention: energy 2,400 ± 25; carbohydrate 66.8 ± 2.2; protein 16.8 ± 1.2; fat 17.6 ± 0.9
Asemi, 2014 (23)	Iran	52	42 to detect a 75-g difference in birth weight	As above	4 weeks	Control: 25.9 ± 1.4 Intervention: 25.8 ± 1.4	Control: 31 ± 4.9 Intervention: 29.2 ± 3.5 (at enrollment)	Control: 30.7 ± 6.3 Intervention: 31.9 ± 6.1	DASH diet: as above	Control: energy 2,352 ± 163; carbohydrate 54.2 ± 7.1; protein 18.2 ± 3.4; fat 28.5 ± 5.6 Intervention: energy 2,407 ± 30; carbohydrate 66.4 ± 2.04; protein 17.0 ± 1.3; fat 17.4 ± 1.0

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Table 1—Continued

Author, year (ref.)	Country	*n*	Estimated sample size	Definition of GDM	Duration of dietary intervention	Gestational age in weeks at enrollment (mean ± SD)	Baseline BMI, kg/m^2 (mean ± SD)	Mean maternal age, years (mean ± SD)	Dietary intervention	Diet composition (mean ± SD)*
Yao, 2015 (36)	China	33	42 to detect a 75-g difference in birth weight; not achieved	50-g glucose challenge → 100 g OGTT results with two or more of fasting >95 mg/dL, 1-h ≥180 mg/dL, 2-h ≥155 mg/dL, or 3-h ≥140 mg/dL	4 weeks	Control: 25.7 ± 1.3 Intervention: 26.9 ± 1.4	Control: 30.9 ± 3.6 Intervention: 30.2 ± 4.1 (at enrollment)	Control: 28.3 ± 5.1 Intervention: 30.7 ± 5.6	DASH diet: same as above	Control: energy 2,386 ± 174; carbohydrate 52.3 ± 7.2; protein 18.0 ± 3.3; fat 28.3 ± 5.1 Intervention: energy 2,408 ± 54; carbohydrate 66.7 ± 2.3; protein 16.9 ± 1.2; fat 17.17 ± 1.16
Low-carbohydrate diets										
Cypryk, 2007 (25)	Poland	30	Not reported	World Health Organization criteria	2 weeks	29.2 ± 5.4	Not reported	28.7 ± 3.7	Low (intervention) vs. high (control) carbohydrate (45% vs. 60% of total energy, respectively)	Control‡: carbohydrate 60; protein 25; fat 15 Intervention‡: carbohydrate 45; protein 25; fat 30
Hernandez, 2016 (12)	U.S.	12	Pilot study to estimate SD	Carpenter and Coustan criteria (43)	30–31 weeks until delivery	Control§: 31.7 ± 2.45 Intervention: 31.2 ± 0.98	Control: 34.3 ± 3.92 Intervention: 33.4 ± 3.43 (at enrollment)	Control: 30 ± 2.45 Intervention: 28 ± 4.90	Low carbohydrate (intervention) vs. higher-complex carbohydrate/ lower fat (control)	Control‡: carbohydrate 60; protein 15; fat 25 Intervention‡: carbohydrate 40; protein 15; fat 45
Moreno-Castilla, 2013 (31)	Spain	152	152 to detect a 22% difference in need for insulin	2006 National Diabetes and Pregnancy Clinical Guidelines (44,45)	≤35 weeks until delivery	Control: 30.1 ± 3.5 Intervention: 30.4 ± 3.0	Control: 26.6 ± 5.5 Intervention: 25.4 ± 5.7 (prepregnancy)	Control: 32.1 ± 4.4 Intervention: 30.4 ± 3.0	Low carbohydrate (intervention) vs. control (40% vs. 55% of total diet energy as carbohydrate)	Control‡: energy 1,800 minimum; carbohydrate 55; protein 20; fat 25 Intervention‡: energy 1,800 minimum; carbohydrate 40; protein 20; fat 40

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Table 1—Continued

Author, year (ref.)	Country	*n*	Estimated sample size	Definition of GDM	Duration of dietary intervention	Gestational age in weeks at enrollment (mean ± SD)	Baseline BMI, kg/m^2 (mean ± SD)	Mean maternal age, years (mean ± SD)	Dietary intervention	Diet composition (mean ± SD)*
Soy protein–enrichment diets										
Jamilian, 2015 (27)	Iran	68	56 (minimum clinical difference not reported)	One-step 75 g OGTT, American Diabetes Association (46)	6 weeks	Not reported	Control: 28.4 ± 3.4 Intervention: 28.9 ± 5.0	Control: 29.3 ± 4.2 Intervention: 28.2 ± 4.6	Soy protein diet had the same amount of protein as control diet but the protein portion was made up of 35% animal protein, 35% soy protein, 30% other plant proteins	Control: energy 2,426 ± 191; carbohydrate 54.6 ± 7.1; protein 14.4 ± 1.7; fat 32.1 ± 5.4 Intervention: energy 2,308 ± 194; carbohydrate 54.6 ± 7.3; protein 15.0 ± 2.6; fat 30.3 ± 4.7
Sarathi, 2016 (14)	India	62	Not reported	International Association of Diabetes and Pregnancy Study Groups criteria (47)	From diagnosis until delivery	Control: 25.56 ± 1.69 Intervention: 25.19 ± 1.92	Not reported	Control: 29.17 ± 3.38 Intervention: 29.43 ± 2.98	Soy protein diet: 25% of cereal part of high-fiber complex carbohydrates replaced with soy	Control‡: energy 1,600–2,000; minimum carbohydrate 175 g Intervention‡: energy 1,600–2,000; minimum carbohydrate 175 g
Fat-modification diets										
Lauszus, 2001 (28)	Denmark	27	20 to detect a difference in cholesterol of 0.65 mmol/L	3-h 75 g OGTT with blood samples taken every 30 minutes, GDM if 2 or more glucoses >3 SD above the mean	34 weeks until delivery	Not reported	Control: 32.2 ± 5.61 Intervention: 35.3 ± 8.65 (at enrollment)	Control: 29 ± 3.74 Intervention: 31 ± 3.61	High monounsaturated fatty acids: source was hybrid sunflower oil with high-content oleic acid and snacks of almonds and hazelnuts	Control: energy 1,727; carbohydrate 50.0 ± 3.6; protein 19.0 ± 3.6; fat 30.0 ± 7.2 Intervention: energy 1,982; carbohydrate 46 ± 3.5; protein 16 ± 3.5; fat 37 ± 3.5

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Table 1—Continued

Author, year (ref.)	Country	*n*	Estimated sample size	Definition of GDM	Duration of dietary intervention	Gestational age in weeks at enrollment (mean ± SD)	Baseline BMI, kg/m^2 (mean ± SD)	Mean maternal age, years (mean ± SD)	Dietary intervention	Diet composition (mean ± SD)*
Wang, 2015 (35)	China	84	Not reported	International Association of Diabetes and Pregnancy Study Groups criteria (47)	~27 weeks until delivery	Control: 27.3 ± 1.96 Intervention: 27.4 ± 1.52	Control: 22.2 ± 3.6 Intervention: 21.4 ± 3.0 (prepregnancy)	Control: 29.7 ± 4.64 Intervention: 30.3 ± 4.17	Polyunsaturated fatty acid meals (50–54% carbohydrate, 31–35% fat with 45–40 g sunflower oil)	Control: energy 1,978 ± 107; carbohydrate 55.4 ± 2.0; protein 17.9 ± 1.0; fat 26.7 ± 1.3 Intervention: energy 1,960 ± 90; carbohydrate 47.7 ± 0.7; protein 18.0 ± 0.7; fat 34.3 ± 0.2
Other diets										
Bo, 2014 (24)	Italy	99 in diet study (total *n* = 200)	200 to detect a 10% difference in fasting glucose (based on exercise portion of trial)	75 g OGTT	24–26 weeks until delivery	Not reported	Control: 26.8 ± 4.1 Intervention: 26.9 ± 4.6	Control: 33.9 ± 5.3 Intervention: 35.1 ± 4.4	Behavioral dietary recommendations: individual recommendations for helping dietary choices	Control: energy 2,116 ± 383; carbohydrate 46.9 ± 5.9; protein 15.6 ± 2.6; fat 37.4 ± 4.2 Intervention: energy 2,156 ± 286; carbohydrate 47.8 ± 4.9; protein 15.5 ± 2.4; fat 36.7 ± 3.9
Rae, 2000 (32)	Australia	124	120 to detect a decrease in insulin use from 40% to 15% and a decrease in macrosomia from 25% to 5%	OGTT fasting glucose >5.4 mmol/L and/or 2-h glucose >7.9 mmol/L (48)	<36 weeks until delivery	Control: 28.3 ± 4.6 Intervention: 28.1 ± 5.8	Control: 38.0 ± 0.7 Intervention: 37.9 ± 0.7 (at diagnosis)	Control: 30.6 Intervention: 30.2 (SD not reported)	Moderate energy restriction (1,590–1,776 kcal/day) vs. control (2,010–2,220 kcal/day)	Control: energy 1,630 ± 339; carbohydrate 41.0 ± 4.6; protein 24.0 ± 2.3; fat 34.0 ± 5.3 Intervention: energy 1,566 ± 289; carbohydrate 42.0 ± 5.7; protein 25.0 ± 2.4; fat 31.0 ± 5.7

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Table 1—Continued

Author, year (ref.)	Country	*n*	Estimated sample size	Definition of GDM	Duration of dietary intervention	Gestational age in weeks at enrollment (mean ± SD)	Baseline BMI, kg/m^2 (mean ± SD)	Mean maternal age, years (mean ± SD)	Dietary intervention	Diet composition (mean ± SD)*
Reece, 1995 (33)	U.S.	50	Post hoc calculation	Not reported	24–29 weeks until delivery	Not reported	Not reported	Not reported	Fiber-enriched diet: fiber taken as fiber-rich foods (40 g/day) and a high-fiber drink (40 g/day)	Control‡: carbohydrate 50; fat 30; fiber 20 g/day Intervention‡: carbohydrate 60; fat 20 with 80 g fiber/day
Valentini, 2012 (34)	Italy	20	Not reported (pilot study)	Fourth International Workshop Conference on Gestational Diabetes Mellitus (49)	From diagnosis (screening at 24–28 weeks) until delivery	Control 27.1 ± 5.9 Intervention: 21.3 ± 6.8	Control: 24.1 ± 4.7 Intervention: 25.7 ± 3.6 (prepregnancy)	Control: 30.2 ± 4.7 Intervention: 28.9 ± 3.3	Ethnic meal plan: foods commonly consumed per participant's ethnicity with the same kcal and nutrient composition as the control diet	Control‡: carbohydrate 53; protein 18; fat 28; fiber 26 g/day Intervention‡: carbohydrate 55; protein 17; fat 28; fiber 21 g/day

Unless otherwise stated, the units are kcal/day for energy, % for carbohydrate, protein, and fat. OGTT, oral glucose tolerance test. *Reported actual dietary intake. When not reported, prescribed dietary intake is reported. †Intervention is defined as dietary intervention different from the usual dietary intervention used in the control group. ‡Indicates prescribed diet. §The control and intervention groups were reversed for the purpose of meta-analysis so it could be included in the low-carbohydrate group.

control group. There were no significant differences in change in HbA_{1c} (seven studies), HOMA-IR (four studies), or in post-lunch or -dinner glucose levels (two studies).

Neonatal Birth Weight Outcomes for All Diets

Pooled mean birth weight was 3,266.65 g (95% CI 3,172.15, 3,361.16) in the modified dietary intervention versus 3,449.88 g (3,304.34, 3,595.42) in the control group. Pooled analysis of all 16 modified dietary interventions including 841 participants demonstrated lower birth weight (mean −170.62 g [95% CI −333.64, −7.60], $I^2 = 88$; $P = 0.04$) and less macrosomia (RR 0.49 [95% CI 0.27, 0.88], $I^2 = 11$; $P = 0.02$) compared with conventional dietary advice (Table 2 and Fig. 1). There was no significant difference in the risk of large-for-gestational-age newborns in modified dietary interventions as compared with control diets (RR 0.96 [95% CI 0.63, 1.46], $I^2 = 0$; $P = 0.85$).

Subgroup Meta-analysis by Types of Dietary Interventions

Pooled analysis of low-GI diets showed a larger decrease in fasting (26,29,30), postprandial, and post-breakfast glucose compared with control diets (26,30) (Table 2). However, the pooled analysis of the DASH diet showed significant favorable modifications in several outcomes, including change in fasting (22,36) and postprandial glucose (22), HOMA-IR (35), HbA_{1c} (22), medication need (22,23,36), infant birth weight (23,36), and macrosomia (23,36) (Tables 2 and 3). Last, pooled analysis of the soy protein–enriched diet demonstrated a significant decrease in medication use and birth weight (14,27) (Tables 2 and 3). One soy–protein intervention (n = 68 participants) described significantly lower HOMA-IR (27) (Table 2).

Behavioral (one study) and ethnic-specific modified dietary interventions (one study) were included. The behavioral change dietary intervention reported significant differences in change in postprandial glucose and in HbA_{1c} (Table 2) (24). The ethnic diet study demonstrated a significantly larger decrease in fasting and postprandial glucose (Table 2) (34). Fat-modification, low-carbohydrate, and energy-restriction diets were not associated with a significant difference in our primary outcomes in the stratified analysis.

Table 2—Pooled analyses of primary maternal glycemic and infant birth weight outcomes

Outcome	Diet subgroup	*N* of studies	*N* of women	Effect estimate	I^2 (%)
Maternal glycemic outcomes					
				Mean [95% CI]	
Change in fasting glucose (mg/dL)	All diets	13	662	−4.07 [−7.58, −0.57]	86
	Low GI (26,29,30)	3	195	−5.28 [−6.83, −3.73]	0
	DASH (22,36)	2	67	−11.55 [−14.00, −9.09]	0
	Low carbohydrate (12,25)	2	42	3.81 [−4.29, 11.92]	69
	Fat modification (28,35)	2	109	4.87 [−0.44, 10.18]	0
	Soy protein (14,27)	2	130	−7.47 [−20.28, 5.34]	91
	Behavior (24)	1	99	−1.50 [−5.66, 2.66]	—
	Ethnic (34)	1	20	−25.34 [−37.57, −13.11]	—
Change in postprandial glucose (mg/dL)	All diets	9	475	−7.78 [−12.27, −3.29]	63
	Low GI (26,30)	2	121	−7.08 [−12.07, −2.08]	4
	DASH (22)	1	34	−45.22 [−68.97, −21.47]	—
	Low carbohydrate (25)	1	30	−3.00 [−10.06, 4.06]	—
	Fat modification (28,35)	2	109	−6.43 [−13.08, 0.22]	0
	Soy protein (14)	1	62	−1.05 [−11.03, 8.93]	—
	Behavior (24)	1	99	−6.90 [−11.68, −2.12]	—
	Ethnic (34)	1	20	−16.28 [−22.83, −9.73]	—
Change in post-breakfast glucose (mg/dL)	All	3	175	−4.76 [−9.13, −0.38]	34
	Low GI (30)	1	83	−8.6 [−14.11, −3.09]	—
	Low carbohydrate (25)	1	30	−3.00 [−8.15, 2.15]	—
	Soy protein (14)	1	62	−1.05 [−9.73, 7.63]	—
Change in post-lunch glucose (mg/dL)	All	2	92	4.50 [−1.90, 10.90]	0
	Low carbohydrate (25)	1	30	4.00 [−4.56, 12.56]	—
	Soy protein (14)	1	62	5.14 [−4.51, 14.79]	—
Change in post-dinner glucose (mg/dL)	All	2	92	1.81 [−5.28, 8.90]	13
	Low carbohydrate (25)	1	30	1.00 [−8.14, 10.14]	—
	Soy protein (14)	1	62	3.03 [−8.20, 14.26]	—
Change in HOMA-IR (μIU/mL × mmol/L)	All	4	212	−1.10 [−2.26, 0.07]	90
	DASH (36)	1	33	−1.90 [−2.36, −1.44]	—
	Low carbohydrate (12)	1	12	0.60 [−1.90, 3.10]	—
	Soy protein (27)	1	68	−2.00 [−3.17, −0.83]	—
	Behavior (24)	1	99	−0.30 [−0.71, 0.11]	—
Change in HbA_{1c} (%)	All	7	407	−0.05 [−0.13, 0.02]	84
	Low GI (29,30)	2	167	0.01 [−0.02, 0.03]	0
	DASH (22)	1	34	−0.25 [−0.42, −0.08]	—
	Fat modification (28)	1	25	0.10 [−0.14, 0.34]	—
	Soy protein (14)	1	62	−0.01 [−0.07, 0.05]	—
	Behavior (24)	1	99	−0.19 [−0.26, −0.12]	—
	Ethnic diet (34)	1	20	−0.05 [−0.27, 0.17]	—
				RR [95% CI]	
Medication treatment	All	15	1023	0.65 [0.47, 0.88]	55
	Low GI (13,26,29,30)	4	293	0.80 [0.55, 1.14]	34
	DASH (22,23,36)	3	119	0.29 [0.17, 0.50]	0
	Low carbohydrate (31)	1	150	1.00 [0.75, 1.34]	—
	Energy restriction (32)	1	117	1.05 [0.47, 2.34]	—
	Fat modification (35)	1	84	Not estimable	—
	Soy protein (14,27)	2	130	0.44 [0.21, 0.91]	0
	Behavior (24)	1	99	0.61 [0.15, 2.42]	—
	Ethnic (34)	1	20	2.00 [0.21, 18.69]	—
	Fiber (33)	1	11	Not estimable	—
Infant birth weight outcomes					
				Mean [95% CI]	
Birth weight (g)	All	16	841	−170.62 [−333.64, −7.60]	88
	Low GI (13,26,29,30)	4	276	−54.25 [−178.98, 70.47]	0
	DASH (22,23,36)	3	119	−598.19 [−663.09, −533.30]	0
	Low carbohydrate (12,25)	2	42	57.73 [−164.93, 280.39]	0
	Energy restriction (32)	1	122	194.00 [−42.58, 430.58]	—
	Fat modification (28,35)	2	109	−139.61 [−294.80, 15.58]	0
	Soy protein (14,27)	2	131	−184.67 [−319.35, −49.98]	0

Continued on p. 1356

Table 2—Continued

Outcome	Diet subgroup	*N* of studies	*N* of women	Effect estimate	I^2 (%)
	Ethnic diet (34)	1	20	−370.00 [−928.87, 188.87]	—
	Fiber (33)	1	22	−94.00 [−446.68, 258.68]	—
				RR [95% CI]	
Large for gestational age	All	8	647	0.96 [0.63, 1.46]	0
	Low GI (13,26,29)	3	193	1.33 [0.54, 3.31]	0
	Low carbohydrate (31)	1	149	0.51 [0.13, 1.95]	—
	Energy restriction (32)	1	123	1.17 [0.65, 2.12]	—
	Soy protein (14)	1	63	0.45 [0.04, 4.76]	—
	Behavior (24)	1	99	0.73 [0.25, 2.14]	—
	Ethnic diet (34)	1	20	0.14 [0.01, 2.45]	—
Macrosomia	All	12	834	0.49 [0.27, 0.88]	11
	Low GI (13,26,29,30)	4	276	0.46 [0.15, 1.46]	0
	DASH (23,36)	2	85	0.12 [0.03, 0.51]	0
	Low carbohydrate (25,31)	2	179	0.20 [0.02, 1.69]	—
	Energy restriction (32)	1	122	1.56 [0.61, 3.94]	—
	Fat modification (35)	1	84	0.35 [0.04, 3.23]	—
	Soy protein (27)	1	68	0.60 [0.16, 2.31]	—
	Ethnic diet (34)	1	20	0.20 [0.01, 3.70]	—

Secondary Outcomes

Weight gain from inclusion was lower for low-carbohydrate diets and cesarean birth for DASH diets (Supplementary Table 2). Specific diet interventions did not show significant between-group differences in maternal gestational weight gain throughout pregnancy, preeclampsia/eclampsia, neonatal hypoglycemia as defined by the authors, preterm birth, neonatal intensive care unit admission, or small-for-gestational-age newborns (Supplementary Tables 2 and 3).

Sensitivity Analysis of Primary Outcomes

Sensitivity analysis was performed to explore reasons for heterogeneity and to assess outcomes when studies with methodological concerns were removed. We were unable to include four studies (22,23,34,36), including all the DASH diet studies, where clarification of certain aspects of the results could not be obtained, even after a direct approach to the authors. The authors of the ethnic diet study responded to queries but did not provide the required information regarding gestational age at randomization (34). After these studies are removed, the changes in postprandial glucose (mean −5.90 mg/dL [95% CI −7.93, −3.88], $I^2 = 0$; $P = 0.0001$), post-breakfast glucose levels (−4.76 mg/dL [−9.13, −0.38], $I^2 = 34$; $P = 0.03$), and birth weight (−74.88 g [−144.86, −4.90], $I^2 = 1$; $P = 0.04$) remained significant when all diets were combined (Table 3). Furthermore, the heterogeneity in most primary outcomes decreased after removal of these four studies.

When dietary subgroups were assessed, low-GI diets had significant differences in changes in fasting (mean −5.33 mg/dL [95% CI −6.91, −3.76]) (26,29,30), postprandial (−7.08 mg/dL [−12.07, −2.08]) (26,30), and post-breakfast (−8.6 mg/dL [−14.11, −3.09]) glucose (26,30). The soy protein–enriched diet had differences in change of HOMA-IR (mean −2.00 [95% CI −3.17, −0.83]) (27), required less medication use (RR 0.44 [95% CI 0.21, 0.91]), and had a lower birth weight (mean −184.67 g [95% CI −319.35, −49.98]) (14,27). The behavior modification diet had significant differences in change in postprandial glucose (mean −6.90 mg/dL [95% CI −9.85, −3.95]) and in HbA_{1c} (−0.19% [−0.26, −0.12]) (24) (Table 3).

Assessment of Bias and Quality of the Evidence

None of the included studies were assessed as having a low risk of bias in all seven items of the Cochrane Collaboration tool (Supplementary Fig. 2). Most studies were high risk for blinding of participants and personnel and for other sources of bias (Supplementary Fig. 3). Studies scored high risk for other sources of bias for concerns such as baseline differences and industry funding. Most studies had an unclear risk of bias for selective outcome reporting and very few had registered protocols (Supplementary Fig. 3).

GRADE assessment for the outcomes of interest reveals overall low to very low quality of evidence (Supplementary Table 4). Considerations to downgrade quality of evidence involved the entire spectrum, including limitations in the study design, inconsistency in study results, and indirectness and imprecision in effect estimates.

Evaluation for Small Study Effect

Funnel plots of means and RRs of the primary outcomes for the main analysis are shown in Supplementary Figs. 4 and 5 and for the sensitivity analysis in Supplementary Figs. 6 and 7. Overall, funnel plot asymmetry improves with the sensitivity analysis compared with the main analysis for neonatal birth weight outcomes.

CONCLUSIONS

In this meta-analysis, we pooled results from 18 studies including 1,151 women with a variety of modified dietary interventions. Remarkably, this is the first meta-analysis with a comprehensive analysis on maternal glucose parameters. Despite the heterogeneity between studies, we found a moderate effect of dietary interventions on maternal glycemic outcomes, including changes in fasting, post-breakfast, and postprandial glucose levels and need for medication treatment, and on neonatal birth weight. After removal of four studies with methodological concerns, we saw an attenuation of the treatment effect. Nonetheless, the change in post-breakfast and postprandial glucose levels and lowering of infant birth weight

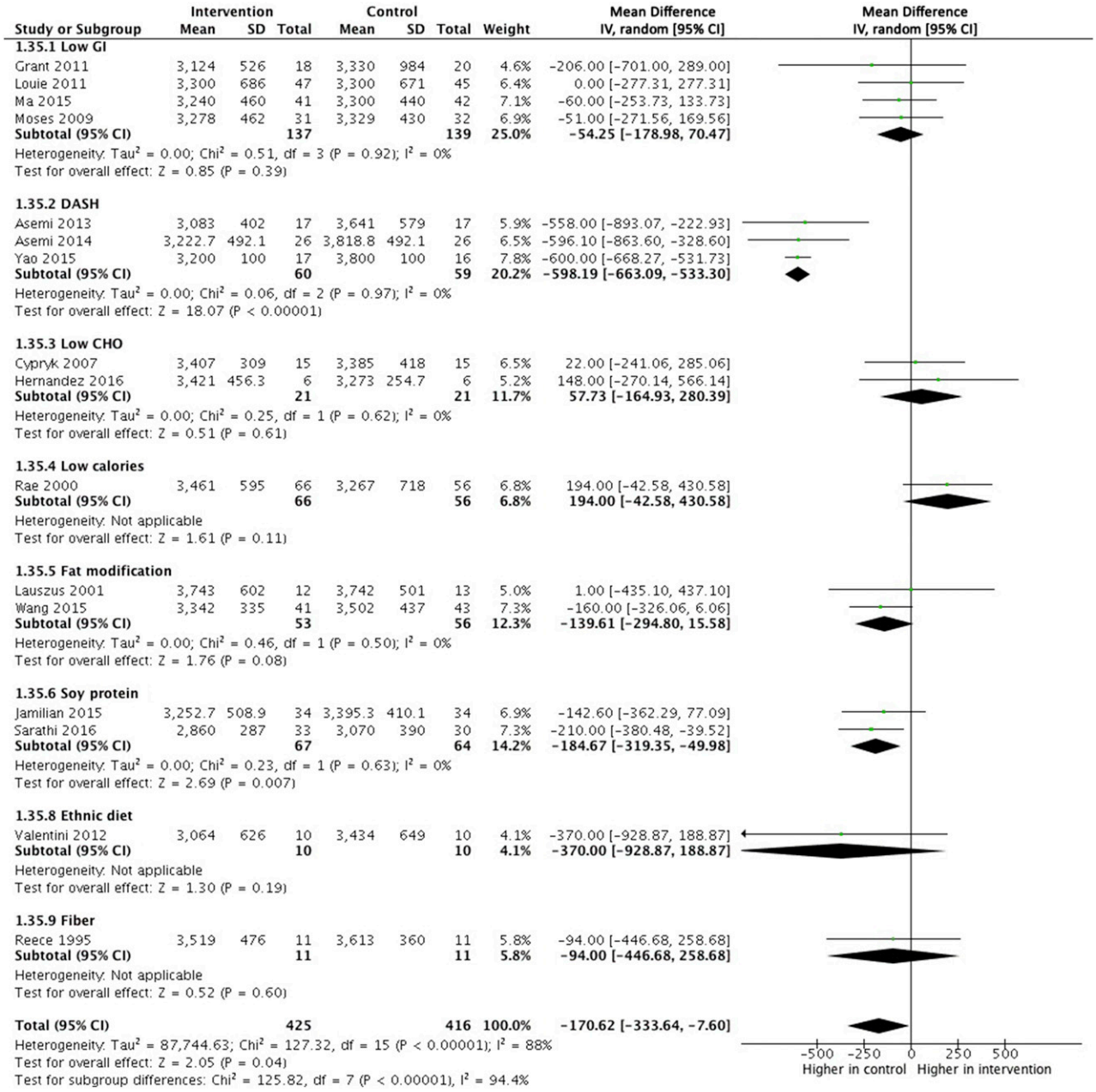

Figure 1—Forest plot of birth weight for modified dietary interventions compared with control diets in women with GDM. Reference citations for studies can be found in Table 1. CHO, carbohydrate; IV, inverse variance.

remained significant. Given the inconsistencies between the main and sensitivity analyses, we consider that conclusions should be drawn from the latter. These data suggest that dietary interventions modified above and beyond usual dietary advice for GDM have the potential to offer better maternal glycemic control and infant birth weight outcomes. However, the quality of evidence was judged as low to very low due to the limitations in the design of included studies, the inconsistency between their results, and the imprecision in their effect estimates.

Previous systematic reviews have focused on the easier-to-quantify outcomes, such as the decision to start additional pharmacotherapy and glucose-related variables at follow-up, but did not address change from baseline (15–17). The most recently published Cochrane systematic review by Han et al. (17) did not find any clear evidence of benefit other than a possible reduction in cesarean section associated with DASH diet. The very high-carbohydrate intake (~400 g/day) and 12 servings of fruit and vegetables in the DASH diet (22,23,36) limit its clinical applicability and generalizability to women from lower socioeconomic, inner city backgrounds in Western countries. The Cochrane review shared one of our primary outcomes, large for gestational age (17). Neither meta-analysis detected a significant difference in risk of large for gestational age because the trials with a larger effect on birth weight (the three DASH studies) did not report on large for gestational age.

Our findings regarding pooled analysis of low-GI dietary interventions are

Table 3—Sensitivity analysis of primary maternal glycemic and infant birth weight outcomes

Outcome	Diet subgroup	*N* of studies	*N* of women	Effect estimate	I^2 (%)
Maternal glycemic outcomes					
				Mean [95% CI]	
Change in fasting glucose (mg/dL)	All diets	10	575	−1.98 [−5.41, 1.45]	74
	Low GI (26,29,30)	3	195	−5.33 [−6.91, −3.76]	0
	DASH	0	0	Not estimable	—
	Low carbohydrate (12,25)	2	42	3.66 [−4.42, 11.73]	57
	Fat modification (28,35)	2	109	4.88 [−1.45, 11.21]	0
	Soy protein (14,27)	2	130	−7.51 [−20.31, 5.30]	90
	Behavior (24)	1	99	−1.50 [−6.47, 3.47]	—
	Ethnic	0	0	Not estimable	—
Change in postprandial glucose (mg/dL)	All diets	7	421	−5.90 [−7.93, −3.88]	0
	Low GI (26,30)	2	121	−7.08 [−12.07, −2.08]	4
	DASH	0	0	Not estimable	—
	Low carbohydrate (25)	1	30	−3.00 [−8.15, 2.15]	—
	Fat modification (28,35)	2	109	−4.85 [−13.32, 3.62]	40
	Soy protein (14)	1	62	−1.05 [−9.73, 7.63]	—
	Behavior (24)	1	99	−6.90 [−9.85, −3.95]	—
	Ethnic	0	0	Not estimable	—
Change in post-breakfast glucose (mg/dL)	All diets	3	175	−4.76 [−9.13, −0.38]	34
	Low GI (30)	1	83	−8.6 [−14.11, −3.09]	—
	Low carbohydrate (25)	1	30	−3.00 [−8.15, 2.15]	—
	Soy protein (14)	1	62	−1.05 [−9.73, 7.63]	—
Change in post-lunch glucose (mg/dL)	All diets	2	92	4.50 [−1.90, 10.90]	0
	Low carbohydrate (25)	1	30	4.00 [−4.56, 12.56]	—
	Soy protein (14)	1	62	5.14 [−4.51, 14.79]	—
Change in post-dinner glucose (mg/dL)	All diets	2	92	1.81 [−5.28, 8.90]	0
	Low carbohydrate (25)	1	30	1.00 [−8.14, 10.14]	—
	Soy protein (14)	1	62	3.03 [−8.20, 14.26]	—
Change in HOMA-IR (μIU/mL × mmol/L)	All diets	3	179	−0.74 [−2.09, 0.61]	75
	DASH	0	0	Not estimable	—
	Low carbohydrate (12)	1	12	0.60 [−1.90, 3.10]	—
	Soy protein (27)	1	68	−2.00 [−3.17, −0.83]	—
	Behavior (24)	1	99	−0.30 [−0.71, 0.11]	—
Change in HbA_{1c} (%)	All diets	5	353	−0.03 [−0.11, 0.05]	87
	Low GI (29,30)	2	167	0.01 [−0.02, 0.03]	0
	DASH	0	0	Not estimable	—
	Fat modification (28)	1	25	0.10 [−0.14, 0.34]	—
	Soy protein (14)	1	62	−0.01 [−0.07, 0.05]	—
	Behavior (24)	1	99	−0.19 [−0.26, −0.12]	—
	Ethnic diet	0	0	Not estimable	—
				RR [95% CI]	
Medication treatment	All diets	11	884	0.82 [0.65, 1.04]	24
	Low GI (13,26,29,30)	4	293	0.80 [0.55, 1.14]	34
	DASH	0	0	Not estimable	—
	Low carbohydrate (31)	1	150	1.00 [0.75, 1.34]	—
	Energy restriction (32)	1	117	1.05 [0.47, 2.34]	—
	Fat modification (35)	1	84	Not estimable	—
	Soy protein (14,27)	2	130	0.44 [0.21, 0.91]	0
	Behavior (24)	1	99	0.61 [0.15, 2.42]	—
	Ethnic	0	0	Not estimable	—
	Fiber (33)	1	11	Not estimable	—
Infant birth weight outcomes					
				Mean [95% CI]	
Birth weight (g)	All diets	12	702	−74.88 [−144.86, −4.90]	1
	Low GI (13,26,29,30)	4	276	−54.25 [−178.98, 70.47]	0
	DASH	0	0	Not estimable	—
	Low carbohydrate (12,25)	2	42	57.73 [−164.93, 280.39]	0
	Energy restriction (32)	1	122	194.00 [−42.58, 430.58]	—
	Fat modification (28,35)	2	109	−139.61 [−294.80, 15.58]	0
	Soy protein (14,27)	2	131	−184.67 [−319.35, −49.98]	0

Continued on p. 1359

Table 3—Continued

Outcome	Diet subgroup	*N* of studies	*N* of women	Effect estimate	I^2 (%)
	Ethnic diet	0	0	Not estimable	—
	Fiber (33)	1	22	−94.00 [−446.68, 258.68]	—
				RR [95% CI]	
Large for gestational age	All diets	7	627	1.00 [0.66, 1.53]	0
	Low GI (13,26,29)	3	193	1.33 [0.54, 3.31]	0
	Low carbohydrate (31)	1	149	0.51 [0.13, 1.95]	—
	Energy restriction (32)	1	123	1.17 [0.65, 2.12]	—
	Soy protein (14)	1	63	0.45 [0.04, 4.76]	—
	Behavior (24)	1	99	0.73 [0.25, 2.14]	—
	Ethnic diet	0	0	Not estimable	—
Macrosomia	All	9	729	0.73 [0.40, 1.31]	0
	Low GI (13,26,29,30)	4	276	0.46 [0.15, 1.46]	0
	DASH	0	0	Not estimable	0
	Low carbohydrate (25,31)	2	179	0.20 [0.02, 1.69]	—
	Energy restriction (32)	1	122	1.56 [0.61, 3.94]	—
	Fat modification (35)	1	84	0.35 [0.04, 3.23]	—
	Soy protein (27)	1	68	0.60 [0.16, 2.31]	—
	Ethnic diet	0	0	Not estimable	—

broadly consistent with those of Viana et al. (16) and Wei et al. (15). Viana et al. (16) noted decreased birth weight and insulin use based on four studies of low-GI diet among 257 women (mean difference −161.9 g [95% CI −246.4, −77.4] and RR 0.767 [95% CI 0.597, 0.986], respectively). Wei et al. (15) also reported decreased risk of macrosomia with a low-GI diet in five studies of 302 women (RR 0.27 [95% CI 0.10, 0.71]). In our analyses of four studies in a comparable number of participants (n = 276), we found the same direction of these effect estimates, without significant between-group differences. This is most likely due to the different studies included. For example, we were unable to obtain effect estimates stratified by type of diabetes in the study by Perichart-Perera et al. (which included women with type 2 diabetes) and therefore did not include this study (37). An important difference between our analyses and that of Wei et al. (15) is that they included DASH diet as a low-GI dietary subtype. We also included a recent study by Ma et al. (30) not included by the previous reviews.

Our sensitivity analyses highlighted concerns regarding some studies included in previous reviews. Notably, after removal of the studies with the most substantial methodological concerns in the sensitivity analysis, differences in the change in fasting plasma glucose were no longer significant. Although differences in the change in postprandial glucose and birth weight persisted, they were attenuated.

This review highlights limitations of the current literature examining dietary interventions in GDM. Most studies are too small to demonstrate significant differences in our primary outcomes. Seven studies had fewer than 50 participants and only two had more than 100 participants (n = 125 and 150). The short duration of many dietary interventions and the late gestational age at which they were started (38) may also have limited their impact on glycemic and birth weight outcomes. Furthermore, we cannot conclude if the improvements in maternal glycemia and infant birth weight are due to reduced energy intake, improved nutrient quality, or specific changes in types of carbohydrate and/or protein.

We have not addressed the indirect modifications of nutrients. For example, reducing intake of dietary carbohydrates to decrease postprandial glucose may be compensated by a higher consumption of fat potentially leading to adverse effects on maternal insulin resistance and fetal body composition. Beneficial or adverse effects of other nutrients such as n-3 long-chain polyunsaturated fatty acid, vitamin D, iron, and selenium cannot be ruled out.

Our study has important strengths and weakness. To our knowledge, ours is the first systematic review of dietary interventions in GDM comprehensively examining the impact of diet on maternal glycemic outcomes assessing the change in fasting and postprandial glucose, HbA_{1c}, and HOMA-IR from baseline. This is especially important given that groups were not well balanced at baseline. Our review also benefits from the rigorous methodology used as well as the scientific, nutritional, and clinical expertise from an international interdisciplinary panel. However, it also has limitations. Baseline differences between groups in postprandial glucose may have influenced glucose-related outcomes. Furthermore, three of the included trials were pilot studies and therefore not designed to find between-group differences (12,26,34). The low number of studies reporting on adherence clearly illustrates that the quality of the evidence is far from ideal. The heterogeneity of the dietary interventions even within a specific type (varied macronutrient ratios, unknown micronutrient intake, and short length of some dietary interventions) and baseline characteristics of women included (such as prepregnancy BMI or ethnicity) may have also affected our pooled results. It should also be noted that the relatively small numbers of study participants limit between-diet comparisons. Last, we were unable to resolve queries regarding potential concerns for sources of bias because of lack of author response to our queries. We have addressed this by excluding these studies in the sensitivity analysis.

Modified dietary interventions favorably influenced outcomes related to maternal glycemia and birth weight. This indicates that there is room for improvement in usual dietary advice for women

with GDM. Although the quality of the evidence in the scientific literature is low, our review highlights the key role of nutrition in the management of GDM and the potential for improvement if better recommendations based on adequately powered high-quality studies were developed. Given the prevalence of GDM, new studies designed to evaluate potential dietary interventions for these women should be based in larger study groups with appropriate statistical power. As most women with GDM are entering pregnancy with a high BMI, evidence-based recommendations regarding both dietary components and total energy intake are particularly important for overweight and obese women. The evaluation of nutrient quality, in addition to their quantity, as well as dietary patterns such as Mediterranean diet (39) would also be relevant. In particular, there is an urgent need for well-designed dietary intervention studies in the low- and middle-income countries where the global health consequences of GDM are greatest.

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The opinions expressed herein and the conclusions of this publication are those of the authors and do not necessarily represent the views of ILSI Europe nor those of its member companies. For further information about ILSI Europe, please email info@ilsieurope.be or call +32 2 771 00 14.

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