

# Nonpharmacological interventions to prevent type 2 diabetes in women diagnosed with gestational diabetes mellitus: a systematic overview

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**Abstract** This systematic overview summarizes the relevant evidence from multiple systematic reviews of the benefits of nonpharmacological interventions for preventing type 2 diabetes mellitus (T2DM) in women diagnosed with gestational diabetes mellitus (GDM). A comprehensive search using the Cochrane Library, CINAHL, EMBASE and MEDLINE via Ovid SP, and PubMed databases was completed on 18 November 2015. Any systematic reviews that evaluated randomized controlled

trials (RCTs) with defined nonpharmacological interventions for preventing T2DM in women diagnosed with GDM were eligible for inclusion. The authors independently performed critical appraisals and quality assessments of the included reviews using the AMSTAR tool, and extracted data were converted to coherent values for tabular summarization. Six eligible reviews of diet and/or exercise, breastfeeding, and reminder interventions were identified; however, the methodologies of the reviews varied greatly, and the majority of the evidence suggested unclear bias. We found inconsistent reporting on the rates at which diet and exercise interventions reduced the risk of T2DM progression, but these interventions were found to be effective at reducing glycemic load. Combined diet, exercise, and breastfeeding interventions proved to be effective at returning women to their postpartum weight. Neither diet alone nor exercise alone proved to be effective at lowering the risk of T2DM. Overall, there was no robust evidence to support the hypothesis that nonpharmacological interventions are effective at lowering the risk of T2DM in women diagnosed with GDM, and there was no consistent evidence showing that these interventions improved the predictor outcomes of T2DM, such as glycemic load or anthropometric changes.

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## Introduction

Gestational diabetes mellitus (GDM) is a condition in which women exhibit glucose intolerance during pregnancy. The prevalence of GDM in a population varies

across ethnicities and has almost doubled over the past few decades [1]. Most pregnant women diagnosed with GDM tend to recede to normal glucose levels after delivery but are at a higher risk of developing type 2 diabetes mellitus (T2DM). A systematic review published in 2009 found that women with GDM were seven times more likely to develop type 2 diabetes compared to women without glycemic complications during pregnancy [2, 3]. The American Diabetes Association Diabetes Guidelines for this high-risk group of women recommend pharmacological interventions as well as nonpharmacological interventions such as periodic blood glucose testing, diet management, and lifestyle changes to improve health outcome and prevent long-term complications that could progress to T2DM [4, 5]. Hence, many randomized controlled trials (RCTs) have been conducted to assess whether nonpharmacological interventions such as lifestyle modifications (e.g., exercise, diet, breastfeeding), including different implementation approaches (e.g., face-to-face counseling, reminder systems, educational programs), can help to reduce the risk of developing T2DM or the risk of developing T2DM predictor outcomes, such as glycemic load or anthropometric changes [6, 7]. The evidence accumulated by these systematic reviews has been scattered and it has not been possible to clearly define any optimal lifestyle interventions because of inconsistent interpretations of the effectiveness of interventions [8–10]. One main concern has been that the reviews have focused on different interventions for the same outcome—some focused on lifestyle interventions as a whole, whereas others focused on the different approaches taken in implementing lifestyle interventions, such as reminder systems or educational lessons. The mixture of information on the effectiveness of interventions hinders researchers and practitioners in promoting best practices, improving the gaps in lifestyle interventions trials, and providing the most suitable alternative care for women who have had GDM to reduce their risk of developing T2DM.

To improve access to the best available evidence and to highlight the gaps present in the multiple systematic reviews that exist, it is necessary to compile and summarize the evidence provided in those systematic reviews. This study was conducted to provide a systematic overview of the existing systematic reviews. It achieves this objective by first summarizing the nonpharmacological interventions defined in the systematic reviews and the intervention approaches used, and then by summarizing the reported data in the systematic reviews on the evidence from RCTs with regard to the effectiveness of the identified interventions at reducing the risk and preventing the development of T2DM in women diagnosed with GDM.

## Methods

The reporting of this overview has been adapted to the PRISMA Statement [11]. This systematic overview was conducted in accordance with the recommendations of *The Cochrane Handbook for Systematic Reviews of Interventions* in reference to overview reviews (chapter 22). The systematic overview protocol was registered with the international prospective register of systematic reviews (PROSPERO registration number: CRD42015029829) on 30 November 2015 [12].

## Search strategy

A complete search was conducted using the Cochrane Library, CINAHL, EMBASE and MEDLINE via Ovid SP, and PubMed databases on 18 November 2015 with no limitations set on date, time, language, document type, and publication status. Keywords (e.g., ‘Pregnancy’ OR ‘Obesity’ OR ‘Weight Gain’ OR ‘Diabetes Mellitus, Gestational’ OR ‘Meta Analysis’ OR ‘Systematic Review’) were collected through experts’ opinions, literature review, controlled vocabulary (e.g., Medical Subject Headings (MeSH), Excerpta Medica Tree (EMTREE), and Cumulative Index to Nursing and Allied Health Literature (CINAHL) Headings), and by reviewing the primary search results. The search strategies were developed with the assistance of a medical information specialist and the strategy is reported in “Search strategy” in the Electronic supplementary material (ESM). The search results were deduplicated using the reference management software (EndNote X5, Thomson Reuters, NY, USA), and then two independent researchers screened and selected relevant reviews. During the process of selecting potential reviews, the reference lists and bibliographies of all the studies included in the review, as well as any other potential reviews found to have been performed, were examined for eligibility.

## Selection criteria

This systematic overview included reviews that looked specifically at women of all ages who had been diagnosed with GDM during pregnancy. Eligible criteria for inclusion were reviews that assessed the effect of nonpharmacological interventions (defined in the reviews) on the prevention of or progression toward T2DM in women with a clinical history of GDM, and reviews that assessed RCTs with relevant study selection criteria, assessed the methodology of the included RCTs, and synthesized the evidence either narratively or in combination with statistical analyses. RCT reviews such as critical reviews, systematic reviews, meta-

analysis reviews, mixed studies reviews, and rapid reviews were eligible for inclusion. Mixed intervention comparisons and meta-analyses conducted on the results of the RCTs were considered for this systematic overview. If the review included observational studies (e.g., cohort studies, cross-sectional or retrospective studies, nonrandomized controlled trials) as well as RCTs, the review was considered for inclusion only when the RCT data were presented separately from the observational studies. Reviews that did not provide RCT data, details on the method used to conduct the review, or clear criteria for participants, specified interventions, and outcomes were excluded from this systematic overview. Reviews that did not evaluate T2DM as one of the outcomes were not eligible for inclusion. Reviews that were published before 2011 were not included since this overview aimed to present the most recent evidence following the adoption of diagnosis and classification guidelines by the International Association of the Diabetes and Pregnancy Study Groups (IADPSG) consensus panel in 2010 [13]. Overviews of reviews, literature reviews that did perform comprehensive searching, scoping reviews, and umbrella reviews were excluded from this systematic overview.

### Data extraction

Two authors (CM and KTN) independently evaluated the review titles and abstracts retrieved from the search of the databases and removed apparent irrelevant reports. The remaining records in the bibliographic database were compared for selection agreement, and when disagreement over the initial selection occurred, it was resolved by discussion between the authors. The full reports of the selected potential reviews were collected and were examined for inclusion eligibility by the two authors independently. To resolve any disagreement in interpretation at this stage of selection, the two authors consulted with other members of the research panel (EO, RM, MK, and NA) who are review and clinical content experts. After finalizing the eligibility of the selected reviews, the two authors independently extracted data from the included reviews, and the extracted data were stored into an electronic data spreadsheet for data reproducibility. The data extraction form was adapted and modified by following the guidance of the *Cochrane Handbook for Systematic Reviews of Interventions* and the *Methodological Expectations of Cochrane Intervention Reviews* (MECIR) 2.3 to ensure consistent data were retrieved from each review article [14, 12]. When a comparison or a RCT was found to be included in more than one review, the comparison or RCT data were collected as individual data and identified as results that overlapped between the reviews. If the review presented insufficient data from the RCT to address the

outcome, that particular published RCT was retrieved for further examination; however, the authors of the published RCT were not contacted for details of missing data.

### Assessment of the methodological quality of the included reviews

The two authors again independently assessed the methodological quality of the included reviews using the AMSTAR (A Measurement Tool to Assess Systematic Reviews) instrument, which is recommended in the *Cochrane Handbook for Systematic Reviews of Interventions* [12, 15]. The AMSTAR instrument consists of 11 assessment criteria as follows:

1. Was an 'a priori' design provided?
2. Was there duplicate study selection and data extraction?
3. Was a comprehensive literature search performed?
4. Was the status of publication (i.e., gray literature) used as an inclusion criterion?
5. Was a list of studies (included and excluded) provided?
6. Were the characteristics of the included studies provided?
7. Was the scientific quality of the included studies assessed and documented?
8. Was the scientific quality of the included studies used appropriately to formulate conclusions?
9. Were the methods used to combine the findings of studies appropriate?
10. Was the likelihood of publication bias assessed?
11. Was the conflict of interest included?

To each question, the response was 'yes' when the review provided clear information that reflected critically on the components identified, and in this case it was given a score of 1. The response was 'no' when the review provided no information on the components identified, in which case it was given a score of 0. The response was 'cannot answer' when the review provided insufficient information on the components identified, and it was given a score of 0 in this case. The response was 'not applicable' when the review provided information that was not relevant to the components identified, in which case it was given a score of 0. An AMSTAR score of 8–11 was rated as high quality, a score of 4–7 as medium quality, and a score of 3 or less as low quality [15]. Disagreements between the authors were resolved by discussion until consensus was reached.

### Qualitative synthesis

Evidence from the included reviews of nonpharmacological interventions for preventing T2DM in women

diagnosed with GDM was reformatted in a summary report for an overview. Overlapping evidence between included reviews was taken into account, and the evidence from the comparisons was summarized by outcome data (e.g., T2DM, glycemic control, or anthropometric change) across the included reviews. When there was no summary of statistical effect estimate available for the outcome, the results were reported in narrative format for each comparison based on the included review's reporting. To standardize the reporting in this systematic overview, summaries of data were converted to risk ratio (RR), based on a two by two contingency table calculation for dichotomous outcomes, or mean difference (MD), based on the average of the mean value and its standard deviation of the outcome measure, which is the absolute difference between the groups compared on same scale [12]. The RR and MD were reported with a 95% confidence interval (CI) estimate for the magnitude and direction of the intervention's effect. Data conversion was only done when sufficient data were available from the included reviews, and re-analysis of data beyond conversions to RR or MD was not undertaken. All statistical conversions were computed using the Review Manager 5.3 software as recommended in the *Cochrane Handbook for Systematic Reviews of Interventions* [16].

## Results

The process of eligible review selection is shown in the PRISMA flow diagram ("PRISMA flow chart" in the ESM). A total of 2284 records were identified from the databases. After removing duplicate records, 1848 title and abstract records were screened to identify relevant reports. Of the 1848 records, 24 potential reviews were selected for full-text examination. After the full-text evaluation, 18 reviews did not meet the eligibility criteria for inclusion and were excluded; a detailed explanation of the exclusion process is provided in "Excluded reviews" in the ESM. The remaining six reviews that complied with the eligibility criteria for inclusion were finalized for qualitative synthesis in this systematic overview. The six eligible reviews [17–22] were different review types: a literature review, a mixed studies review with meta-analyses, a systematic review, a Cochrane review with meta-analyses, and a mixed studies review. The reviews shared similar characteristics in terms of their eligibility criteria, comparators, and outcome measures (Table 1). Only three of the reviews included [18, 19, 21] assessed the quality or risk of bias of the primary studies, and the methods used to assess the quality or the risk of bias differed among the reviews.

All the included reviews specified that women previously diagnosed with GDM were their target population. The included reviews did not provide information on the diagnostic criteria used to determine GDM among the RCTs, except for one review [19], which reported that the included trial did not provide information on the diagnostic criteria used to determine GDM. Three reviews [17, 19, 20] included RCTs exclusively, and the other three reviews [18, 21, 22] included RCTs as well as observational studies, pre-post studies, thematic studies, and quality care studies, which were assessed separately from the RCTs. Two reviews [18, 20] performed meta-analyses. Of the six reviews included, five examined lifestyle interventions (e.g., physical activity/exercise, diet, breastfeeding, and psychosocial support) and one review examined a reminder system. The comparators were either standard care or not receiving the intervention as control. All the included reviews evaluated the incidence of T2DM as the key outcome and assessed other predictive outcomes of T2DM, such as glycemic level and anthropometric changes. Physical activity goal achievement and change in dietary intake were also sought by three reviews, but these outcomes were considered secondary measures and were not included in this systematic overview. A total of 14 RCTs [23–36] were assessed by the included reviews, and one of the reviews [20] did not have overlapping trials with the other five (Table 2). A total of 3,149 women diagnosed with GDM were included in the 14 RCTs, and the trials were completed in Australia, Canada, China, Malaysia, and the US.

## Methodological quality of the included reviews

Two of the included reviews [19, 20] were rated as high quality based on AMSTAR scores of 8 and 10, respectively. Two of the included reviews [18, 21] were rated as medium quality based on AMSTAR scores of 7 and 6, respectively. The remaining two included reviews [17, 22] were rated as low quality, with an AMSTAR score of 2 for both. A table of the quality assessment results for each included review is presented in "AMSTAR assessment" in the ESM.

## Interventions for preventing T2DM in women diagnosed with GDM

All six of the included reviews [17–22] assessed the outcome of conversion to T2DM in women previously diagnosed with GDM who participated in RCTs involving lifestyle interventions or reminder interventions (Table 3a). The reviews provided evidence from six trials: Cheung et al. (2011), Clark et al. (2009), Ratner et al. (2008), Shek

**Table 1** Characteristics of the included reviews

Review ID	Review type	Search period (publication date)	No. of databases searched	Quality assessment used in the review	No. of included studies	Interventions	Intervention approach	Outcomes assessed
Chasan-Taber 2015 [17]	Literature	Not stated	1	Not stated	9 RCTs	Exercise and/or diet Breastfeeding	In-person instruction Web-based Telephone Group sessions Mailings Text messaging	T2DM Biomarkers of insulin resistance Weight change Breastfeeding Physical activity achievements Dietary intake
Gilinsky 2015 [18]	Mixed studies meta-analyses	(1980–April 2014)	5	The Cochrane Collaboration's risk of bias criteria	11 RCTs 2 Pre-post	Exercise and/or diet Breastfeeding	Face-to-face counseling Web-based pedometer Telephone-based education Group exercise Group education Electronic (SMS text/e-mail) Newsletters Breastfeeding counseling 5-Day meal plan Free child care	T2DM Dietary intake Anthropometric change Impaired glucose level Physical activity achievements Sedentary time Breastfeeding Blood pressure Lipids content
Guo 2016 [19]	Systematic	(January 1996–July 2014)	13	Methodological rigor of included studies	12 RCTs	Exercise and/or diet Breastfeeding Psychosocial support	Individual counseling Individual meetings Home visits Education Internet forum sessions Telephone Group sessions Pedometer Postcards Self-help booklet Handouts	T2DM Weight-related measures (BMI) Insulin resistance (2 h OGTT or HOMA-IR) Physical activity achievements Breastfeeding Adherence
Middleton 2014 [20]	Meta-analyses (Cochrane review)	April–1 June 2013	5	The Cochrane Collaboration's risk of bias tool and criteria	1 RCTs	Reminder system	Reminder of any modality (post, email, phone) Direct call or SMS text	T2DM 2 h OGTT Other blood glucose test Health-related quality of life



**Table 1** continued

Review ID	Review type	Search period (publication date)	No. of databases searched	Quality assessment used in the review	No. of included studies	Interventions	Intervention approach	Outcomes assessed
Morton 2014 [21]	Mixed studies	(1946–2014)	5	Risk of bias instrument	6 RCTs 5 Observational	Exercise and/or diet	Advice by telephone Individual counseling Recommendation Monitoring Lessons	T2DM Impaired glucose level Weight BMI Waist-to hip-ratio
Peacock 2014 [22]	Mixed studies	February 2011–November 2013 (1998–2013)	3	CONSORT algorithm scoring method	8 RCTs 5 Observational 14 Thematic 3 Quality care	Exercise and/or diet Positive lifestyle change	Counseling Phone-based education Web-based education Text message reminders Internet forum Physiologist Phone-based motivation	T2DM Physical activity achievements Returning to pre-pregnant weight Weight loss Dietary intake

et al. (2014), Wein et al. (1999), and Yu et al. (2012). Overlapping evidence between reviews is highlighted in Table 3a [23, 24, 31, 33, 35, 36]. The interventions described in the included reviews were separated into five categories: diet and exercise vs control; diet, exercise, and psychosocial support vs health education materials; diet vs control; exercise vs usual care; and reminder system vs no reminder. When sufficient reported data were available, the values were converted to RR values with 95% CI, and these values are presented in the summary of systematic overview table (Table 4).

*Diet and exercise vs control for preventing T2DM*

Five of the included reviews [17–19, 21, 22] presented data from two trials that assessed the outcome of T2DM in a total of 689 women diagnosed with GDM who participated in a diet and exercise intervention (Table 3a). Ratner et al. 2008 reported that there was a significant risk reduction of 53%,  $n = 239$ , when compared to the control group [31]. Shek et al. 2014 reported no significant difference in the rate of conversion to T2DM between groups (RR 0.77, 95% CI 0.51–1.16,  $n = 450$ ) (Table 4) [33].

*Diet, exercise, and psychosocial support vs health education materials for preventing T2DM*

One included review evaluated the effect of a diet, exercise, and psychosocial support intervention on the development of T2DM in women with a history of GDM from one trial [19]. Yu et al. 2012 reported an annual incidence rate of 5.1% in the intervention group compared to 17.9% in the control group who received health education materials, with RR 0.32, 95% CI 0.10–1.10,  $n = 118$  (Table 3a) [36]. The review indicated that there was a significant difference between the two groups, but that this difference did not reach statistical significance, as the higher limit of the CI exceeded 1, which represents the same risk as the control group (Table 4).

*Diet vs control for preventing T2DM*

Four of the included reviews [17–19, 21] evaluated the effect of diet interventions on the development of T2DM based on comparison data acquired from one trial (Table 3a). Wein et al. 1999 reported the annual incidence rate to be 6.1% in the intervention group compared to 7.3% in the usual care group, with an incidence rate ratio of 0.83, 95% CI 0.47–1.48,  $n = 200$  (Table 4) [35]. The relative risk reported by one of the included reviews (Morton 2014) was based on Cox regression analysis (RR 0.63, 95% CI 0.35–1.14,  $n = 200$ ), and no significant difference was indicated between the intervention group and the control group.

**Table 2** Nonpharmacological intervention RCT studies included in the reviews

ID of included trials	( <i>n</i> = total number of women diagnosed with GDM)	Chasan-Taber 2014	Gilinsky 2015	Guo 2016	Middleton 2014	Morton 2014 <sup>a</sup>	Peacock 2014 <sup>a</sup>
Cheung et al. 2011 [23]	( <i>n</i> = 43)	I	I	I			I
Clark et al. 2009 [24]	( <i>n</i> = 256)				I		
Ferrara et al. 2011 [25]	( <i>n</i> = 197)	I	I	I			I
Hu et al. 2012 [26]	( <i>n</i> = 1180)	I	I	I			
Ji et al. 2011 [27]	( <i>n</i> = 130)			I			
Kim et al. 2012 [28]	( <i>n</i> = 49)	I	I	I			I
McIntyre et al. 2012 [29]	( <i>n</i> = 28)	I	I	I			I
Peterson and Jovanovic 1995 [30]	( <i>n</i> = 25)		I				
Ratner et al. 2008 [31]	( <i>n</i> = 350)	I	I	I		I	I
Reinhardt et al. 2012 [32]	( <i>n</i> = 38)	I	I	I			I
Shek et al. 2014 [33]	( <i>n</i> = 450)		I	I		I	
Shyam et al. 2013 [34]	( <i>n</i> = 77)	I	I	I		I	
Wein et al. 1999 [35]	( <i>n</i> = 200)	I	I	I		I	
Yu et al. 2012 [36]	( <i>n</i> = 126)			I			

I: Trial was included in the review. (Observational studies included in the reviews are not included in this table.)

<sup>a</sup> Pharmacological RCTs were not included in this table

### *Exercise vs usual care for preventing T2DM*

Two of the included reviews evaluated the results of an exercise intervention on T2DM outcomes in one trial [18, 19]. Cheung et al. 2011 reported the annual incident rate to be 6.3% in the intervention group compared to 0% in the control group (Table 3a) [23]. The relative risk of T2DM was shown to be greater in the intervention group compared to the usual care group (RR 3, 95% CI 0.13–68.57, *n* = 32), but a statistically significant difference was not observed due to the wide CI with the inclusion of the null value (Table 4).

### *Reminder system vs. no reminder for preventing T2DM*

One included review [20] evaluated a reminder system intervention from one trial, Clark et al. 2009, but the trial did not assess the outcome of T2DM development [24].

### **Interventions for glycemic load in women diagnosed with GDM**

Five of the included reviews [17–21] examined glycemic load as a predictor of T2DM based on reports from 11 trials: [24, 26–31, 33–36]. Data from Clark et al. 2009, Ji 2011, Peterson and Jovanovic 1995, Ratner et al. 2008, and Yu et al. 2012 were not reported in more than one review (Table 3b) [24, 27, 30, 31, 36]. The interventions described in the included reviews were separated into five categories:

diet and exercise vs control; diet, exercise, and psychosocial support vs health education materials; diet vs control; exercise vs usual care; and reminder system vs no reminder. Glycemic outcomes from the trials were assessed using standardized measurements for fasting glucose (FG), fasting insulin (FI), 2-h post-load plasma glucose (2 h OGTT), homeostasis model assessment for insulin resistance (HOMA-IR), and glycated hemoglobin (HbA1c). Hu et al. 2012, Kim et al. 2012, McIntyre et al. 2012, Peterson and Jovanovic 1995, and Yu et al. 2012 compared the difference in change from the baseline to the study end-point between groups, whereas the other trials compared only the overall difference in the end-point measurement between groups [26, 28–30, 36]. The mean and standard deviation data described in the included reviews were converted to MD (95% CI) in the summary of systematic overview table (Table 4).

### *Diet and exercise vs control for glycemic load*

Three of the included reviews evaluated diet and exercise interventions for glycemic outcomes based on results from five trials that included 1416 women diagnosed with GDM (Table 3b) [17–19]. Hu et al. 2012 reported that there was no significant difference in the change from the baseline between compared groups in terms of FG (MD 0.00, 95% CI –0.11 to 0.11, *n* = 404), and 2 h OGTT (MD –0.25, 95% CI –0.55 to 0.05, *n* = 404); however, the decreases in FI (MD –8.60, 95% CI –14.31 to –2.89, *n* = 404) and

**Table 3** Summary of nonpharmacological interventions and outcome data across the included reviews

Review ID	AMSTAR score	Included trials	Intervention vs. comparator	Intervention approach	Data on incidence rate of T2DM intervention vs. comparator	Reported quality assessment of each trial
a) Data on type 2 diabetes mellitus						
Chasan-Taber 2015 [17]	2	Ratner et al. 2008 Wein et al. 1999	Diet and exercise (DPP) vs. control Diet vs. control	Individualized in-person and group session for 2.8 years Telephone and mailing for a median of 51 months	Risk reduction rate of 53% in intervention group, $p = 0.002$ Annual incidence rate: 6.1 vs. 7.3%, (IRR 0.83, 95% CI: 0.47–1.48)	Unclear <sup>a</sup>
Gilinsky 2015 [18]	7	Cheung et al. 2011 Shek et al. 2014	Exercise vs. control Diet and exercise vs. control	Face-to-face counseling session and goal-directed phone calls for 12 months Face-to-face consultation repeated every 3 months for 36 months and monitoring	Not stated No difference in conversion rate to T2DM between groups	Unclear <sup>a</sup>
Guo 2016 [19]	8	✓ Raimer et al. 2008 ✓ Wein et al. 1999 Yu et al. 2012 ✓ Cheung et al. 2011 ✓ Raimer et al. 2008 ✓ Shek et al. 2014 ✓ Wein et al. 1999	Diet, exercise, and psychosocial support vs. health education materials	Four individual/telephonic sessions for 24 months	Annual incidence rate: 5.1 vs. 17.9% ( $d = 0.40$ ) Annual incidence rate: 6.3 vs. 0% ( $d = 0.38$ )	Low <sup>a</sup> Unclear <sup>a</sup> 5 MR score 3 MR score 6 MR score 5 MR score 3 MR score
Middleton 2014 [20]	10	Clark et al. 2009	Reminder system vs. no reminder	Telephonic sessions for 3 months Postal reminder after 3 months from delivery	Not available for evaluation	(–)
Morton 2014 [21]	6	✓ Raimer et al. 2008 ✓ Shek et al. 2014 ✓ Wein et al. 1999	✓ ✓ ✓	✓ ✓ ✓	✓ (RR 0.50, $p = 0.006$ ) comparing most active and least active quartiles ✓ (RR 0.77, 95% CI: 0.51–1.16) ✓ (RR 0.63, 95% CI: 0.35–1.14)	Unclear randomization and concealment; not blinded <sup>b</sup> Not blinded <sup>b</sup> Unclear randomization; not concealed and blinded <sup>b</sup>
Peacock 2014 [22]	2	✓ Raimer et al. 2008	✓ Lifestyle intervention vs. metformin vs. placebo and control	✓	✓ Lifestyle intervention and metformin reduce the risk of T2DM compared to placebo and control	



Table 3 continued

Review ID	AMSTAR score	Included trials	Intervention vs. comparator	Intervention approach	Data on glycemia and metabolites: fasting glucose, fasting insulin, 2 h OGTT (mmol/l or otherwise indicated) or HOMA-IR intervention vs. comparator	Reported quality assessment of each trial
b) Data on glycemic load						
Chasan-Taber 2015 [17]	2	Hu et al. 2012	Diet and exercise (TGDMPP) vs. usual care	Individualized in-person for 1 year	FG: (bb) $-0.09 \pm 0.52$ vs. $-0.09 \pm 0.6$ , $p = 0.97$ ; FI, pmol/l: (bb) $-11.8 \pm 27.4$ vs. $-3.2 \pm 31.2$ , $p = 0.004$	
		Kim et al. 2012	Exercise vs. usual care	Web-based for 13 weeks	FG: (bb) $0.038 \pm 0.62$ vs. $-0.046 \pm 0.57$ , $p = 0.65$ ; 2 h OGTT: (bb) $-0.42 \pm 1.8$ vs. $-0.48 \pm 1.6$ , $p = 0.91$	
		McIntyre et al. 2012	Exercise vs. usual care	Individualized in-person and telephone for 12 weeks	FG, mmol/L: (bb) $0.25 \pm 0.56$ vs. $0.12 \pm 0.42$ ; FI, (bb) mU/mL: $1.49 \pm 4.23$ vs. $0.06 \pm 3.89$	
		Shyam et al. 2013	LGI diet vs. usual care	Telephone and mailings for 6 months	2 h OGTT, mmol/L, median (interquartile range): (bb) $-0.2$ (2.8) vs. $0.8$ (2.0), $p = 0.025$ ; FI, $<2$ mU/L: $61.5$ vs. $52.6\%$ , $p = 0.228$	
Gilinsky 2015 [18]	7	Peterson and Jovanovic 1995	Diet (crossover)	Face-to-face weekly or bi-weekly for 12 weeks	No changes in serum fasting insulin at follow-up	Unclear <sup>a</sup>
		Shek et al. 2014	Diet and exercise vs. control	Face-to-face every 3 months up to 36 months	No difference at last follow-up among intervention group on all glycemic measures	Unclear <sup>a</sup>
		Wein et al. 1999	Diet and exercise vs. usual care	Face-to-face and telephone for 3 months up to 6 years	FG and 1-h blood glucose increased from baseline to last follow-up point in both groups; reduction in 2-h blood glucose in intervention group but increased in the controls, $p < 0.02$	Unclear <sup>a</sup>
		✓ Hu et al. 2012	✓	✓ Personalized, face-to-face and telephone for 12 months	✓ Intervention group have reduction in fasting insulin and HOMA-IR	Unclear <sup>a</sup>
		✓ Kim et al. 2012	✓	✓	✓ All glycemic measures indicated no significant difference, $p \geq 0.10$	Low <sup>a</sup>
		✓ McIntyre et al. 2012	✓	✓	✓	Unclear <sup>a</sup>
		✓ Shyam et al. 2013	✓	✓	✓ Conversion rate from dysglycemia to normoglycemia: 64% in LGI group vs. 38% in the control group, $p = 0.38$	Unclear <sup>a</sup>

Table 3 continued

Review ID	AMSTAR score	Included trials	Intervention vs. comparator	Intervention approach	Data on glycaemia and metabolites: fasting glucose, fasting insulin, 2 h OGTT (mmol/l or otherwise indicated) or HOMA-IR intervention vs. comparator	Reported quality assessment of each trial
Guo 2016 [19]	8	Ji et al. 2011	Diet and exercise vs. oral information	Home visit and telephone for 4 months up to 12 months	FB: 5.06 ± 0.23 vs. 5.39 ± 0.24 ( <i>d</i> = 1.11); 2 h OGTT: 8.75 ± 0.42 vs. 9.45 ± 0.53 ( <i>d</i> = 1.49); HOMA-IR: 1.10 ± 0.41 vs. 1.37 ± 0.21 ( <i>d</i> = 0.79).	7 MR score
		Ratner et al. 2008	Diet and exercise (DPP) vs. usual care	In-person and group sessions for 24 weeks	2 h OGTT: significant difference between group	6 MR score
		Yu et al. 2012	Diet, exercise, and psychosocial support versus health education materials	In-person and telephone for 24 months	HOMA-IR decrease: 0.66 ± 0.05 vs. 0.31 ± 0.03 ( <i>d</i> = -0.48)	5 MR score
		✓ Hu et al. 2012	✓	✓	✓	6 MR score
		✓ Kim et al. 2012	✓	✓	✓ 2 h OGTT: (bb) -0.4 ± 1.8 vs. -0.5 ± 1.6 ( <i>d</i> = -0.06)	5 MR score
		✓ McIntyre et al. 2012	✓	✓	✓ HOMA-IR: (bb) 0.43 ± 1.28 vs. -0.08 ± 1.02 ( <i>d</i> = -0.44)	4 MR score
		✓ Shek et al. 2014	✓	✓	✓	5 MR score
		✓ Shyam et al. 2013	✓	✓	✓ FG mmol/L, median (interquartile range): (bb) -0.2 (0.6) vs. 0.1 (0.6) ( <i>d</i> = 0.50)	6 MR score
		✓ Wein et al. 1999	✓	✓	✓	3 MR score
Middleton 2014 [20]	10	Clark et al. 2009	Reminder system	Postal reminder after 3 months from delivery	Not available for evaluation	(-)
Morton 2014 [21]	6	✓ Shyam et al. 2013	✓	✓	✓ Change in 2 h OGTT from baseline ( <i>p</i> = 0.025)	Not blinded and outcomes were not assessed adequately; unclear selective outcome reporting <sup>b</sup>

Table 3 continued

Review ID	AMSTAR score	Included trials	Intervention vs. comparator	Intervention approach	Data on measurement of weight (kg) change, BMI ( $\text{kg}/\text{m}^2$ ) change or otherwise indicated Intervention vs. comparator	Reported quality assessment of each trial
c) Data on anthropometric changes						
Chasan-Taber 2015 [17]	2	Cheung et al. 2011 Ferrara et al. 2011 Hu et al. 2012 Kim et al. 2012 McIntyre et al. 2012 Ratner et al. 2008 Reinhardt et al. 2012 Shyam et al. 2013	Exercise vs. control Diet, exercise, and breastfeeding (DEBI) vs. usual care Diet and exercise (TGMPP) vs. usual care Exercise vs. usual care Exercise vs. usual care Diet and exercise (DPP) vs. control Diet and exercise vs. usual care LGI diet vs. usual care	Individualized in-person, telephone, and mailing for 12 months Individualized in-person and telephone for 12 months Individualized in-person for 1 year Web-based for 13 weeks Individualized in-person and telephone for 12 weeks Individualized in-person and group session for 2.8 years Telephone and mailings for 6 months In-person, text messaging, and e-mails for 6 months	BMI, median (interquartile range): 28 (23.9–34.3) vs. 25.5 (22.5–28.7), $p = 0.14$ Postpartum weight goal achievement: 37.5 vs. 21.4%, $p = 0.07$ Weight change: (bb) $-1.4 \pm 3.44$ vs. $-0.21 \pm 3.52$ , $p = 0.001$ ; BMI change: (bb) $-0.50 \pm 1.41$ vs. $-0.09 \pm 1.37$ , $p = 0.004$ Weight change: (bb) $-0.14$ vs. $-1.5$ , $p = 0.13$ Weight change: (bb) $0.97 \pm 3.7$ vs. $0.22 \pm 4.2$ Weight loss: $5.13 \pm 0.43$ at 6 months and $1.6 \pm 0.80$ at 3 years in both intervention and control GDM groups combined vs. $4.03 \pm 0.40$ at 3 years in the non-GDM groups Weight difference in change: regression coefficient $-4.0$ , 95% CI $-7.6$ to $-0.5$ ; BMI difference in change: regression coefficient $-1.5$ , 95% CI $-2.8$ to $-0.1$ Goal achievement: 33% versus 8%, $p = 0.01$	Unclear <sup>a</sup> Unclear <sup>a</sup> Unclear <sup>a</sup> Unclear <sup>a</sup> Unclear <sup>a</sup> Unclear <sup>a</sup> Unclear <sup>a</sup> Unclear <sup>a</sup>
Gilinsky 2015 [18]	7	Peterson and Jovanovic 1995 Wein et al. 1999 Shek et al. 2014 ✓ Cheung et al. 2011	Diet (crossover) Diet and exercise vs. usual care Diet and exercise vs. control ✓	Face-to-face weekly or bi-weekly, for 12 weeks Face-to-face and telephone for 3 months up to 6 years Face-to-face every 3 months up to 36 months ✓	Weight loss (bb) at 6 weeks occurred in both groups, $p \leq 0.03$ , but not significant at 12 weeks BMI: (bb) increased in both groups BMI and percentage of body fat were significantly lower in intervention group in some visits but not consistently throughout ✓	Unclear <sup>a</sup> Unclear <sup>a</sup> Unclear <sup>a</sup> Unclear <sup>a</sup>

Table 3 continued

Review ID	AMSTAR score	Included trials	Intervention vs. comparator	Intervention approach	Data on measurement of weight (kg) change, BMI (kg/m <sup>2</sup> ) change or otherwise indicated Intervention vs. comparator	Reported quality assessment of each trial
		✓ Ferrara et al. 2011	✓	✓	✓	Low <sup>a</sup>
		✓ Hu et al. 2012	✓	✓	✓ Body fat change, %: $-0.79 \pm 2.60$ vs. $0.06 \pm 2.30$ , $p = 0.001$ ; WC, cm: $-1.67 \pm 5.45$ vs. $-0.15 \pm 5.07$ , $p = 0.004$ ; HC, cm: $-2.15 \pm 4.83$ vs. $-1.71 \pm 4.06$ , $p = 0.32$	Unclear <sup>a</sup>
		✓ Kim et al. 2012	✓	✓	✓	Low <sup>a</sup>
		✓ McIntyre et al. 2012	✓	✓	✓ No change in weight and anthropometric measures in either group at follow-up	Unclear <sup>a</sup>
		✓ Ratner et al. 2008	✓	✓	✓	Low <sup>a</sup>
		✓ Reinhardt et al. 2012	✓	✓	✓ Weight: $74.1 \pm 16.3$ vs. $79.2 \pm 13.5$ , regression coefficient $-1.5$ , 95% CI $-2.8$ to $-0.1$ ; BMI: $28 \pm 5.7$ vs. $29.6 \pm 5.2$ , regression coefficient $-4.0$ , $-7.6$ to $-0.5$ ; WC, cm: $90.5 \pm 12.0$ vs. $97.8 \pm 10.8$ , regression coefficient $-5.1$ 95% CI $-10.4$ to $0.3$	High <sup>a</sup>
		✓ Shyam et al. 2013	✓	✓	✓ WC, cm: $80.5 \pm 9.1$ vs. $81.5 \pm 10.1$ ; WHR: $0.79 \pm 0.05$ vs. $0.79 \pm 0.05$	Unclear <sup>a</sup>
Guo 2016 [19]	8	✓ Ji et al. 2011	Diet and exercise vs. oral information	Home visit and telephone for 4 months up to 12 months	Weight: $54.94 \pm 6.34$ vs. $55.53 \pm 7.58$ , ( $d = 0.09$ ); BMI: $22.14 \pm 0.62$ vs. $23.87 \pm 8.62$ , ( $d = 0.22$ ).	7 MR score
		✓ Yu et al. 2012	Diet, exercise, and psychosocial support vs. health education materials	In-person and telephone for 24 months	BMI decrease: (bb) $3.1 \pm 0.3$ vs. $1.0 \pm 0.1$ , ( $d = -0.67$ )	5 MR score
		✓ Cheung et al. 2011	✓	✓	✓	3 MR score
		✓ Ferrara et al. 2011	✓	✓	✓	5 MR score
		✓ Hu et al. 2012	✓	✓	✓ BMI change, kg/m <sup>2</sup> : (bb) $-0.50 \pm 1.41$ vs. $-0.09 \pm 1.37$ ( $-0.30$ )	6 MR score
		✓ Kim et al. 2012	✓	✓	✓ Weight change: (bb) $-1.5 \pm 3.4$ vs. $-0.1 \pm 2.2$ (0.48); BMI change, kg/m <sup>2</sup> : (bb) $-0.5 \pm 1.3$ vs. $-0.1 \pm 0.8$ ( $-0.38$ )	5 MR score
		✓ McIntyre et al. 2012	✓	✓	✓	4 MR score

Table 3 continued

Review ID	AMSTAR score	Included trials	Intervention vs. comparator	Intervention approach	Data on measurement of weight (kg) change, BMI (kg/m <sup>2</sup> ) change or otherwise indicated Intervention vs. comparator	Reported quality assessment of each trial
		✓ Rainer et al. 2008	✓	✓	✓	6 MR score
		✓ Reinhardt et al. 2012	✓	✓	✓	5 MR score
		✓ Shek et al. 2014	✓	✓	✓	5 MR score
		✓ Shyam et al. 2013	✓	✓	✓ Weight: 64.0 ± 11.7 vs. 64.5 ± 13.0 ( <i>d</i> = 0.11); BMI 25.8 ± 4.7 vs. 26.3 ± 4.8 ( <i>d</i> = -0.013)	6 MR score
		✓ Wein et al. 1999	✓	✓	✓	3 MR score
Morton 2014 [21]	6	✓ Shyam et al. 2013	✓	✓	✓	Not blinded and outcomes not assessed adequately; unclear selective outcome reporting <sup>b</sup>
Peacock 2014 [22]	2	✓ Ferrara et al. 2011	✓	✓	✓	
		✓ Kim et al. 2012	✓	✓	✓	
		✓ Reinhardt et al. 2012	✓	✓	✓	

*IRR* incidence rate ratio, *RR* risk ratio, *CI* confidence interval, *d* Cohen's *d*, *p* *p* value, *DPP* Diabetes Prevention Project, *MR score* methodological rigor score for each study, *TGDMPP* Tianjin Gestational Diabetes Mellitus Prevention Program, *M* ± *SD* mean and standard deviation, *FG* fasting glucose, *FI* fasting insulin, *2 h OGTT*: 2-h post-load plasma glucose, *LGI* low glycemic index, *HOMA-IR* homeostasis model assessment for insulin resistance, (*bb*) change between baseline and the end of study, (-) cannot estimate, *DEBI* diet, exercise, and breastfeeding intervention, *95% CI* 95% confidence interval, *BMI* body mass index, *WC* waist circumference, *HC* hip circumference, *WHR* waist-to-hip ratio

✓ Same information reported or with additional information provided by the authors in the review

<sup>a</sup> Overall risk of bias judgment for each study

<sup>b</sup> Risk of bias judgment within the study



**Table 4** Summary of the systematic overview table

Nonpharmacological interventions compared to usual care/alternative method of care for preventing T2DM in women diagnosed with GDM

*Patient or population:* women diagnosed with gestational diabetes mellitus (GDM)

*Setting:* location at which the RCT was conducted

*Intervention:* any nonpharmacological interventions

*Comparison:* usual care/alternative method of care

*Outcomes:* type 2 diabetes mellitus (T2DM), glycemic load, anthropometric change

*Number of included reviews:* 6

Outcomes (Intervention versus comparator)	Anticipated absolute effects* (95% CI)		Relative effect (95% CI)	No. of women diagnosed with GDM (Studies)	Comments
	Risk with usual care/ alternative method of care (Narrative report by individual trial)	Risk with nonpharmacological intervention			
Type 2 diabetes mellitus (Diet and exercise vs. control)	Study population 191 per 1000		RR 0.77 (0.51–1.16)	450 (1 RCT)	Ratner et al. 2008 was a four-factorial RCT where 111 of 350 GDM women were allocated to the metformin therapy group, 117 were in the intervention group, and 122 were in the control group
	147 per 1000 (97–222)  (Ratner et al. 2008)				
(Diet, exercise, and psychosocial support vs. health education materials) (Diet vs. control)	Study population 172 per 1000		RR 0.32 (0.10–1.10)	118 (1 RCT)	
	55 per 1000 (17–189)				
(Exercise vs. usual care)	Study population 60 per 1000		RR 0.63 (0.35–1.14)	200 (1 RCT)	
	38 per 1000 (21–68)				
(Reminder system vs. no reminder)	Study population 0 per 1000		RR 3.00 (0.13–68.57)	32 (1 RCT)	
	0 per 1000 (0–0)				
Glycemic load (Diet and exercise vs. control)	Study population See comments (Hu et al. 2012)		Not estimable	256 (1 RCT)	No data were contributed from the trial reported by Clark et al. 2009
	(Hu et al. 2012)				
			FG: (bb) MD 0.00 (–0.11 to 0.11)	404 (1 RCT)	Hu et al. 2012 had 404 of 1180 women with GDM who completed the trial study. Ratner et al. 2008 was a four-factorial RCT where 111 of 350 GDM women were allocated to the metformin therapy group
			FI: (bb) MD –8.60 (–14.31 to –2.89)		
			2 h OGTT: (bb) MD –0.25 (–0.55 to 0.05)	130 (1 RCT)	
			HOMA-IR: (bb) MD –0.25 (–0.45 to –0.05)		
	(Ji et al. 2011)		FG: MD –0.33 (–0.41 to –0.25)	130 (1 RCT)	
			2 h OGTT: MD –0.70 (–0.87 to –0.53)		
			HOMA-IR: MD –0.27 (–0.38 to –0.16)	239 (1 RCT)	
			HbA1c: MD –2.08 (CI –2.13 to –2.03)		
	(Ratner et al. 2008)		2 h OGTT: significant difference between groups	239 (1 RCT)	
			No difference at last follow-up among intervention groups based on all glycemic measures		
	(Shek et al. 2014)		FG: MD –0.40 (–0.71 to –0.09)	193 (1 RCT)	
			2 h OGTT: MD –0.10 (–0.73 to 0.53)		
	(Wein et al. 1999)			193 (1 RCT)	

**Table 4** continued

Nonpharmacological interventions compared to usual care/alternative method of care for preventing T2DM in women diagnosed with GDM

*Patient or population:* women diagnosed with gestational diabetes mellitus (GDM)*Setting:* location at which the RCT was conducted*Intervention:* any nonpharmacological interventions*Comparison:* usual care/alternative method of care*Outcomes:* type 2 diabetes mellitus (T2DM), glycemic load, anthropometric change*Number of included reviews:* 6

Outcomes (Intervention versus comparator)	Anticipated absolute effects* (95% CI)		Relative effect (95% CI)	No. of women diagnosed with GDM (Studies)	Comments
	Risk with usual care/alternative method of care (Narrative report by individual trial)	Risk with nonpharmacological intervention			
(Diet, exercise and psychosocial support vs. health education materials)	(Yu et al. 2012)		HOMA-IR reduction: (bb) MD 0.35 (0.33 to 0.37)	118 (1 RCT)	
(Diet vs. control)	(Peterson and Jovanovic 1995)		No changes in serum fasting insulin at follow-up	25 (1 RCT)	Peterson and Jovanovic 1995 was a crossover RCT of 25 obese women, of whom 13 had previous GDM
	(Shyam et al. 2013)		FG: MD 0.10 (−0.31 to 0.51); 2 h OGTT: MD 0.00 (−1.02 to 1.02); FI < 2 mU/L: 61.5% vs. 52.6%, <i>p</i> = 0.228	77 (1 RCT)	
(Exercise vs. usual care)	(Kim et al. 2012)		FG: (bb) MD 0.08 (−0.28 to 0.45); Log FI: (bb) MD −0.19 (−0.43 to 0.05); 2 h OGTT: (bb) MD 0.06 (−0.98 to 1.10)	42 (1 RCT)	
	(McIntyre et al. 2012)		FG: (bb) MD 0.13 (−0.25 to 0.51); FI: (bb) MD 1.43 (−1.76 to 4.62); HOMA-IR: (bb) MD 0.51 (−0.39 to 1.41)	25 (1 RCT)	
(Reminder system vs. no reminder)	See comments		Not estimated	256 (1 RCT)	No data were contributed from the trial reported by Clark et al. 2009
Anthropometric changes	(Hu et al. 2012)		Weight: (bb) MD −1.19 (−1.87 to −0.51); BMI: (bb) MD −0.41 (−0.68 to −0.14); Body fat: (bb) MD −0.85 (−1.33 to −0.37); WC: (bb) MD −1.52 (−2.55 to −0.49); HC: (bb) MD −0.44 (−1.31 to 0.43)	404 (1 RCT)	Hu et al. 2012 had 404 of 1180 women with GDM who completed the trial study. Ratner et al. 2008 was a four-factorial RCT where 111 of 350 GDM women were allocated to the metformin therapy group
(Diet and exercise vs. control)	(Ji et al. 2011)		Weight: MD −0.59 (−3.00 to 1.82); BMI: MD −1.73 (−3.88 to 0.42)	130 (1 RCT)	
	(Ratner et al. 2008)		Weight loss at the end point of the study in both lifestyle intervention and control groups combined	239 (1 RCT)	
	(Reinhardt et al. 2012)		Weight: MD −5.10 (−15.67 to 5.47); BMI: MD −1.06 (−5.45 to 2.25); WC: MD −7.30 (−15.35 to 0.75)	31 (1 RCT)	
	(Shek et al. 2014)		BMI and percentage of body fat were significantly lower in the intervention group at some visits but not consistently throughout	450 (1 RCT)	

**Table 4** continued

Nonpharmacological interventions compared to usual care/alternative method of care for preventing T2DM in women diagnosed with GDM

*Patient or population:* women diagnosed with gestational diabetes mellitus (GDM)

*Setting:* location at which the RCT was conducted

*Intervention:* any nonpharmacological interventions

*Comparison:* usual care/alternative method of care

*Outcomes:* type 2 diabetes mellitus (T2DM), glycemic load, anthropometric change

*Number of included reviews:* 6

Outcomes (Intervention versus comparator)	Anticipated absolute effects* (95% CI)		Relative effect (95% CI)	No. of women diagnosed with GDM (Studies)	Comments
	Risk with usual care/alternative method of care (Narrative report by individual trial)	Risk with nonpharmacological intervention			
			Data from narrative report by individual trial (intervention group vs. comparison group) Mean difference (95% CI), or otherwise indicated		
	(Wein et al. 1999)		BMI: MD -0.20 (-1.88 to 1.48)	193 (1 RCT)	
(Diet, exercise, and psychosocial support vs. health education materials)	(Yu et al. 2012)		BMI decrease: (bb) MD 2.10 (2.02 to 2.18)	118 (1 RCT)	
(Diet, exercise, and breastfeeding vs. usual care)	(Ferrara et al. 2011)		Postpartum weight goal achievement: 37.5 vs. 21.4%, <i>p</i> = 0.07	197 (1 RCT)	
(Diet vs. control)	(Peterson and Jovanovic 1995)		Weight lost at 6 weeks from baseline in the intervention groups ( <i>p</i> ≤ 0.03) but not at 12 weeks follow-up	25 (1 RCT)	Peterson and Jovanovic 1995 was a crossover RCT of 25 obese women, of whom 13 had previous GDM
	(Shyam et al. 2013)		Weight: MD -0.50 (-6.03 to 5.03); BMI: MD -0.50 (-2.62 to 1.62); WC: MD -1.00 (-5.30 to 3.30); WHR: MD 0.00 (-0.02 to 0.02)	77 (1 RCT)	
(Exercise vs. usual care)	(Cheung et al. 2011)		BMI: MD 3.40 (-2.21 to 9.01)	34 (1 RCT)	
	(Kim et al. 2012)		Weight: (bb) MD -1.36 (-3.13 to 0.41); BMI: (bb) MD -0.46 (-1.13 to 0.21); WC: (bb) MD -0.97 (-4.57 to 2.63)	42 (1 RCT)	
	(McIntyre et al. 2012)		Weight: (bb) MD 0.75 (-2.40 to 3.90); WC: (bb) MD 3.25 (-1.5 to 8.00); Body fat: (bb) MD 2.20 (-0.82 to 5.22)	25 (1 RCT)	

It was not considered appropriate to use The Grades of Recommendation, Assessment, Development and Evaluation (GRADE) approach to evidence quality, as diverse methodological approaches were used in the interventions and various review designs were employed. The methodological quality of the included systematic reviews was assessed by the AMSTAR instrument and is presented in the ESM

CI confidence interval, RR risk ratio, MD mean difference, (bb) change between baseline and end of study, FG fasting glucose, FI fasting insulin, 2 h OGTT 2-hour post-load plasma glucose, HOMA-IR: homeostasis model assessment for insulin resistance, HbA1c: glycated hemoglobin, BMI body mass index, WC waist circumference, HC hip circumference, WHR waist-to-hip ratio, weight units: kg, BMI units: kg/m<sup>2</sup>

\* The risk in the intervention group (and its 95% confidence interval) is based on the assumed risk in the comparison group and the relative effect of the intervention (and its 95% CI)

HOMA-IR (MD -0.25, 95% CI -0.45 to -0.05, *n* = 404) were significantly larger in the intervention group than in the control group (Table 4) [26]. Ji 2011 showed that there were statistically significant differences in the decrease in

FG (MD -0.33, 95% CI -0.41 to -0.25, *n* = 130), 2 h OGTT (MD -0.70, 95% CI -0.87 to -0.53, *n* = 130), HOMA-IR (MD -0.27, 95% CI -0.38 to -0.16, *n* = 130), and HbA1c (MD -2.08, 95% CI -2.13 to

−2.03,  $n = 130$ ) between the intervention group and the control group [27]. Ratner et al. 2008 reported that there was a significant difference in the decrease in 2 h OGTT in the intervention group compared to the control [31]. Shek et al. 2014, however, reported no difference in any glycemic measure between the intervention and control groups at final follow-up [33]. Wein et al. 1999 reported a statistically significant decrease in FG (MD −0.40, 95% CI −0.71 to −0.09,  $n = 193$ ) in the intervention group compared to the control group, but observed no statistically significant difference in 2 h OGTT (MD −0.10, 95% CI −0.73 to 0.53,  $n = 193$ ) [35].

#### *Diet, exercise, and psychosocial support vs health education materials for glycemic load*

One included review evaluated a diet, exercise, and psychosocial support intervention for glycemic outcomes from one trial (Table 3b) [19]. Yu et al. 2012 reported that the reduction in HOMA-IR (baseline compared to end point) was significantly larger in the intervention group than in the control group (MD 0.35, 95% CI 0.33 to 0.37,  $n = 118$ ) (Table 4) [36].

#### *Diet vs. control for glycemic load*

Four of the included reviews evaluated diet intervention-associated glycemic outcomes from two trials that included 102 women diagnosed with GDM [17–19, 21]. Peterson and Jovanovic 1995 found no change in FI at follow-up [30]. Shyam et al. 2013 reported that 61.5% of the intervention group had an overall FI measurement of less than 2 mU/L compared to 52.6% of the control group,  $p = 0.228$  (Table 3b) [34]. Meanwhile, no significant difference was found for FG (MD 0.10, 95% CI −0.31 to 0.51,  $n = 77$ ) and 2 h OGTT (MD 0.00, 95% CI −1.02 to 1.02,  $n = 77$ ) between the intervention group and the control group (Table 4).

#### *Exercise vs. usual care for glycemic load*

Three of the included reviews examined exercise interventions for glycemic outcomes based on results from two trials that included 49 women diagnosed with GDM (Table 3b) [17–19]. Kim et al. 2012 reported that FG (MD 0.08, 95% CI −0.28 to 0.45,  $n = 42$ ), 2 h OGTT (MD 0.06, 95% CI −0.98 to 1.10,  $n = 42$ ), and log FI (MD −0.19, 95% CI −0.43 to 0.05,  $n = 42$ ) did not show statistically significant differences between the intervention group and the usual care group when the change from the baseline to the end point was compared (Table 4) [28]. McIntyre et al. 2012 reported that changes in baseline to end-point measurements for FG (MD 0.13, 95% CI −0.25

to 0.51,  $n = 25$ ), FI (MD 1.43, 95% CI −1.76 to 4.62,  $n = 25$ ), and HOMA-IR (MD 0.51, 95% CI −0.39 to 1.41,  $n = 25$ ) were slightly larger in the intervention group than in the usual care group; however, the CIs were too wide and included the null value [29].

#### *Reminder system vs. no reminder for glycemic load*

One included review [20] evaluated one trial of a reminder system intervention for glycemic outcome measurements, Clark et al. 2009, but no data was contributed by the trial (Table 3b) [24].

### **Interventions for anthropometric changes**

Five included reviews [17–19, 21, 22] evaluated anthropometric changes in women diagnosed with GDM, such as body mass index (BMI) in  $\text{kg/m}^2$ , waist circumference (WC) or hip circumference (HC) in cm, waist-to-hip ratio (WHR), and weight in kg. The definitions in regard to the measurements used in the reviews were referenced from World Health Organization (WHO) criteria (Table 3c). Five interventions were described: diet and exercise vs control; diet, exercise, and psychosocial support vs health education materials; diet, exercise, and breastfeeding vs usual care; diet vs control; and exercise vs usual care. The included reviews assessed data from 13 trials on anthropometric changes as a predictor of T2DM: Cheung et al. 2011, Ferrara et al. 2011, Hu et al. 2012, Ji 2011, Kim et al. 2012, McIntyre et al. 2012, Peterson and Jovanovic 1995, Ratner et al. 2008, Reinhardt et al. 2012, Shek et al. 2014, Shyam et al. 2013, Wein et al. 1999, and Yu et al. 2012 [23, 25–36]. Of the 13 trials, data from Ji 2011 and Yu et al. 2012 were not assessed by more than one review [27, 36]. Hu et al. 2012, Kim et al. 2012, McIntyre et al. 2012, Peterson and Jovanovic 1995, Shek et al. 2014, and Yu et al. 2012 measured the difference in the change between baseline and end-point measurements between compared groups, whereas the other trials showed only the overall end-point difference between the compared groups [26, 28–30, 33, 36].

#### *Diet and exercise vs control for anthropometric changes*

Four of the included reviews [17–19, 22] examined the effect of diet and exercise interventions on anthropometric changes in 1447 women diagnosed with GDM from six trials (Table 3c). Hu et al. 2012 reported a significant difference in change from the baseline to the end-point between the compared groups for weight (MD −1.19, 95% CI −1.87 to −0.51,  $n = 404$ ), BMI (MD −0.41, 95% CI −0.68 to −0.14,  $n = 404$ ), percentage of body fat (MD −0.85, 95% CI −1.33 to −0.37,  $n = 404$ ), and WC (MD

−1.52, 95% CI −2.55 to −0.49,  $n = 404$ ), but not for HC (MD −0.44, 95% CI −1.31 to 0.43,  $n = 404$ ) (Table 4) [26]. Ji 2011 reported no significant difference in weight (MD −0.59, 95% CI −3.00 to 1.82,  $n = 130$ ) and BMI (MD −1.73, 95% CI −3.88 to 0.42,  $n = 130$ ) between the intervention group and the control group [27]. Ratner et al. 2008 observed weight loss at the end-point of the study in both the lifestyle intervention and control groups (Table 3c) [31]. Reinhardt et al. 2012 reported no significant difference between the compared groups for weight (MD −5.10, 95% CI −15.67 to 5.47,  $n = 31$ ), BMI (MD −1.06, 95% CI −5.45 to 2.25,  $n = 31$ ), and WC (MD −7.30, 95% CI −15.35 to 0.75,  $n = 31$ ) [32]. Shek et al. 2014 indicated that BMI and percentage body fat were significantly lower in the intervention group during some visits, but not consistently throughout the study period [33]. Wein et al. 1999 reported that there was no significant difference between the compared groups for BMI (MD −0.20, 95% CI −1.88 to 1.48,  $n = 193$ ); instead, an increase in BMI was observed when comparing the end result to the baseline in both groups [35].

#### *Diet, exercise, and psychosocial support vs health education materials for anthropometric changes*

One included review [19] evaluated a diet, exercise, and psychosocial support intervention for anthropometric outcomes based on the results of one trial (Table 3c). Yu et al. 2012 reported that the decrease in BMI from the baseline to the study end-point was significantly larger in the intervention group compared to the control group (MD 2.10, 95% CI 2.02–2.18,  $n = 118$ ) (Table 4) [36].

#### *Diet, exercise, and breastfeeding vs usual care for anthropometric changes*

Four of the included reviews [17–19, 22] examined a diet, exercise, and breastfeeding intervention for anthropometric outcomes based on data gathered from one trial involving 197 women diagnosed with GDM (Table 3c). Ferrara et al. 2011 reported that 37.5% of the women in the intervention group reached the postpartum weight goal, compared to 21.4% of the women in the control group (Table 4) [25].

#### *Diet vs control for anthropometric changes*

Four of the included reviews [17–19, 21] evaluated diet interventions for anthropometric outcomes in two trials that included 102 women diagnosed with GDM (Table 3c). Peterson and Jovanovic 1995 observed a significant loss of weight at 6 weeks from the baseline measurement in the intervention groups, but after the completion of the trial at 12 weeks, weight loss had attenuated compared to that seen

at 6 weeks (Table 3c) [30]. Shyam et al. 2013 found no statistically significant difference between the intervention group and the control group for weight (MD −0.50 95% CI −6.03 to 5.03,  $n = 77$ ), BMI (MD −0.50 95% CI −2.62 to 1.62,  $n = 77$ ), WC (MD −1.00, 95% CI −5.30 to 3.30,  $n = 77$ ), and WHR (MD 0.00, 95% CI −0.02 to 0.02,  $n = 77$ ) (Table 4) [34].

#### *Exercise vs usual care for anthropometric changes*

Four of the included reviews [17–19, 22] assessed data on exercise interventions for anthropometric outcomes from three trials that included a total of 101 women diagnosed with GDM (Table 3c). Cheung et al. 2011 reported an increase in BMI (MD 3.40, 95% CI −2.21 to 9.01,  $n = 34$ ) in the intervention group compared to the control group, but the difference did not reach statistical significance (Table 4) [23]. Kim et al. 2012 reported that there were no significant differences in the reduction in weight (MD −1.36, 95% CI −3.13 to 0.41,  $n = 42$ ), BMI (MD −0.46, 95% CI −1.13 to 0.21,  $n = 42$ ), and WC (MD −0.97, 95% CI −4.57 to 2.63,  $n = 42$ ) between the baseline and the end point when comparing the intervention group with the control group [28]. McIntyre et al. 2012 reported that there was no significant difference between the intervention and control groups in the baseline-to-end-point changes in weight (MD 0.75, 95% CI −2.40 to 3.90,  $n = 25$ ), WC (MD 3.25, 95% CI −1.5 to 8.00,  $n = 25$ ), and body fat (MD 2.20, 95% CI −0.82 to 5.22,  $n = 25$ ) [29].

## **Discussion**

This systematic overview compiled evidence from six reviews into one accessible document and highlighted important evidence that could inform future clinical practice and research. In addition, this study identified six types of nonpharmacological interventions: diet and exercise; diet, exercise, and psychosocial support; diet alone; exercise alone; diet, exercise, and breastfeeding; and reminder system. These interventions were assessed for three key outcomes: prevention of T2DM, reduction in glycemic load, and anthropometric changes. Based on our current summary of the six review data, there is no robust evidence demonstrating that nonpharmacological interventions are effective at preventing T2DM in women diagnosed with GDM. The evidence found in the included reviews was obtained from randomized controlled trials; however, only four of the included reviews assessed the quality of each trial, and the methods used were not consistent between the reviews [18–21]. The methodology in the included reviews varied greatly, even though the outcome and intervention interests were the same. Most of the relevant evidence was obtained



from the two most recent reviews included [18, 19], and they concluded that the effectiveness of the nonpharmacological interventions at preventing T2DM in women diagnosed with GDM was still lacking and that more interventions using technology with long-term efficacy evaluations were warranted. Since quality assessments were not practiced under methodological standards in each included review except for Middleton 2014, it is highly possible that the evidence drawn from these included reviews was at risk of largely unclear bias, which lowers the reliability of their interpretations [20]. Future reviews of lifestyle interventions for women with GDM should use standardized methods to appraise the validity of the evidence used.

For the diet and exercise intervention, four of the included reviews reported that Ratner et al. 2008 found a statistically significant reduction in T2DM progression, but Shek et al. 2014 did not [31, 33]. Differences in the implementation of the intervention could be one of the factors contributing to the inconsistency among the reviews; Ratner et al. 2008 assessed women enrolled in the Diabetes Prevention Program in the United States, who underwent a minimum of 150 min of physical activity with individualized sessions, whereas Shek et al. 2014 only involved counseling by a dietician. Similarly, the included reviews suggested that a diet and exercise intervention was effective at lowering the glycemic load in women diagnosed with GDM, but our systematic overview found that the only measurement that showed a consistent reduction in the intervention group was HOMA-IR. All of the identified trials in the included reviews presented inconsistent results for the other measured glycemic outcomes, so it was not possible to draw conclusions about the true effect of the diet and exercise intervention. Anthropometric outcomes from diet and exercise interventions were also demonstrated to be inconsistent. The included reviews described the intervention as being effective based on the difference between groups in the change between the baseline and the end of the study (e.g., Hu et al. 2012 and Shek et al. 2014), but the overall mean difference between groups did not reach statistical significance. This could imply that individual achievements account for a larger proportion of the statistical difference in anthropometric changes as opposed to the average end-point measurement of the population. Since anthropometric measures are highly influenced by multiple confounding factors, such as sociodemographic characteristics, genetic background, and stress, it would be advantageous to consider the confounding factors and conduct a sensitive analysis (e.g., subgroup analysis or meta-regression) in future systematic reviews [37].

One included review evaluated the effect of a diet, exercise, and psychosocial support intervention based on one trial by Yu et al. 2012, and showed a significant difference in the annual incidence of T2DM, but the

difference in relative risk between the intervention and control groups did not reach statistical significance. The review did report, however, that there was a significant difference between the comparison groups in the lowering of HOMA-IR and BMI [19, 36]. This evidence suggests that home visits with psychosocial support could be beneficial for promoting diet and exercise motivation.

Neither diet alone nor exercise alone proved to be effective interventions for lowering the risk of T2DM, reducing glycemic load, or anthropometric outcomes. The reviews suggested that dietary intake is strongly related to culture or belief and physical activity is related to the condition of the individual; therefore, compliance with either diet or exercise intervention alone requires high motivation from the population. Additionally, hormonal changes among the study participants could have induced tremendous stress during their dietary control, and vigorous exercise could have also resulted in a sudden increase in food intake [38]. Perhaps it would be worth evaluating these factors and considering them in future intervention design. With regards to diet, exercise, and breastfeeding, Ferrara et al. 2011 suggested that the intervention was effective at returning women to their postpartum weight. Weight change is one of the main predictor outcomes for T2DM. This is consistent with a recent prospective cohort study called the Study of Women, Infant Feeding, and Type 2 diabetes mellitus after GDM pregnancy (SWIFT), which suggested that breastfeeding was effective at supporting weight reduction in women [39]. As for reminder interventions, there was a lack of studies for assessment and comparison [20].

Our overview provided an opportunity to undertake a comparison of the diverse evidence for the benefits of different interventions aimed at preventing T2DM in women diagnosed with GDM; however, the limitations of this systematic overview should be recognized when interpreting the summaries. This systematic overview of reviews could only provide evidence reported in the reviews; it could not evaluate the quality of the RCTs directly. In the included reviews (except in one review: Middleton 2014), there was no description of the diagnostic criteria that were used to confirm GDM participants, and there was no description of the diagnostic criteria for T2DM that were used in the RCTs. Although the key challenge remains intervention heterogeneity, this systematic overview was able to unify information on the outcomes of women diagnosed with GDM.

## Conclusions

This systematic overview summarized evidence from multiple reviews and found that there is insufficient evidence to support the idea that nonpharmacological

interventions are effective at lowering the risk of T2DM. Based on the review reports, there is no consensual evidence that nonpharmacological interventions can improve predictor outcomes of T2DM such as glycemic load or anthropometric changes. Future systematic reviews should be updated with more interventions studies and should be conducted with standardized reporting methods; meanwhile, policymakers may use this overview of the evidence to identify knowledge gaps for future research.

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#### Compliance with ethical standards

**Conflict of interest** Celine Miyazaki, Kanako Tanase-Nakao, Naoko Arata, Rintaro Mori, Maki Kawasaki, and Erika Ota declare no competing financial interests.

**Ethics approval** This study respects the fundamental ethical principles consistent with the Declaration of Helsinki of The World Medical Association (WMA) 2013. This systematic overview only reviews published medical research studies that respected fundamental ethical principles, and it does not involve any animal or human subjects, including research on identifiable human material and data. The reporting of this study complies with the PRISMA checklist for systematic review and meta-analyses and was conducted only by individuals with the appropriate ethics and scientific education, training, and qualifications.

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