Review Article

Systematic review and meta-analysis of nutritional interventions to prevent of gestational hypertension or/and preeclampsia among healthy pregnant women

Vajihe Imanpour, Mehri Khoshhali[1], Masoomeh Goodarzi-Khoigani[1], Roya Kelishadi[2]

Department of Physiology, School of Medicine, Isfahan University of Medical Sciences, Isfahan, Iran, [1]Child Growth and Development Research Center, Research Institute For Primordial Prevention of Non-communicable Disease, Isfahan University of Medical Sciences, Isfahan, Iran, [2]Department of Pediatrics, Child Growth and Development Research Center, Research Institute For Primordial Prevention of Non-communicable Disease, Isfahan University of Medical Sciences, Isfahan, Iran

Background: Researchers have shown that diet is associated with hypertensive disorders of pregnancy, and there are some reports of performed meta-analyses on observational studies. However, very few randomized-controlled trials have systematically summarized. Thus, we reviewed and meta-analyzed the effects of nutritional interventions on risks of gestational hypertension (GH) or/and preeclampsia (PE). **Materials and Methods:** A systematic search was performed using Medline, Cochrane library, Google Scholar, ISI Web of Science, Scopus, and ProQuest to find randomized clinical trials assessing the effect of nutritional interventions on incidences of GH or/and PE compared to control or placebo interventions. **Results:** After considering duplicates, 1066 articles were screened from the database searches. Full-text articles were retrieved for 116 records, while 87 did not have the inclusion criteria and were later omitted. Twenty-nine studies were eligible, but 8 studies were not included in the meta-analysis due to insufficient data. Finally, seven studies were included in qualitative analysis. Furthermore, 7 studies (693 in intervention vs. 721 in control) were pooled for managed nutritional interventions, three (1255 vs. 1257) for a Mediterranean-style diet, and 4 (409 vs. 312) for sodium restricted. Our results revealed that managed nutritional programs were effective in reducing the incidence of GH (odds ratio [OR] = 0.37; 95% confidence interval [CI] = 0.15, 0.92); I^2 = 66.9%; P = 0.010), but not for PE (OR = 0.50; 95% CI = 0.23, 1.07); I^2 = 58.9%; P = 0.032. The Mediterranean-style diets in three trials (1255 vs. 1257) did not reduce the risk of PE (OR = 1.10; 95% CI = 0.71, 1.70); I^2 = 2.3%; P = 0.359). Likewise, sodium-restricted interventions in four trials (409 vs. 312) did not decrease total risk of GH (OR = 0.99; 95% CI = 0.68, 1.45); I^2 = 0%; P = 0.520). Meta-regression did not indicate any significant association between maternal age, body mass index, gestational weight gain, and start time of all interventions with the incidence of GH or/and PE (P > 0.05). **Conclusion:** The present meta-analysis showed that Mediterranean-style diets and sodium-restriction interventions did not decrease the incidence of GH or/and PE in healthy pregnancies; however, managed nutritional programs reduced the risk of GH, the total incidence of GH and PE, but not PE.

Keywords: Diet, Food, and Nutrition; Hypertension, Pregnancy-Induced; Nutrition survey; Pre-eclampsia

How to cite this article: Imanpour V, Khoshhali M, Goodarzi-Khoigani M, Kelishadi R. Systematic review and meta-analysis of nutritional interventions to prevent of gestational hypertension or/and preeclampsia among healthy pregnant women. J Res Med Sci 2023;28:25.

INTRODUCTION

Gestational hypertension (GH) and preeclampsia (PE) are the most frequent hypertensive disorders of pregnancy (HDPs). GH is one of the important causes of maternal mortality worldwide with a mean incidence of 6.3%.[1,2] PE increases the risk of perinatal mortality and is the cause of approximately 15% of preterm birth and 10% of stillbirth.[3] GH is defined a new rise in systolic blood pressure (SBP) ≥140 mmHg and/or diastolic blood

Access this article online

Quick Response Code:

Website: www.jmsjournal.net

DOI: 10.4103/jrms.jrms_89_22

Address for correspondence: Dr. Masoomeh Goodarzi-Khoigani, Child Growth and Development Research Center, Research Institute For Primordial Prevention of Non-communicable Disease, Isfahan University of Medical Sciences, Isfahan, Iran.
E-mail: masoumeh_goodarzi@nm.mui.ac.ir, masoomeh.goodarzi.kh@gmail.com
Submitted: 31-Jan-2022; **Revised:** 12-Oct-2022; **Accepted:** 27-Oct-2022; **Published:** 06-Apr-2023

pressure (DBP) ≥90 mmHg which detected after 20 weeks of gestation; and PE is defined by the combination of GH and proteinuria (emission of ≥ 300 mg protein every 24 h).[3,4]

Researchers showed that diet is associated with HDPs, for example, a cohort study showed that higher consumption of saturated fat in the first trimester of gestation and lower intakes of manganese, Vitamin C, Vitamin E, fiber, and carbohydrate in the third trimester enhanced the risk of PE.[5] Furthermore, an inverse association between following the dietary approaches to stop hypertension (DASH) dietary pattern and risk of PE was reported in a case–control study.[6] Moreover, randomized-controlled trials (RCTs) showed that GH and PE can be effectively controlled by nutritional interventions during pregnancy.[7-9] In opposite, some RCTs were not effective on GH or/and PE.[10-12]

Reviews systematically summarized the correlation between nutrient intake and incidence of GH or/and PE in observational studies; and concluded that greater energy, lesser magnesium, and calcium intakes during pregnancy were associated with HDP.[13] In addition, a higher intake of fish, whole grains, legumes, vegetables, and fruits were related to decreased risk of HDPs.[14] But RCTs, especially in healthy pregnancy, have been rarely meta-analyzed; thus, we conducted a comprehensive review and meta-analysis of the effects of nutritional interventions on risks of GH or/and PE.

MATERIALS AND METHODS

Search strategy

This systematic review and meta-analysis were reported with a prospective protocol in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines and PICO. We searched Scopus, Medline, Cochrane library, Google Scholar, ISI Web of Science, ProQuest, and reference lists of selected papers from April to November 2021. In the first step of electronic searches, we selected related RCTs and meta-analyses with the following search terms in the titles, abstracts, or keywords: ([“nutrition*education” OR “nutrition* intervention” OR “education* intervention” OR “nutrition* counselling”] AND [“Pre-Eclampsia” OR “Hypertension, Pregnancy-Induced” OR “Gestational Hypertension”]). Then, the reference lists of eligible RCTs and related meta-analyses were surveyed to determine studies that were not found by the electronic searches, while those reported the effect of nutritional interventions on GH or/and PE. In the next stage, the full text of the specified papers was assessed to find trials which fulfilled the inclusion criteria and reported the effect of only dietary interventions on GH or/and PE, compared to control or placebo interventions. Non-randomized, animal studies, and RCTs based on mixed diet and/or other interventions such as physical activity (except 30 min of walking per day which is a common recommendation) were excluded. Studies were selected in two stages by the corresponding author (M G-KH) and were confirmed by the second author (M Kh). This systematic review and meta-analysis were registered by PROSPERO team (CRD42021259200). Endnote software (Thomas Reuters, Philadelphia, PA) was used to control the findings of the search as identified by the stated strategies.

Subjects

They were healthy, nonsmoking, pregnant women with a singleton pregnancy, free of history of any acute and chronic medical problems, usage of drugs, alcohol, tobacco, or medications during the pregnancy. In addition, they were free of high-risk pregnancies caused by diabetes mellitus, endocrine disease, or chronic hypertension.

Study selection

Data extraction

The first author’s name, publication year, country, description of intervention, intervention duration, age, pre-pregnancy body mass index (BMI), educational goals, sample size, incidence of GH or/and PE (before and after intervention) were extracted by the corresponding author (M G-KH), and were confirmed by the second author (M Kh) [Tables 1 and 2].

Quality assessment

Cochrane risk of bias assessment tool was used to evaluate randomization performance and methods, allocation concealment, baseline imbalances, extent of blinding (patients, caregivers, data collectors, outcome assessors, and data analysts), rate of loss to follow-up, and monitoring of adherence.

We scrutinized the study quality and risk of bias of involved RCTs through pregnancy with the Cochrane collaboration’s tool for assessing the risk of bias [Table 3]. One author (M G-KH) evaluated the quality, and the other author(M KH) approved that.

Statistical analysis

The effect sizes of odds ratio (OR) and their 95% confidence intervals (CIs) or standard errors (SE) were extracted from original studies. For studies where the ORs were not reported, they were calculated from available frequencies. The potential heterogeneity across studies was assessed using Cochran’s Q-test and expressed using the I^2 index. A random-effects model was used to estimate the pooled OR to measure the effect of nutritional interventions on GH or PE in healthy pregnancy which is valuable clinically, and the total risk of GH and PE, which was estimated because of small number of included studies. Age, gestational

Table 1: Study characteristics

Author, year of publication, and Country	Sample of intervention (*n*)	Sample of control (*n*)	Nutritional intervention	PE or GH Intervention versus control, rate (%)
Abdel-Aziz *et al*., 2018,[7]* Cairo	75	72	Nutritional goals based on the food pyramid, eating healthy foods, correct eating habits, prevention of EGWG, limit unhealthy foods and snacks, recommend to walk for 30 min (3 times/week). Having regular meals based on the food groups of the food pyramid	PIH 4 (5.3) versus 14 (19.4) *P*<0.009
Van Buul *et al*., 1997,[10] The Netherlands	25	25	A diet containing 20 mmol/day of sodium. Salt should not be added during cooking or at the table	13.6% versus 12.9% No significant differences between two groups regarding systolic, diastolic, and mean arterial BP
Abrha *et al*., 2020,[29] Ethiopia	203	212	Baseline calcium intake of 1200 mg/day in accordance with 24-h recall and the benefit of that was explained to participants Also, a dietary plan was presented to mothers	As calcium intake enhaced1 unit, the BP decreased by 0.99 unit (AOR=−0.99; 95% CI: 0.993-0.998) Differences in health facility delivery were significant (τ=0.81, *P*<0.001)
Sun and Niu, 2020,[8] China	582	580	To eat at least 100 g of white button mushrooms/day which could be provided based on individuals' preferences	GH 24 (4.1) versus 48 (8.2) *P*=0.023 PE 4 (0.7) versus 12 (2.1) *P*=0.014
Khoury *et al*., 2005,[11] Norway	141	149	Consumption a cholesterol-lowering diet (150 mg cholesterol per day)	PE 7 versus 8 Not significant
Thornton *et al*., 2009,[12] New york	116	116	Intake of 18-24 kcal/kg well-adjusted nutritional regimen, but not<2000 calories per day, having 30 min of walking per day	PE *P*=0.326 7 (6.0) versus 11 (9.5) GH *P*=0.46 3 (2.6) versus 10 (8.6)
Wolff *et al*., 2008,[15] Denmark	23	27	A healthy regimen according to Danish guideline	PE 0 (0%) versus 1 (4%) *P*=Not written
Yang *et al*., 2018,[18] China	39	39	A clear and reasonable diet, to manage the individuals' diet based on the targets and participants' preferences	GH 1 versus 8 *P*<0.05
Jiang *et al*., 2019,[26] China	44	41	DASH and the amount of received salt was 4 g/day. The Control group received a medical diet which its total energy was calculated by ideal body weight × (25-35) kcal/day	PE *P*<0.05
Vesco *et al*., 2013,[27] USA	56	58	Intervention arm in weekly sessions were advised to follow the individualized calorie goals and DASH diet without sodium limitation	GH, PE OR: 0.85 95% CI for OR: 0.24-2.96 *P*=0.02 5 (9%)
Chan *et al*., 2006,[28] USA	25 (dairy)	24 (orange juice)	23 (control) The orange juice in addition to calcium group was advised to consumption at least 4 portions of orange juice in addition to calcium >1200 mg to be similar to the dairy group. The dairy group was advised to intake of at least 4 portions of dairy/day to receive >1200 mg calcium. Having a proper nutrition was asked of all women. The control group received their routine diet	Not significant differences were observed between systolic and diastolic BP
Seo *et al*., 2020,[22] Korea	98	44	Following the World Health Organization and "Dietary Reference Intakes for Koreans" recommendations for low sugar and low sodium	PE and GH *P*=0.48 SBP 117±9.7 versus 123.9±14.5 *P*=0.047 DBP 71.8±6.5 versus 78.0±12.2 *P*=0.018

Contd...

Table 1: Contd...

Author, year of publication, and Country	Sample of intervention (*n*)	Sample of control (*n*)	Nutritional intervention	PE or GH Intervention versus control, rate (%)
Assaf-Balut *et al*., 2018,[19] USA	500	500	Healthy diet, to walk ≥30 min/day. The key advise was consumption of at least 40 mL of EVOO and 25-30 g of pistachios per day	PIH 13 (3.0) versus 19 (4.30) *P*=0.195 PE 7 (1.6) versus 11 (2.5) *P*=0247
H. Al Wattar *et al*., 2019,[20] England	675	625	A Mediterranean-style diet. The control group was advised to follow of the UK national recommendations	PE *P*=0.19 34 (6.2%) versus 27 (4.6%)
Rhodes, 2010,[25] USA	22 Low-GL diet	16 Low-fat diet		SBP (mm Hg) 0±9 versus 2±14 *P*=0.47 DBP (mm Hg) 1±5 versus 3±6 *P*=0.31
Phelan *et al*., 2011,[16] Providence, Rhode Island	90 Normal 81 Overweight	92 Normal 86 Overweight	The intervention was advised to a calorie goals (20 kcal/kg) and physical activity (30 min of walking most days of the week). The control group received their routine diet	PE 3 versus 9 in normal weight 17 versus 11 in overweight *P*=0.02 GH: 3 versus 11 in normal weight 17 versus 11 in overweight OR: 0.15; 95% CI: 0.02-0.75; *P*=0.02
Melero *et al*., 2020[21]	128 (IG)	132 (CG)	284 (RW)	The intervention and RW were recommended to intake ≥40 mL/day of EVOO and 25-30 g of pistachios at least 3 days a week	
Luo *et al*., 2014,[17] China	131	145	A medical nutrition plan for interventional groups, an individualized calorie intake diet. The same preliminary information of the study	PE 4 (3.1%) versus 16 (11.0%) *P*=0.011
Knuist *et al*., 1998,[23] The Netherlands	184	117	A diet with lesser than 50 mmol sodium per day. The group assigned to normal diet was requested not to change their eating habits	Change in DBP (mmHg) +6.5±9.6 versus+6.5±10.4
Steegers *et al*., 1991,[24] The Netherlands	17	19	A diet including 20 mmol sodium daily compared to continue to unrestricted dietary intake	BP during pregnancy did not show major differences

*The number of reference of each study has been superscripted. BP=Blood pressure; OR=Odds ratio; AOR=Adjusted OR; CI=Confidence interval; DBP=Diastolic BP; SBP=Systolic BP; PE=Preeclampsia; GH=Gestational hypertension; PIH=Pregnancy-induced hypertension; DASH=Dietary Approaches to Stop Hypertension; GL=Glycemic; EVOO=Extra virgin olive oil; EGWG=Excessive gestational weight gain; RW=Real-world group; CG=Control group; IG= Intervention group

weight gain (GWG), pre-gravid BMI, and starting trimester of intervention were extracted for meta-regression as the possible source of heterogeneity [Table 2]. The sensitivity analyses were considered by excluding one or several studies at a time to gauge the robustness of our results. Publication bias was investigated by Egger's test. All statistical analyses were conducted using software STATA 12.0 (STATA Corp, College Station, Texas, USA).

RESULTS

Study selection process

We showed search results and selection process in Figure 1.

Description of included trials

A total of 1066 studies were retrieved and 21 were included in our study that examined the effect of nutritional interventions on the incidence of GH or/and PE [Figure 1]. Fourteen trials (4647 participants) were selected for meta-analysis, of which 7 trials (693 vs. 721 women) have studied the effect of educational and nutritional interventions (educational and nutritional interventions which administrated gestational weight gain, also recommended healthy and balanced nutrition based on national guidelines [managed nutritional programs]) on the incidence of GH or/and PE,[7,9,12,15-18] three trials (1255 vs. 1257 women) examined the impact of the Mediterranean diet,[19-21] four evaluated the influence of sodium-restricted trials (409 vs. 312 women).[10,22-24] In addition, seven trials were eligible for systematic review, of which 1 study assessed the effect of low-glycemic diet compared to low-fat diet,[25] two evaluated the impact of DASH diet,[26,27] two studied the influence of enhancement of calcium intake[28,29] on risk of GH or/and PE, one trial surveyed the effect of mushroom diet,[8] and one intervention investigated the effect of cholesterol-lowering diet.[11] Eligible studies have been performed on pregnant women with all BMI groups, except three trials which did not include only underweight

Table 2: Date for met-regression

Study	Age (years) I versus C	GWG (kg) I versus C	*P*	Prepregnancy BMI (kg/m²)	Prepregnancy BMI group (1, 2, 3, 4)	Duration (weeks)	Beginning trimester (1 or 2 or 3)
Abdel-Aziz *et al*.[7]*	20-30		<0.001		1, 2, 3, 4	28-34	1 (<12 weeks)
Van Buul *et al*.[10]	28.1 versus 28.3	8.8 (−3.6-23.7) versus 11 (−1.3-20.8)	0.005	25.4 (20.0-43.2) versus 26.5 (19.5-45.8)	2, 3, 4	Ut most 26	2 and 3 (at least 14 weeks)
Abrha *et al*.[29]	20-49 Mean (39±15)	11.8±3.9 versus 12.5±4.5	0.017	BMI<30	1, 2, 3	12-16	3
Sun and Niu[8]	31.2±4.5 versus 31.4±4.3			22.47±3.66 versus 22.61±4.01	1, 2, 3, 4	12-16	1 and 2 (<20th week)
Khoury *et al*.[11]		8.9±3.1 versus 9.4±3.0	OR: 0.5 (95% CI: 0.2-1.3)	19-32 24.3±2.9 versus 24.3±2.7	2, 3, 4		1 and 2 and 3 (between inclusion and week 30
Thornton *et al*.[12]	26.8 versus 27.3	11±14.96 versus 31±16.31 95% CI: 8.59-14.10 versus 95% CI: 27.82-33.82	<0.001	BMI ≥30 37.41±7.01 versus 38.22±7.48	4	2 (12-28 weeks)	12-28 Ut most 28 Duration
Wolff *et al*.[15]		6.6±5.5 versus 13.3±7.5	0.002	BMI ≥30 34.9±4 versus 34.6±3	4	~21-27 weeks	2 15±2 versus 16±3 CI: 0-3 weeks
Yang *et al*.[18]	25.5±5.3 versus 24.6±3.7				1, 2, 3, 4	-	-
Zhang[9]	27.84±3.60 versus 27.70±3.73				1, 2, 3, 4	Utmost 28-<28 weeks	2 and 3 (at least 12 weeks)
Jiang *et al*.[26]						~28	~2 and 3
Vesco *et al*.[28]	31.8			36.2	4	20-30 weeks Mean 25 weeks	2 and 3 (at 10-20 weeks) Baseline to 34 weeks
Zerón *et al*.[16]		9±4.5 versus 10.7±3.8	0.192	29.5±2.6 versus 25.5±3.9	1, 2, 3, 4	39.8±0.6 38.1±1.5	
Chan *et al*.[30]	16±0.6 versus 16.6±0.6 16±0.6 versus 16.7±0.6	13.9±12.0 versus 14.1±9.6 13.9±12.0 versus 14.0±11.1	>0.05	25±4 versus 25±5 26±5 versus 25±5	1, 2, 3, 4	20-21 weeks	2
Seo *et al*.[22]	33.2±3.7 versus 33.5±3.6	11.8±5.2 versus 10.6±5.2	0.352	<30 22.3±3.5 versus 21.7±4.5	1, 2, 3	At least 16 weeks until childbirth	2 Mean age of gestation for beginning 14.3±4.6 versus 16.4±4.6 weeks
Assaf-Balut *et al*.[19]	33.2±5.0 versus 32.7±5.3	9.9±4.7 versus 9.4±4.3	0.116	22.9±3.6 versus 23.3±4.0	1, 2, 3, 4	28-32 weeks	1 (at 8-12 weeks)
H. Al Wattar *et al*.[20]	31.4±5.2 versus 30.9±5.2	6.8±5.6 versus 8.3±6.4			2, 3, 4	>22 weeks	1, 2 (<18 weeks)
Rhodes *et al*.[25]	33.7±3.9 versus 33.2±3.7	6.4±4.5 versus 6.9±4.2	0.74	≥25 <45 32.1±4.6 versus 31.2±3.1	3, 4		2 (13-28 weeks)
Phelan *et al*.[16]	28.6±5.2 versus 28.8±5.2	NW: 15.3±4.4 versus 16.2±4.6 OW/OB: 14.7±6.9 versus 15.1±7.5		26.32±5.6 versus 26.48±5.9	1, 2, 3, 4	~24-28	2 (13.6±1.8 versus 13.5±1.8) weeks
Melero *et al*.[21]	31.7±5.4 versus 31.3±5.6	IG versus CG GWG to 36-38 weeks: 12.3±5.4 versus 11.3±6.3	0.209	24.1±3.4 versus 24.4±4.0	1, 2, 3, 4	24-26 weeks	2 (12-14 weeks)
Melero *et al*.[21]		IG versus RW GWG to 36-38 weeks: 12.3±5.4 versus 12.5±6.6	0.075	24.1±3.4 versus 23.4±3.6		24-26 weeks	2 (12-14 weeks)

Contd...

Table 2: Contd...

Study	Age (years) I versus C	GWG (kg) I versus C	*P*	Prepregnancy BMI (kg/m²)	Prepregnancy BMI group (1, 2, 3, 4)	Duration (weeks)	Beginning trimester (1 or 2 or 3)
Luo *et al.*[17]	27.3±6.08 versus 27.1±6.12	7.58± 1.59 versus 12.57±4.62	<0.001	21.8±3.08 versus 22.9±2.94	1, 2, 3, 4	27-32	1 (<13 weeks) Mean weeks of gestation 10.1±2.63 versus 10.5±2.46
Knuist *et al.*[23]	27.6±4.2 versus 27.5±4.8		>0.05		1, 2, 3, 4 As if	~21-32	1 and 2 (<20 weeks)
Steegers *et al.*[24]	27 versus 27		<0.01		1, 2, 3, 4 As if	26 weeks	2 and 3 (11-13 weeks)

*The number of reference of each study has been superscripted. Prepregnancy BMI group (1, 2, 3, 4); 1=Under-weight; 2=Normal; 3=Over-weight; 4=Obese. GWG (kg)=Gestational weight gain; BMI=Body mass index; CI=Confidence interval; OR=Odds ratio; RW= Real-world group; CG= Control group; IG= Intervention group; OW/OB=Over weight/Obese; NW=Normal weight

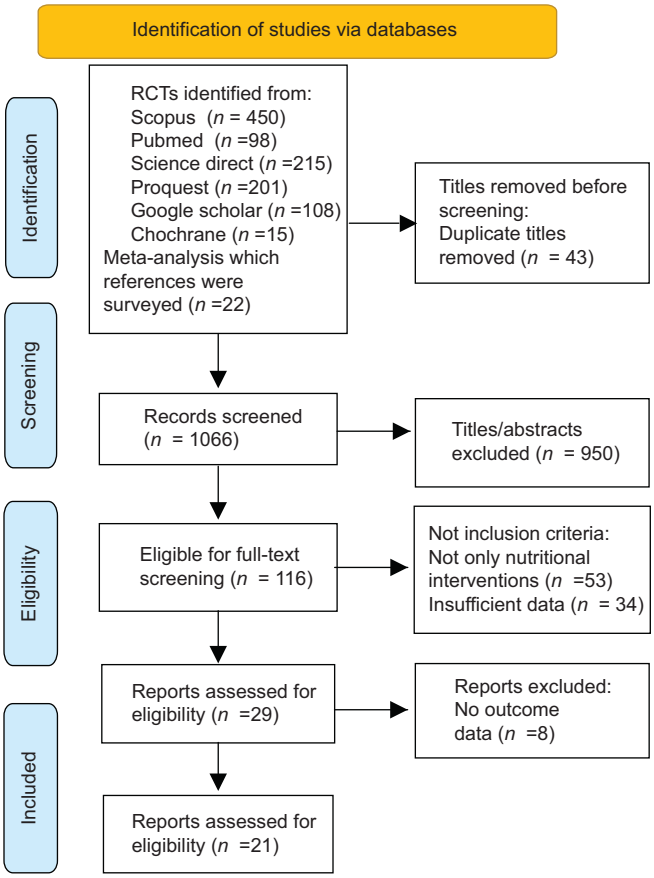

Figure 1: PRISMA flow diagram of RCTs. RCTs: Randomized controlled trials

women.[10,11,19] In addition, three studies included only obese women (BMI ≥30 kg/m²)[12,15,27] and one[25] included overweight and obese women, while two studies[21,28] did not include only obese women. The association of participants' characteristics included age, GWG, pre-pregnancy BMI, duration of intervention, and starting trimester with the impact of interventions on the rate of GH or/and PE were used for meta-regression analysis [Table 2].

Quality of the included studies

The quality of RCTs is shown in Table 3.

Bias assessment

We provided the risk of bias assessment in Table 3, and cases of deficiency were classified as unwritten risk of bias. In all studies, the randomization method has been developed by a numerical list created by a computer system. The sequence generation was observed in all except two studies.[17,24] Allocation concealment from researchers and participants and allocation implementations were not observed in five[21,24,27] and six[7,21,24,27] studies, respectively. The blinding of participants and personnel was not written in 12 studies [Table 3]. The outcomes were analyzed in a blinded way in all except 12 trials [Table 3].

Table 3: Evaluation of bias risk of included randomized-controlled trials

First author, year published	Random ization	Allocation concealment (selection bias)			Blinding of participants (performance bias)	Blinding of care providers (performance bias)	Blinding of outcome assessment (detection bias)	Similarity of baselines	Incomplete outcome data (attrition bias) of	Selective reporting (reporting bias)	Intention to treat analysis
		Sequence generation	Allocation concealment	Allocation implementation							
Abdel-Aziz[7]*	Yes	Yes	Yes	Not-written	Not-written	Not-written	Not-written	Yes	Not	Not	Not-written
van Buul[10]	Yes	Yes	Yes	Yes	Not-written	Not-written	Not-written	Yes	Not	Not	No
Abrha[29]	Yes	Yes	Yes	Yes	Not	Yes	Yes	Yes	Not	Not	Not-written
Sun[8]	Yes	Yes	Yes	Yes	Not-written	Not-written	Not-written	Yes	Not	Not	Not-written
Khoury[11]	Yes	Yes	Yes	Yes	Not	Yes	Yes	Yes	Not	Not	Yes
Thornton[12]	Yes	Yes	Yes	Yes	Not-written	Not-written	Not-written	Yes	Yes	Not	Not-written
Wolff[15]	Yes	Yes	Yes	Yes	Not-written	Yes	Not-written	Yes	Not	Not	Not-written
Yang[18]	Yes	Not-written	Not-written	Not-written	Not-written	Not-written	Not-written	Yes	Not	Not	Not-written
Jiang[26]	Yes	Yes	Yes	Yes	Not	Yes	Yes	Yes	Not	Not	Not-written
Seo[22]	Yes	Yes	Not-written	Not-written	Not-written	Not-written	Not-written	Yes	Not	Not	Not-written
Assaf-Balut[19]	Yes	Yes	Yes	Yes	Not	Not	Yes	Yes	Not	Not	Not-written
H. Al Wattar[20]	Yes	Yes	Yes	Yes	Not	Not	Yes	Yes	Not	Not	Not-written
Rhodes[25]	Yes	Yes	Not-written	Not-written	Not-written	Not-written	Not-written	Not-written	Not-written	Not-written	Not-written
Phelan[16]	Yes	Yes	Yes	Yes	Not	Yes	Yes	Yes	Not	Not	Not-written
Melero[21]	Yes	Yes	Yes	Yes	Not-written	Not-written	Not-written	Yes	Not	Not	Not-written
Luo[17]	Not-written	Not-written	Not-written	Not-written	Not-written	Not-written	Not-written	Yes	Not	Not	Not-written
Knuist[23]	Yes	Yes	Yes	Yes	Not-written	Not-written	Not-written	Yes	Not	Not	Not-written
Steegers[24]	Yes	Yes	Yes	Yes	Not-written	Not-written	Not-written	Yes	Not	Not	Not-written

*The number of reference of each study has been superscripted

Meta-analysis results

Effect of managed nutritional programs on the risk of gestational hypertension or/and preeclampsia

Results of a meta-analysis using a random effect model on 7 studies demonstrated that managed nutritional interventions might be a protective trial against the risk of GH (OR = 0.37; 95% CI = 0.15, 0.92); I^2 = 66.9%; P = 0.010). However, it was not correlated with risk of PE (OR = 0.50; 95% CI = 0.23, 1.07); I^2 = 58.9%; P = 0.032). Considering the total risk of GH and PE as outcome, dietary interventions were effective on total risk of GH and PE (OR = 0.44; 95% CI = 0.25, 0.77); I^2 = 60.2%, P = 0.004) [Figure 2].

The heterogeneity was significant for all grouping. The P value for Egger's test was 0.079, thus, there was no noticeable publication bias among involved studies. Meta-regression determined that there was no significant association between maternal age, BMI, GWG, and start time of intervention with the effect of dietary interventions on the risk of GH and also total risk of GH and PE in pregnant women (P > 0.05). Results of sensitivity analysis demonstrated that after excluding Phelan *et al.*'s trial performed in over-weight women,[16] the effect of dietary interventions was associated with the risk of GH with no heterogeneity (OR = 0.25; 95% CI = 0.14, 0.45); I^2 = 0%; P = 0.988), and also PE (OR = 0.35; 95% CI = 0.20, 0.61); I^2 = 0%; P = 0.764). Moreover, they were related with total risk of GH and PE (OR = 0.30; 95% CI = 0.20, 0.45); I^2 = 0%; P = 0.969) [Figure 3].

Effect of the Mediterranean-style diet on risk of gestational hypertension or/and preeclampsia

Based on the random effect model in three studies, the Mediterranean-style diet did not reduce the risk of PE (OR = 1.10; 95% CI = 0.71, 1.70); I^2 = 2.3%; P = 0.359), and only one study had reported about GH values. In addition, it was not associated with the overall risk of GH and PE in healthy pregnancy (pooled OR = 1.07; 95% CI = 0.72, 1.59); I^2 = 0.0%, P = 0.536) [Figure 4]. The P value for Egger's test was 0.145, thus, there was obvious publication bias among these studies. Meta-regression did not show a significant association between maternal age, BMI, GWG, and start time of intervention with the effect of the Mediterranean-style diet on risk of PE and overall risk of GH and PE (P > 0.05). Sensitivity analysis displayed that after omitting Al Wattar *et al.*'s study,[20] the pooled effect size and heterogeneity decreased (OR = 0.71; 95% CI = 0.34, 1.51); I^2 = 0%, P = 0.714). However, deleting each of the other two studies[19,21] did not change pooled OR and heterogeneity significantly. Moreover, by replacing real-world group instead of the intervention group in Melero *et al.*'s study,[21] the pooled effect size (OR) decreased (pooled OR = 0.61; 95% CI = 0.28, 1.35); I2 = 66.3%, P = 0.031), however, this change was not significant.

Effect of sodium-restricted diets on the risk of gestational hypertension or/and preeclampsia

Obtained results of the random effect model on four studies indicated that sodium-restricted interventions did not have a significant effect on the risk of GH (OR = 0.99; 95% CI = 0.68, 1.45); I^2 = 0%; P = 0.520) with no significant

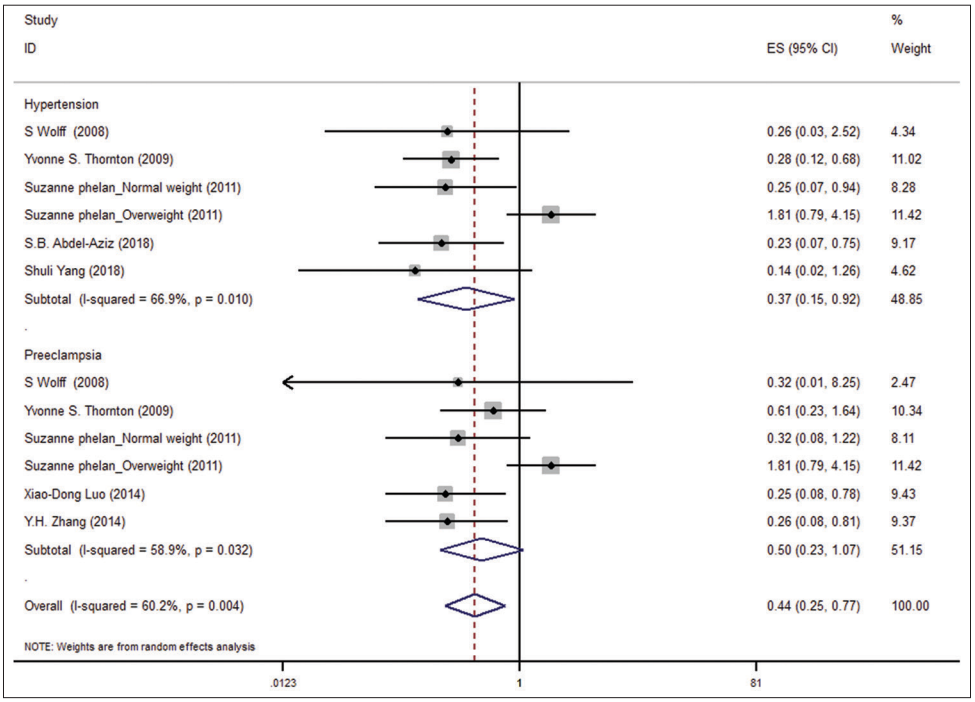

Figure 2: Effect of managed nutritional programs on the risk of GH or/and PE. GH = Gestational hypertension; PE = Preeclampsia

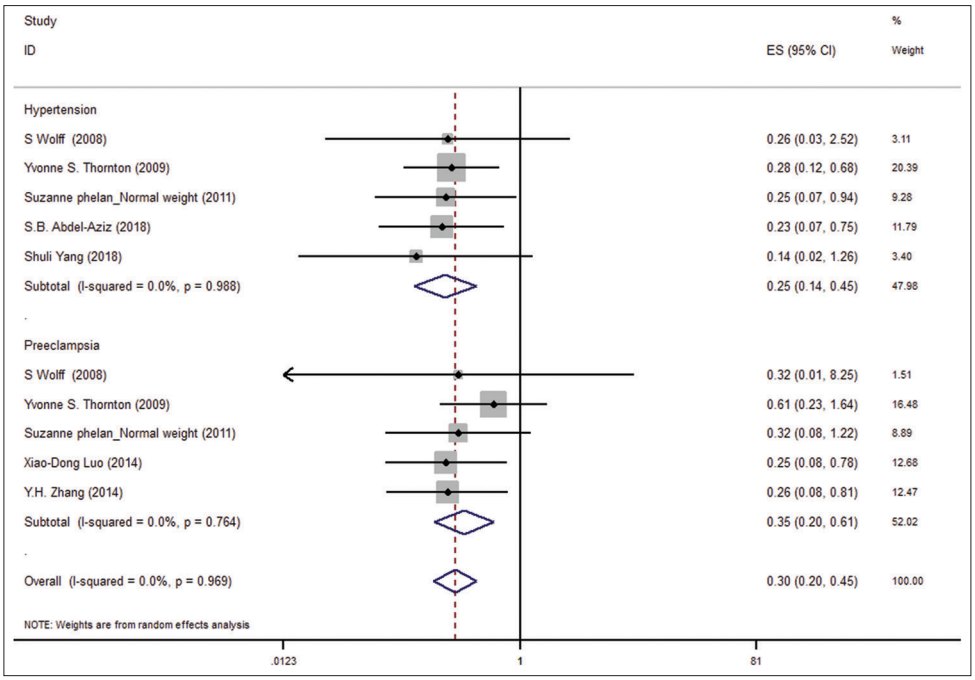

Figure 3: Effect of managed nutritional interventions on risk of GH or/and PE after excluding Phelan *et al*.'s trial performed in over-weight women. GH = Gestational hypertension; PE = Preeclampsia

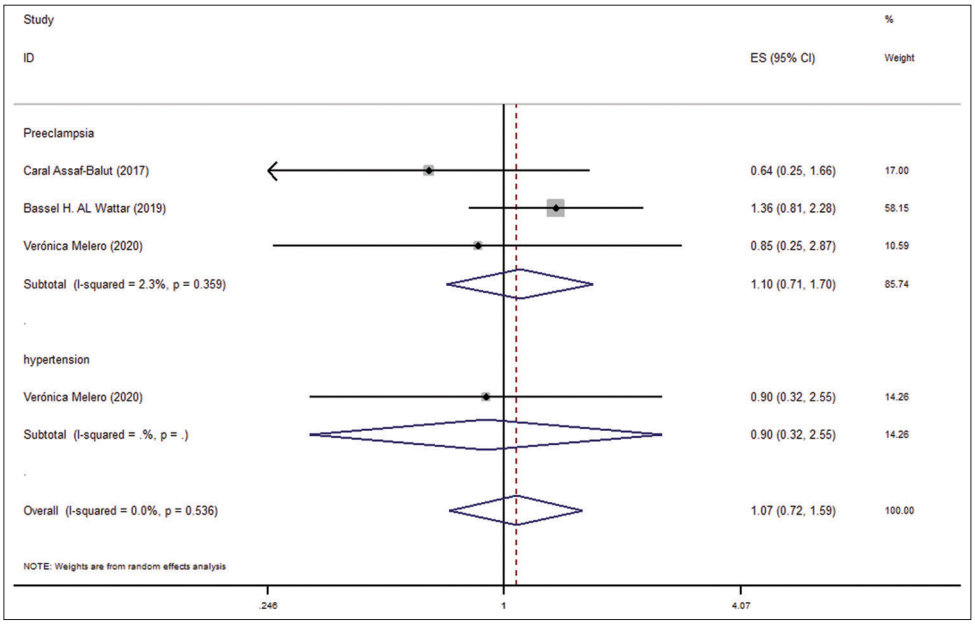

Figure 4: Effect of the Mediterranean-style diet on risk of GH or/and PE. GH = Gestational hypertension; PE = Preeclampsia

heterogeneity ($P > 0.05$). In addition, it was not significant with total risk of GH and PE (OR = 0.96; 95% CI = 0.65, 1.42); $I^2 = 1.7\%$; $P = 0.397$) [Figure 5]. The P value for Egger's test was 0.279 which showed no noticeable publication bias among these studies. Only one study[21] reported incidence of PE which were not enough for subgroup analysis. Meta-regression specified that there was no significant association between maternal age, BMI, GWG, and start time of intervention with the effect of dietary sodium restriction intervention on risks of GH or/and PE in pregnant women ($P > 0.05$). Sensitivity analysis showed that after omitting Knuist *et al*.'s study,[23] the pooled OR decreased and heterogeneity increased, but they were not significant (OR = 0.78; 95% CI = 0.27, 2.24); $I^2 = 26.1\%$; $P = 0.255$).

DISCUSSION

This meta-analysis showed that managed nutritional interventions were effective on total risks of GH, also GH,

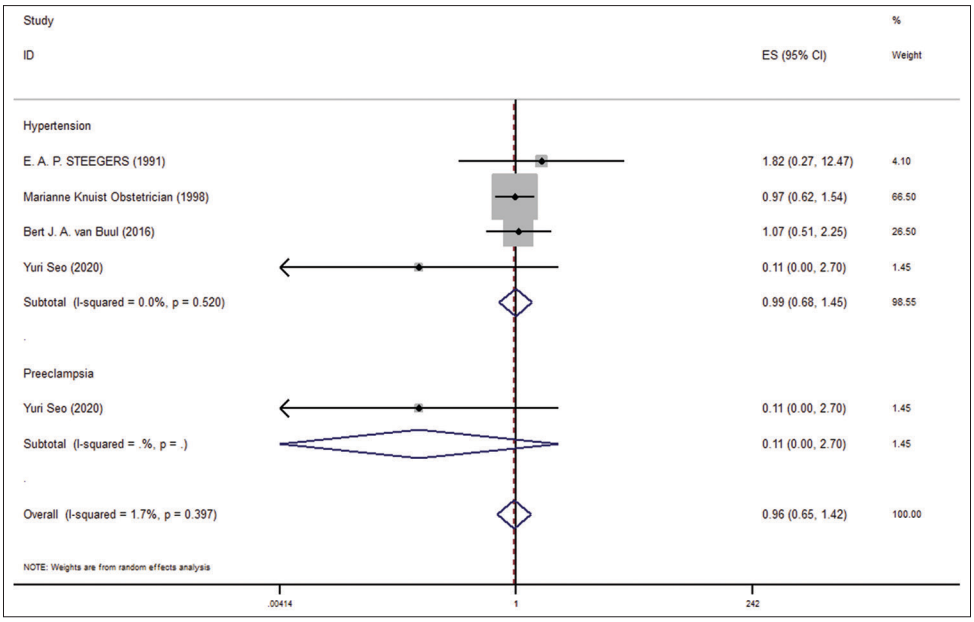

Figure 5: Effect of sodium-restricted intervention on risk of GH or/and PE. GH = Gestational hypertension; PE = Preeclampsia

and PE in healthy pregnancy, while Mediterranean-style and sodium-restricted diets were not effective on risks of GH or/and PE.

Effect of educational and nutritional interventions (managed nutritional programs) on risk of gestational hypertension or/and preeclampsia

Meta-analysis on managed nutritional interventions was effective on total risk of GH and PE, and also on GH. We did not find another meta-analysis with positive effects of dietary interventions on GH and/or PE; but one trial showed that nutritional intervention in accordance with guideline decreased SBPs and DBPs significantly in participants with GH.[30] Alike, another revealed that a supervised nutritional trial decreased levels of SDB and DBP.[31] Researchers showed that inappropriate nutrition increases oxidative stress in women, which can reduce telomere length and consequently, its aggregations and lead to enhanced cell aging, tissue senescence, and ultimately PE.[32] Therefore, nutritional interventions to make balance and diversity may be effective on GH or/and PE. In opposite, one meta-analysis assessing the effect of 11 and 14 nutritional interventions on the risk of PE and HDPs, did not show significant impacts in overweight and obese pregnant women.[33] As after excluding Phelan *et al*.'s trial performed in overweight women,[16] the impact of managed dietary interventions on the incidence of PE and/or GH was significant with no heterogeneity. However, further clinical trials and meta-analyses are required for better judgment.[33]

Effect of the Mediterranean diet

Data analysis on RCTs did not reduce the total risk of PE and GH, and also PE, with a low heterogeneity. Similarly, Zhang *et al*.'s meta-analysis showed that the Mediterranean diet was not effective on gestational diabetes mellitus.[34] Likewise, moderate and high adherence to the Mediterranean diet was not associated with the prevalence of hypertension among 9,408 adults; however, mean levels of SBP and DBP decreased after 6 years of follow-up, and it seems that the Mediterranean diet can reduce age-related changes in BP.[35] As some researchers reported that the intakes and plasma values of Vitamins C, E are higher in people who consume Mediterranean diet, which these may relate to lower incidence of PE.[36] Apparently, the consumption of Mediterranean-style diet for a long time has useful effects on rate of HDPs which needs further surveys.

Effect of sodium-restriction interventions

Our meta-analysis did not demonstrate a significant effect on GH or/and PE after sodium restriction diets. Likewise, a Cochrane review on 603 women did not observe a significant correlation after salt-restricted diet.[37] Furthermore, a historical review concluded that salt restriction did not prevent gestational hypertension.[38] One observational study reported that the highest sodium intake compared to the lowest intake was correlated with a higher risk of gestational hypertension and PE.[39] It seems that sodium intake control is necessary and useful, but its restriction did not decrease incidence of GH and PE.

In continuation of the discussion, we also reviewed the evidence from RCTs regarding the impact of nutritional interventions which were not eligible in the meta-analysis [Table 1].

Effect of mushroom

Sun *et al*. reported that consumption of 100 g of white button mushroom per day, from before pregnancy to the 20th week of gestation, reduced the incidence of GH and PE compared to the control group.[8] One particular bioactive compound identified in mushrooms is L-ergothioneine, a water-soluble thiol and an unusual antioxidant, i.e., resistant to autoxidation and may be practical for the prevention and treatment of PE. Furthermore, as a special physiological transporter, it can accumulate in organs with high oxidative stress, which is useful for the treatment of PE. In addition, L-ergothioneine encodes SLC22A4, suggesting its association with various inflammatory and metabolic conditions.[14,40] Furthermore, another study showed the relationship between the consumption of 100 g of white button mushrooms and the increase of antioxidant biomarkers in medical syndrome.[41]

Effect of a cholesterol-lowering diet

In one study, healthy nonsmoking white women were recommended to eat a diet that included fish, low-fat dairy products and meat, oils, fruits, vegetables, whole grains, and legumes from 17 to 20 weeks of pregnancy. As a result, the amounts of total cholesterol and LDL cholesterol decreased significantly, but there was no significant effect on GH and PE.[11] In contrast, a meta-analysis of observational studies showed that lower levels of HDL cholesterol as well as higher levels of total cholesterol, non-HDL cholesterol, and triglycerides during pregnancy, were associated with the risk of PE in the third trimester.[42] In addition, comparative study showed that a significant enhancement in plasma total cholesterol, triglycerides, low-density lipoprotein cholesterol, very low-density lipoprotein cholesterol, and also a significant decline in high-density lipoprotein cholesterol could begin endothelial dysfunction and appearance of PE. It seems that early management of an altered lipid profile has a potential role to control PE.[43]

Effect of dietary calcium intake

Two trials instructed pregnant women to increase their dietary calcium intake to assess the incidence of PE/GH or both. In one, adolescent mothers were randomly assigned to receive calcium-supplemented orange juice or dairy products, both of which (intervention group) contained 1200 mg of calcium compared to the control group. Mean SBPs and DBPs did not change after two interventions.[28] Pregnant women in the second study were trained to increase calcium intake through daily meals, considering that the target calcium intake was 1200 mg/day, and it was estimated by recalling the 24-h diet. The result was that blood pressure changes in the intervention group were 62% lower than in the control (adjusted OR = 0.38;95% CI: 0.19,051).[29] We did not find any other trial, but a meta-analysis of observational studies showed that higher unadjusted energy intake and lower unadjusted intakes of magnesium and calcium were associated with HDPs.[13] Similarly, a few meta-analyses showed that calcium supplementation is an effective strategy for the prevention of PE, especially in pregnant women who are at risk of HDPs because of obesity, ethnicity, age, gender, and low value of calcium intake.[44-46] However, further trials are needed to discover the ideal dose.[46] Recently, in 415 healthy pregnant women with adequate Vitamin D and calcium intake, no significant association was observed between intakes of both Vitamin D and calcium and gestational blood pressure[47] which confirms the above findings and may give an explanation for different results regarding the relationship between calcium intake and blood pressure. One review concluded that the association between calcium intake and blood pressure is related to calciotropic hormones as blood pressure regulators. Parathyroid hormone (PTH) enhances cytosolic calcium levels and vascular reactivity and consequently blood pressure. Low-calcium consumption stimulates the synthesis of calcitriol directly or by PTH, and calcitriol causes an increase in intracellular calcium in vascular muscle cells. In addition, low-calcium intake causes the release of renin and the increase of angiotensin II and aldosterone by PTH.[48]

Effect of dietary approaches to stop hypertension diet

A calorie-appropriate DASH diet without sodium restriction did not change the incidence of GH and PE.[27] We did not find any intervention of DASH diet in healthy pregnant women, but one observational study reported that higher scores of following of DASH diet were not correlated to the risk of HDPs.[49] Another RCT on 151 women with a history of PE within five years did not change weight and BP.[50] Oppositely, Jiang *et al*.'s study showed that DASH diet in pregnancy with chronic hypertension or GH decreased the incidence of PE versus the control.[26] In addition, a meta-analysis of RCTs in participants with or without comorbidities, including medial problems, showed that dash diet decreased systolic and diastolic BP, and these changes in both variables were greater in higher baseline BP or BMI.[51] Another RCT showed that consumption of DASH diet for 4 weeks in participants with GDM was effective on systolic but not DBP, too.[52] The effect of DASH diet on GH/PE appears to be weaker in healthy pregnancy, which is similar to the finding of others in the nonpregnant state.[51] Likewise, another meta-analysis showed that following of DASH diet in pregnant women with cardiometabolic disease decreased the incidence of PE.[53]

Effect of a low-glycemic diet

Rhodes *et al*.'s study in overweight and obese pregnant women compared low-glycemic index (GI) diet with a low-fat diet. The low-GL diet (including 45% carbohydrate, 35% fat, and 20% protein) was considered to be moderately

decreasing total carbohydrate and substituting higher glycemic index carbohydrates with lower GI carbohydrates. In low-fat diet, participants received low-fat, low saturated fat, and high complex carbohydrate without consideration of glycemic index, which naturally such diets are moderately high in GL and include 55% carbohydrate, 25% fat, and 20% protein.[25] Mean changes of SBPs and DBPs were not significantly different between the two groups, and obtained results were consistent with a meta-analysis of RCTs that compared low-carbohydrate diets with low-fat diets in adults and both diets decreased blood pressure.[54] In addition, a meta-analysis comparing the influences of low-carbohydrate regime with low-fat regime on metabolic risk factors demonstrated that both diets were alike in decreasing adult BPs. Another study in overweight and obese subjects demonstrated that both diets reduced SBP and DBP by 10 and 5 mmHg, respectively.[55]

We performed a complete search for RCTs that reported the effects of nutritional interventions (only nutritional interventions and not a combination of nutritional interventions with other interventions) on gestational hypertension or/and PE in healthy pregnant women. In addition, we meta-analyzed similar trials categorized into different intervention groups. Moreover, important confounding variable such as age, GWG, pre-pregnancy BMI, and starting trimester of intervention were extracted for meta-regression. Finally, we reviewed seven trials which their results could be useful. The present study was restricted by small number of RCTs. Furthermore, the exclusion of studies not written in English may have restricted the number of studies included in this review.

CONCLUSION

The present meta-analysis showed that Mediterranean-style diets and sodium-restriction interventions did not decrease the incidence of GH or/and PE in healthy pregnancies; however, managed nutritional programs reduced the risk of GH, the total incidence of GH and PE, but not PE

Financial support and sponsorship
Nil.

Conflicts of interest
There are no conflicts of interest.

REFERENCES

1. Zainul Rashid MR, Lim JF, Nawawi NH, Luqman M, Zolkeplai MF, Rangkuty HS, *et al*. A pilot study to determine whether progestogen supplementation using dydrogesterone during the first trimester will reduce the incidence of gestational hypertension in primigravidae. Gynecol Endocrinol 2014;30:217-20.
2. Walker RL, Hemmelgarn B, Quan H. Incidence of gestational hypertension in the Calgary Health Region from 1995 to 2004. Can J Cardiol 2009;25:e284-7.
3. Amaral LM, Wallace K, Owens M, LaMarca B. Pathophysiology and current clinical management of preeclampsia. Curr Hypertens Rep 2017;19:61.
4. Antwi E, Amoakoh-Coleman M, Vieira DL, Madhavaram S, Koram KA, Grobbee DE, *et al*. Systematic review of prediction models for gestational hypertension and preeclampsia. PLoS One 2020;15:e0230955.
5. Khoigani MG, Paknahad Z, Mardanian F. The relationship between nutrients intake and preeclampsia in pregnant women. Journal of Research in Medical Sciences 2012;17(Spec 2):S217.
6. Cao Y, Liu Y, Zhao X, Duan D, Dou W, Fu W, *et al*. Adherence to a dietary approaches to stop hypertension (DASH)-style diet in relation to preeclampsia: A case-control study. Sci Rep 2020;10:9078.
7. Abdel-Aziz SB, Hegazy IS, Mohamed DA, Abu El Kasem MM, Hagag SS. Effect of dietary counseling on preventing excessive weight gain during pregnancy. Public Health 2018;154:172-81.
8. Sun L, Niu Z. A mushroom diet reduced the risk of pregnancy-induced hypertension and macrosomia: a randomized clinical trial. Food Nutr Res 2020;64.
9. Zhang YH. Comprehensive effect assessment of medical nutrition guidance during pregnancy towards the health of mothers and children. Clin Exp Obstet Gynecol 2015;42:644-8.
10. van Buul BJ, Steegers EA, van der Maten GD, Delemarre FM, Jongsma HW, Oosterbaan HP, *et al*. Dietary sodium restriction does not prevent gestational hypertension: A dutch two-center randomized trial. Hypertens Pregnancy 1997;16:335-46.
11. Khoury J, Henriksen T, Christophersen B, Tonstad S. Effect of a cholesterol-lowering diet on maternal, cord, and neonatal lipids, and pregnancy outcome: A randomized clinical trial. Am J Obstet Gynecol 2005;193:1292-301.
12. Thornton YS, Smarkola C, Kopacz SM, Ishoof SB. Perinatal outcomes in nutritionally monitored obese pregnant women: A randomized clinical trial. J Natl Med Assoc 2009;101:569-77.
13. Schoenaker DA, Soedamah-Muthu SS, Mishra GD. The association between dietary factors and gestational hypertension and pre-eclampsia: A systematic review and meta-analysis of observational studies. BMC Med 2014;12:157.
14. Kibret KT, Chojenta C, Gresham E, Tegegne TK, Loxton D. Maternal dietary patterns and risk of adverse pregnancy (hypertensive disorders of pregnancy and gestational diabetes mellitus) and birth (preterm birth and low birth weight) outcomes: a systematic review and meta-analysis. Public health nutrition. 2019;22:506-20.
15. Wolff S, Legarth J, Vangsgaard K, Toubro S, Astrup A. A randomized trial of the effects of dietary counseling on gestational weight gain and glucose metabolism in obese pregnant women. Int J Obes (Lond) 2008;32:495-501.
16. Phelan S, Phipps MG, Abrams B, Darroch F, Schaffner A, Wing RR. Randomized trial of a behavioral intervention to prevent excessive gestational weight gain: The Fit for Delivery Study. Am J Clin Nutr 2011;93:772-9.
17. Luo XD, Dong X, Zhou J. Effects of nutritional management intervention on gestational weight gain and perinatal outcome. Saudi Med J 2014;35:1267-70.
18. Yang S, Si L, Jia Y, Jian W, Yu Q, Wang M, *et al*. Effects of nutrition health education and targeted nutrition guidance on maternal nutritional status and maternal and infant outcomes. Methods 2018;5:7.
19. Assaf-Balut C, García de la Torre N, Durán A, Fuentes M, Bordiú E, Del Valle L, *et al*. A Mediterranean diet with additional extra virgin olive oil and pistachios reduces the incidence of gestational

diabetes mellitus (GDM): A randomized controlled trial: The St. Carlos GDM prevention study. PLoS One 2017;12:e0185873.
20. H Al Wattar B, Dodds J, Placzek A, Beresford L, Spyreli E, Moore A, *et al.* Mediterranean-style diet in pregnant women with metabolic risk factors (ESTEEM): A pragmatic multicentre randomised trial. PLoS Med 2019;16:e1002857.
21. Melero V, García de la Torre N, Assaf-Balut C, Jiménez I, Del Valle L, Durán A, *et al.* Effect of a Mediterranean Diet-Based Nutritional Intervention on the Risk of Developing Gestational Diabetes Mellitus and Other Maternal-Fetal Adverse Events in Hispanic Women Residents in Spain. Nutrients 2020;12:3505.
22. Seo Y, Jeong YS, Koo KA, Yang JI, Park YK. Maternal nutrition intervention focused on the adjustment of salt and sugar intake can improve pregnancy outcomes. Food Science & Nutrition 2020;8:3900-11.
23. Knuist M, Bonsel GJ, Zondervan HA, Treffers PE. Low sodium diet and pregnancy-induced hypertension: a multi-centre randomised controlled trial. BJOG: An International Journal of Obstetrics & Gynaecology 1998;105:430-4.
24. Steegers EA, Van Lakwijk HP, Jongsma HW, Fast JH, De Boo T, Eskes TK, *et al.* (Patho) physiological implications of chronic dietary sodium restriction during pregnancy; a longitudinal prospective randomized study. BJOG: An International Journal of Obstetrics & Gynaecology 1991;98:980-7.
25. Rhodes ET, Pawlak DB, Takoudes TC, Ebbeling CB, Feldman HA, Lovesky MM, *et al.* Effects of a low–glycemic load diet in overweight and obese pregnant women: a pilot randomized controlled trial. The American journal of clinical nutrition 2010;92:1306-15.
26. Jiang F, Li Y, Xu P, Li J, Chen X, Yu H, *et al*. The efficacy of the Dietary Approaches to Stop Hypertension diet with respect to improving pregnancy outcomes in women with hypertensive disorders. Journal of Human Nutrition and Dietetics 2019;32:713-8.
27. Vesco K, Leo M, Gillman M, King J, McEvoy C, Karanjaa N, *et al*. LB 2: Impact of a weight management intervention on pregnancy outcomes among obese women: The Healthy Moms Trial. American Journal of Obstetrics & Gynecology. 2013;208:S352.
28. Chan GM, McElligott K, McNaught T, Gill G. Effects of dietary calcium intervention on adolescent mothers and newborns: a randomized controlled trial. Obstetrics & Gynecology. 2006;108(3 Part 1):565-71.
29. Abrha MW, Abarha A, Gebretsadik A, Ayele B, Gebretensae H, Gebre-egziabher E, *et al*. Examining the Effect of Education on Dietary Calcium Intake in Reducing Blood Pressure Variability Among Pregnant Mothers in Tigray Region, Northern Ethiopia, Two Arm, Randomized Control Trail Parallel Design. 2020.
30. Pan Y, Yu Y, Liu R. Application of Prenatal Health Education and Nutrition Intervention in Patients with Pregnancy-induced Hypertension. In 8th International Conference on Education, Management, Information and Management Society (EMIM 2018) 2018. (pp. 13-17).
31. Zerón HM, Flores AP, Chávez AA, Alanís AG, Ferreyra MD, Benítez JG, *et al*. Pregnancy weight gain limitation by a supervised nutritional program influences placental NF-κB/IKK complex expression and oxidative stress. Oman medical journal 2013;28(3):167.
32. Godhamgaonkar AA, Sundrani DP, Joshi SR. Role of maternal nutrition and oxidative stress in placental telomere attrition in women with preeclampsia. Hypertension in Pregnancy. 2021;40:63-74.
33. Syngelaki A, Sequeira Campos M, Roberge S, Andrade W, Nicolaides KH. Diet and exercise for preeclampsia prevention in overweight and obese pregnant women: Systematic review and meta-analysis. J Matern Fetal Neonatal Med 2019;32:3495-501.
34. Zhang Y, Xia M, Weng S, Wang C, Yuan P, Tang S. Effect of Mediterranean diet for pregnant women: A meta-analysis of randomized controlled trials. J Matern Fetal Neonatal Med 2022;35:4824-9.
35. Núñez-Córdoba JM, Valencia-Serrano F, Toledo E, Alonso A, Martínez-González MA. The Mediterranean diet and incidence of hypertension: The Seguimiento Universidad de Navarra (SUN) study. Am J Epidemiol 2009;169:339-46.
36. Llurba E, Gratacós E, Martín-Gallán P, Cabero L, Dominguez C. A comprehensive study of oxidative stress and antioxidant status in preeclampsia and normal pregnancy. Free Radic Biol Med 2004;37:557-70.
37. Duley L, Henderson-Smart D, Meher S. Altered dietary salt for preventing pre-eclampsia, and its complications. Cochrane Database Syst Rev 2005;(4):CD005548.
38. Steegers EA, Eskes TK, Jongsma HW, Hein PR. Dietary sodium restriction during pregnancy; a historical review. Eur J Obstet Gynecol Reprod Biol 1991;40:83-90.
39. Arvizu M, Bjerregaard AA, Madsen MT, Granström C, Halldorsson TI, Olsen SF, *et al.* Sodium intake during pregnancy, but not other diet recommendations aimed at preventing cardiovascular disease, is positively related to risk of hypertensive disorders of pregnancy. J Nutr 2020;150:159-66.
40. Kerley RN, McCarthy C, Kell DB, Kenny LC. The potential therapeutic effects of ergothioneine in pre-eclampsia. Free Radic Biol Med 2018;117:145-57.
41. Calvo MS, Mehrotra A, Beelman RB, Nadkarni G, Wang L, Cai W, Goh BC, Kalaras MD, Uribarri J. A Retrospective Study in Adults with Metabolic Syndrome: Diabetic Risk Factor Response to Daily Consumption of Agaricus bisporus (White Button Mushrooms). Plant Foods Hum Nutr 2016;71(3):245-51.
42. Spracklen CN, Smith CJ, Saftlas AF, Robinson JG, Ryckman KK. Maternal hyperlipidemia and the risk of preeclampsia: A meta-analysis. Am J Epidemiol 2014;180:346-58.
43. Yadav S, Agrawal M, Hariharan C, Dewani D, Vadera K, Krishna N. A comparative study of serum lipid profile of women with preeclampsia and normotensive pregnancy. J Datta Meghe Inst Med Sci Univ 2018;13:83.
44. Patrelli TS, Dall'asta A, Gizzo S, Pedrazzi G, Piantelli G, Jasonni VM, *et al.* Calcium supplementation and prevention of preeclampsia: A meta-analysis. J Matern Fetal Neonatal Med 2012;25:2570-4.
45. Imdad A, Jabeen A, Bhutta ZA. Role of calcium supplementation during pregnancy in reducing risk of developing gestational hypertensive disorders: A meta-analysis of studies from developing countries. BMC Public Health 2011;11 Suppl 3:S18.
46. Sun X, Li H, He X, Li M, Yan P, Xun Y, *et al*. The association between calcium supplement and preeclampsia and gestational hypertension: A systematic review and meta-analysis of randomized trials. Hypertens Pregnancy 2019;38:129-39.
47. Forde H, Crowley RK, McKenna MJ, Kilbane MT, Conway M, McDonnell CM, *et al*. No effect of calcium and vitamin D intake on maternal blood pressure in a healthy pregnant population. Eur J Obstet Gynecol Reprod Biol 2021;264:8-14.
48. Villa-Etchegoyen C, Lombarte M, Matamoros N, Belizán JM, Cormick G. Mechanisms involved in the relationship between low calcium intake and high blood pressure. Nutrients 2019;11:E1112.
49. Arvizu M, Bjerregaard AA, Madsen MT, Granström C, Halldorsson TI, Olsen SF, *et al.* Sodium intake during pregnancy, but not other diet recommendations aimed at preventing cardiovascular disease, is positively related to risk of hypertensive disorders of pregnancy. J Nutr 2020;150:159-66.
50. Rich-Edwards JW, Stuart JJ, Skurnik G, Roche AT, Tsigas E, Fitzmaurice GM, *et al*. Randomized trial to reduce cardiovascular risk

in women with recent preeclampsia. J Womens Health (Larchmt) 2019;28:1493-504.
51. Siervo M, Lara J, Chowdhury S, Ashor A, Oggioni C, Mathers JC. Effects of the Dietary Approach to Stop Hypertension (DASH) diet on cardiovascular risk factors: A systematic review and meta-analysis. Br J Nutr 2015;113:1-15.
52. Asemi Z, Tabassi Z, Samimi M, Fahiminejad T, Esmaillzadeh A. Favourable effects of the Dietary Approaches to Stop Hypertension diet on glucose tolerance and lipid profiles in gestational diabetes: A randomised clinical trial. Br J Nutr 2013;109:2024-30.
53. Li S, Gan Y, Chen M, Wang M, Wang X, O Santos H, *et al*. Effects of the Dietary Approaches to Stop Hypertension (DASH) on pregnancy/neonatal outcomes and maternal glycemic control: A systematic review and meta-analysis of randomized clinical trials. Complement Ther Med 2020;54:102551.
54. Hu T, Mills KT, Yao L, Demanelis K, Eloustaz M, Yancy WS Jr., *et al*. Effects of low-carbohydrate diets versus low-fat diets on metabolic risk factors: A meta-analysis of randomized controlled clinical trials. Am J Epidemiol 2012;176 Suppl 7:S44-54.
55. Meckling KA, O'Sullivan C, Saari D. Comparison of a low-fat diet to a low-carbohydrate diet on weight loss, body composition, and risk factors for diabetes and cardiovascular disease in free-living, overweight men and women. J Clin Endocrinol Metab 2004;89:2717-23.