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Review Article

Effects of dietary interventions on pregnancy outcomes: a systematic review and meta-analysis

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Abstract

Dietary intake during pregnancy influences maternal health. Poor dietary practices during pregnancy have been linked to maternal complications. The objective was to determine the effect of dietary intervention before or during pregnancy on pregnancy outcomes. A systematic review was conducted without date restrictions. Randomised controlled trials (RCTs) evaluating whole diet or dietary components and pregnancy outcomes were included. Two authors independently identified papers for inclusion and assessed methodological quality. Meta-analysis was conducted separately for each outcome using random effects models. Results were reported by type of dietary intervention: (1) counselling; (2) food and fortified food products; or (3) combination (counselling + food); and collectively for all dietary interventions. Results were further grouped by trimester when the intervention commenced, nutrient of interest, country income and body mass index. Of 2326 screened abstracts, a total of 28 RCTs were included in this review. Dietary counselling during pregnancy was effective in reducing systolic [standardised mean difference (SMD) −0.26, 95% confidence interval (CI) −0.45 to −0.07; $P < 0.001$] and diastolic blood pressure (SMD −0.57, 95% CI −0.75 to −0.38; $P < 0.001$). Macronutrient dietary interventions were effective in reducing the incidence of preterm delivery (SMD −0.19, 95% CI −0.34 to −0.04; $P = 0.01$). No effects were seen for other outcomes. Dietary interventions showed some small, but significant differences in pregnancy outcomes including a reduction in the incidence of preterm birth. Further high-quality RCTs, investigating micronutrient provision from food, and combination dietary intervention, are required to identify maternal diet intakes that optimise pregnancy outcomes.

Keywords: diet, pregnancy, randomised controlled trial, systematic review, meta-analysis

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Introduction

The association between early life nutrition and long-term health has been of interest for decades (Bhutta 2013). There is an abundance of evidence to suggest that when a woman has a good state of health and nutrition prior to and during pregnancy, there is a greater chance of a successful pregnancy and birth outcome (Goldberg 2002; Erick 2008; Ritchie & King 2008; Nichols-Richardson 2011). Inadequate nutrition during this time, particularly the first trimester, impairs fetal growth (Antal *et al*. 1997; Derbyshire *et al*. 2006; Northstone *et al*. 2008) and has long-term negative consequences for the mother and the developing fetus (Godfrey & Robinson 1998; Moore *et al*. 2004; Northstone *et al*. 2008; Derbyshire *et al*. 2009).

Gestational diabetes mellitus (GDM) and pregnancy hypertensive disorders are among the most common complications of pregnancy (Sibai 2003; Zhang *et al*. 2006) affecting 2–5% (Gilmartin *et al*. 2008), and 10% (Roberts *et al*. 2011) of all pregnancies, respectively. Pregnancies complicated by these metabolic conditions are associated with adverse maternal, fetal and neonatal outcomes (Brown *et al*. 2000; Östlund *et al*. 2003; Roberts *et al*. 2003, 2005). GDM is associated with an increased risk of pregnancy hypertensive disorders and caesarean section (American Diabetes Association 2003), while increasing the risk of neonatal outcomes: intrauterine fetal death, preterm delivery and fetal macrosomia (American Diabetes Association 2003). In the United States, pregnancy hypertensive disorders, are one of the leading causes of maternal death (Peters & Flack 2004), associated with an increased risk of placental abruption, ante- and post-partum haemorrhage, and acute renal or hepatic failure for the mother (National High Blood Pressure Education Program Working Group on High Blood Pressure in Pregnancy 2000). Pregnancy hypertensive disorders are also associated with preterm birth, intrauterine growth restriction and perinatal death for the neonate (Brown *et al*. 2000, Roberts *et al*. 2003, National High Blood Pressure Education Program Working Group on High Blood Pressure in Pregnancy 2000, Lowe *et al*. 2009). Maternal diet is one of the key risk factors for developing pregnancy hypertensive disorders and GDM and therefore may be a key preventative strategy on these and other pregnancy outcomes (King 2006). However, little is known about the effectiveness of dietary intervention during pregnancy (Roseboom *et al*. 2011).

The objective of this study was to synthesise the best of the available evidence by conducting a systematic review and meta-analysis, to determine whether dietary interventions have any effect on pregnancy outcome. Dietary interventions could consist of dietary counselling, food and/or fortified food products, or a combination of both, administered either before or during pregnancy.

Materials and methods

The review protocol was developed using the Cochrane Handbook for Systematic Reviews of Interventions (The Cochrane Collaboration 2011) and Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) for reporting the methods and outcomes (Liberati *et al*. 2009; Moher *et al*. 2009).

Eligibility criteria

Table 1 highlights the inclusion and exclusion criteria for the selection of publications. Any publication identified in the search reporting one or more pregnancy, neonatal or infant outcomes was considered. At least three randomised controlled trials (RCTs) needed to be identified for each outcome for inclusion in the results to allow for meta-analysis. The outcome definitions were those used by the authors; however, where multiple definitions existed between publications, the term was used broadly. For example, preterm delivery included those classified as <36 weeks gestation (de Groot *et al*. 2004), <37 weeks (Mora *et al*. 1978; Rush *et al*. 1980; McDonald *et al*. 1981; Kafatos *et al*. 1989; Van Buul *et al*. 1997; Smuts *et al*. 2003b; Khoury *et al*. 2005; Bech *et al*. 2007; Aaltonen *et al*. 2008; Thornton *et al*. 2009; Vinter *et al*. 2011) and unspecified (Mardones-Santander *et al*. 1988; Briley *et al*. 2002; Smuts *et al*. 2003a; Quinlivan *et al*. 2011). Nutrient deficiencies were excluded from

Key messages

- Dietary counselling during pregnancy reduces maternal blood pressure, but not hypertensive disorders.
- Dietary interventions focused on modifying macronutrient intakes during pregnancy reduces the incidence of preterm delivery.
- Further research is needed to elucidate the role of maternal diet, particularly micronutrient provision and combination dietary interventions to optimise pregnancy outcomes.

Table 1. Criteria for the selection of studies

Inclusion	Exclusion
Studies that reported any pregnancy, neonatal or infant outcomes in preconception or pregnant women, any age, weight or body mass index, without date limits.	Health conditions that may influence dietary intake (i.e. unrepresentative sample such as gestational diabetes).
At least three randomised controlled trials per outcome.	Studies published in languages other than English.
Any healthy, human population.	Studies in animals.
Randomised or pseudo-randomised controlled trials on dietary interventions, including counselling, food and/or both provided by any health professional.	Case study, editorial, conference proceeding.
Any intensity, frequency or timing of intervention.	Studies on gestational weight gain.
Positive or neutral methodological quality.*	Trials solely on nutrient supplementation (i.e. tablet form; no macronutrient change).
	Multiple births.
	Duplicate populations (the data reporting the smaller number of participants for the same pregnancy outcome was excluded).
	Negative methodological quality.*

*American Dietetic Association Quality Criteria Checklist for Primary Research (see Academy of Nutrition and Dietetics 2012).

this review as they were considered risk factors rather than clinical outcomes. This review focuses on dietary intake from food, rather than nutritional supplement use. Publications that targeted the whole diet, single food groups, individual food and fortified foods were included. Fortified foods included the provision of one or more foods that has essential vitamins and/or minerals added to enhance the foods nutritional content; for example docosahexaenoic acid (DHA) added to a cereal-based bar to increase the omega 3 fatty acid content. Fortified foods modify both the macro- (carbohydrate, protein, fat) and micronutrient (vitamins and minerals) content of the maternal diet. Supplement-only trials including the provision of a pill, capsule or tablet containing vitamins, minerals or amino acids were excluded. The types of dietary interventions included dietary counselling, modifying or providing food and fortified food products, or a combination of both.

Search strategy

A research librarian (DB) guided the systematic search for publications in December 2011. The search was conducted without date limits, using 10 electronic databases: EMBASE; Pre-Medline; MEDLINE; Proquest; Web of Science; CINAHL; Scopus; The Cochrane Library; Mosby Index; and Maternity and Infant Care. The following MeSH terms, words and combinations of words were searched: pregnancy; diet OR food OR beverage OR nutrient OR macro-nutrient OR micro-nutrient; and randomised controlled trial OR randomized controlled trial. Keywords were searched as free text in the title, abstract, or topic and combined using the Boolean operator 'AND'. Our MeSH terms were intentionally broad so as to capture as many dietary interventions and outcomes as possible. Preconception was defined as up to 3 months before pregnancy; however, we did not explicitly include 'preconception' as a search term in isolation from the term 'pregnancy'. Limits included English language and humans. Additional publications were identified from the reference lists of included papers. All primary trials included in systematic reviews and meta-analyses were independently assessed for eligibility. Outcomes were divided into two groups: pregnancy (this paper), and neonatal and infant (presented elsewhere).

Selection process

All records identified were first assessed for eligibility based on the information contained in the title and abstract, by two independent reviewers (EG and AH), as recommended by the PRISMA guidelines for systematic reviews of randomised trials (Liberati *et al*.

2009). When multiple publications were available for the same trial, the publication reporting the greatest number of participants for each outcome was selected. The full text of all publications that appeared to meet the eligibility screening (Table 1) was retrieved and subjected to a second assessment for relevance (EG and AH). Any discrepancy in assessment between reviewers was resolved through discussion. Selected full texts were then assessed for methodological quality by two independent reviewers (EG and SD) using the Quality Criteria Checklist for Primary Research in the American Dietetic Association Evidence Analysis Manual (Academy of Nutrition and Dietetics 2012). Any discrepancies were discussed. The Quality Criteria Checklist includes 10 structured validity questions that address scientific quality and soundness, including bias, confounding and the appropriateness of the interventions and measures. The Quality Criteria Checklist enabled a systematic and objective rating to be given to each publication. The highest methodological quality papers were designated 'Positive', meeting most of the validity criteria including all priority criteria (Academy of Nutrition and Dietetics 2012). 'Neutral' quality publications met most of the validity criteria, but failed one or more of the four priority criteria, indicating the study was not exceptionally strong (Academy of Nutrition and Dietetics 2012). 'Negative' publications failed to meet six or more of the validity criteria (Academy of Nutrition and Dietetics 2012) and were excluded from the results.

Data extraction

One reviewer (EG) extracted relevant data from all included publications, with a second independent reviewer (AH/JL) extracting data from approximately half to ensure accuracy. The following variables were data extracted: study design, aim, quality, participant characteristics, intervention type, dietary modification, assessment, compliance and outcome. For the meta-analysis, one reviewer (EG) extracted the data from each publication into an Excel spread sheet, verified by a second reviewer (AB) for the following variables: author, year, outcome, type of intervention, trimester when the intervention commenced, nutrient of interest (macro- or micronutrient), body mass index (BMI; underweight/nutritional risk, overweight/obese, or no restriction), country income (high or low), quality rating (positive or neutral), participant numbers per group and by outcome, mean, standard deviation, 95% confidence interval (CI), median, range and odds ratio where possible for each outcome. Trials were divided into either macro- or micronutrient, depending on their focus. For example, an intervention using a high-energy and high-protein beverage would be classified 'macronutrient', while an intervention targeting calcium using either dairy or fortified orange juice would be classified 'micronutrient'. BMI was defined according to the baseline nutritional status of the mother. The Organisation for Economic Co-operation and Development (OECD) criteria (The World Bank Group 2012) was used to classify 'high-' and 'low-' income countries. Corresponding authors were emailed for additional data or clarification when needed. In trials reporting a range of gestational ages at study commencement, the most advanced week was used to calculate length of intervention.

Statistical analysis

The main measure of effect was the standardised mean difference (SMD). The SMD is determined by taking the difference in the mean outcome between the intervention and control group in one publication, and dividing it by the pooled standard deviation for the outcome across the whole trial. The SMD accounts for differences in variance between studies by allowing the size of the intervention effect in each publication to be expressed relative to the variability observed within that study. A secondary measure of effect, the raw mean difference (RMD), presented in the common units for each outcome [e.g. weeks gestation, mmHg for blood pressure (BP)] was also included. Trials containing more than one intervention (Rush *et al*. 1980; Ross *et al*. 1985; Chan *et al*. 2006), or control group (Smuts *et al*. 2003a) had their effects averaged, as there were no significant differences on pregnancy outcomes (Lipsey & Wilson 2001). Dichotomised outcomes had the log-odds and

standard errors converted to SMD via the method created by Hasselblad and Hedges (1995; Chinn 2000). Means and variance were approximated for trials reporting medians using the method reported by Hozo *et al*. (2005). Trials reporting outcomes with zero counts for both intervention and control groups were excluded from the analysis. For outcomes reporting zero counts for one intervention group only, the Firth penalized likelihood method was used to approximate the odds ratio (Firth 1993; Heinze & Schemper 2002) and 95% CIs.

Meta-analyses were performed for each outcome and dietary intervention type using a random effects model with random weights applied for each study. Subgroup analyses were performed for each outcome by dietary intervention, trimester when the intervention commenced, nutrient of interest, BMI and country income. The I^2 statistic was applied to describe the total variation in study estimates because of heterogeneity. Funnel plots were used as a visual tool for investigating the presence of potential bias and Egger's test used to test for funnel plot asymmetry (Egger *et al*. 1997). Statistical analyses were performed using the *metan* command in the statistical software package Intercooled Stata, version 12 (Stata, College Station, Texas, USA) (StataCorp 2011). *P*-values ≤0.01 were considered statistically significant to adjust for the multiple comparisons that were made.

Results

Description of studies

The trial selection process is summarised in Fig. 1. Of the 2326 papers screened, data were extracted from 28 publications, which included 8322 participants. The earliest published study was in 1978 (Mora *et al*. 1978) and the latest in 2011 (Luoto *et al*. 2011; Courville *et al*. 2011; Quinlivan *et al*. 2011;

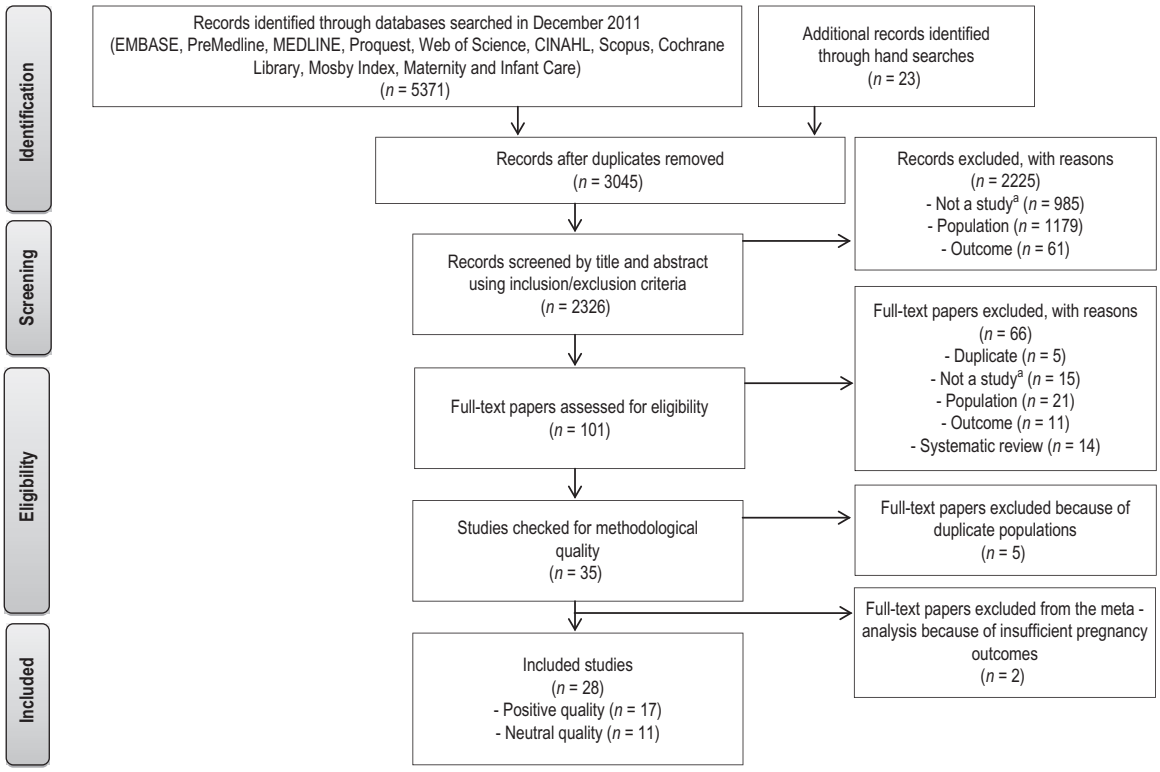

Fig. 1. Flow diagram for the selection of included studies.
[a]Not a study means not a study design of interest. It includes observational trials, editorials and conference papers.

Vinter *et al.* 2011). Twenty-four publications were performed in high-income OECD countries: 10 in the United States, 3 each in Finland, Denmark and the Netherlands, and 1 each in Australia, Norway, Italy, Greece and Chile (Supporting Information Table S1).

The methodological quality and characteristics of included publications are shown in Supporting Information Table S1. All studies were of positive or neutral quality according to the American Dietetic Association Quality Criteria Checklist (Academy of Nutrition and Dietetics 2012). Fourteen publications compared dietary counselling with standard antenatal care (no intervention) (Kafatos *et al.* 1989; Van Buul *et al.* 1997; Knuist *et al.* 1998; Briley *et al.* 2002; Bonomo *et al.* 2005; Khoury *et al.* 2005; Chan *et al.* 2006; O'Connor & Whaley 2007; Wolff *et al.* 2008; Asbee *et al.* 2009; Thornton *et al.* 2009; Luoto *et al.* 2010; Quinlivan *et al.* 2011; Vinter *et al.* 2011). Twelve publications evaluated the effect of specific food and fortified food products (Mora *et al.* 1978, 1979; Rush *et al.* 1980; McDonald *et al.* 1981; Metcoff *et al.* 1985; Ross *et al.* 1985; Mardones-Santander *et al.* 1988; Smuts *et al.* 2003a,b; de Groot *et al.* 2004; Bech *et al.* 2007; Courville *et al.* 2011), two of which came from the Columbian Longitudinal Study of Malnutrition and Intellectual Development (Mora *et al.* 1978, 1979). Two publications from the Finnish Mother–Infant Nutrition and Probiotic Intervention assessed the effect of combined dietary counselling, and food and fortified food products (Aaltonen *et al.* 2008; Luoto *et al.* 2010).

All but one publication (McDonald *et al.* 1981) studied the effect of dietary intervention during pregnancy on pregnancy outcomes. McDonald *et al.* (1981) included pre-pregnancy (periconception) dietary intervention. Twenty (of 28) publications, included two groups: dietary intake (intervention) vs. usual care or dietary intake (control). Dietary intervention during the second and third trimester was the most frequently reported period of intervention (22 publications). Dietary intervention ranged in duration from 10 (Rush *et al.* 1980) to greater than 40 weeks (pre-pregnancy intervention) (McDonald *et al.* 1981). Dietitians or nutritionists were the most frequent dietary intervention providers (12 publications) and macronutrients (24 publications) were more commonly targeted in the intervention than micronutrients (4 publications). Fourteen publications did not report any dietary data (Metcoff *et al.* 1985; Ross *et al.* 1985; Van Buul *et al.* 1997; Knuist *et al.* 1998; Smuts *et al.* 2003a,b; Bonomo *et al.* 2005; Bech *et al.* 2007; O'Connor & Whaley 2007; Asbee *et al.* 2009; Thornton *et al.* 2009; Courville *et al.* 2011; Quinlivan *et al.* 2011; Vinter *et al.* 2011), with 12 of these not conducting nutritional assessments during the intervention period (Metcoff *et al.* 1985; Ross *et al.* 1985; Van Buul *et al.* 1997; Knuist *et al.* 1998; Smuts *et al.* 2003a,b; Bonomo *et al.* 2005; Bech *et al.* 2007; O'Connor & Whaley 2007; Asbee *et al.* 2009; Thornton *et al.* 2009; Vinter *et al.* 2011). Six trials (7 publications) recruited women who were underweight or nutritionally at risk (Mora *et al.* 1978, 1979; Rush *et al.* 1980; McDonald *et al.* 1981; Metcoff *et al.* 1985; Ross *et al.* 1985; Mardones-Santander *et al.* 1988), while four trials recruited overweight or obese women as their target population (Wolff *et al.* 2008; Thornton *et al.* 2009; Quinlivan *et al.* 2011; Vinter *et al.* 2011). The pregnancy outcomes included: hypertensive disorders (pregnancy-induced hypertension and preeclampsia), BP, GDM, caesarean section, length of gestation, preterm and postterm delivery.

Effects of dietary intervention

Table 2 shows the effect of dietary intervention components and all dietary intervention trials on meta-analysed pregnancy outcomes. There was one trial (2 publications) in our review on the combination (counselling + food) dietary intervention (Aaltonen *et al.* 2008; Luoto *et al.* 2010). Aaltonen *et al.* (2008) analysed the effect of combination dietary intervention on the pregnancy outcomes preeclampsia, GDM, BP, length of gestation, preterm and postterm (Aaltonen *et al.* 2008), while Luoto *et al.* (2010) analysed combination dietary intervention and caesarean section only (Luoto *et al.* 2010). However, because of insufficient data for pooling, this trial was

Table 2. Effect of dietary interventions during pregnancy on pregnancy outcomes*

	Number of trials (references)	Number of participants		Intervention Number or Mean ± SD	Control Number or Mean ± SD	SMD† (95% CI)	RMD‡	*P*-value	I^2 (%)
		Int	Con						
Dietary counselling									
Hypertensive disorders	7 (Van Buul *et al.* 1997; Khoury *et al.* 2005; Wolff *et al.* 2008; Asbee *et al.* 2009; Thornton *et al.* 2009; Luoto *et al.* 2011; Vinter *et al.* 2011)	811	791	77	96	−0.12 (−0.30 to 0.06)	0.85	0.20	0
Systolic BP (mmHg)	3 (Chan *et al.* 2006; Wolff *et al.* 2008; Vinter *et al.* 2011)	225	207	115.65 ± 5.80	117.81 ± 7.61	−0.26 (−0.45 to −0.07)	−0.66	<0.001	0
Diastolic BP (mmHg)	3 (Chan *et al.* 2006; Wolff *et al.* 2008; Vinter *et al.* 2011)	225	207	71.81 ± 4.31	75.02 ± 6.50	−0.57 (−0.75 to −0.38)	−2.76	<0.001	0
Gestational diabetes	6 (Wolff *et al.* 2008; Asbee *et al.* 2009; Thornton *et al.* 2009; Luoto *et al.* 2011; Quinlivan *et al.* 2011; Vinter *et al.* 2011)	624	582	191	172	−0.27 (−0.72 to 0.17)	0.70	0.23	69
Length of gestation (weeks)	12 (Kafatos *et al.* 1989; Van Buul *et al.* 1997; Knuist *et al.* 1998; Bonomo *et al.* 2005; Khoury *et al.* 2005; Chan *et al.* 2006; O'Connor & Whaley 2007; Wolff *et al.* 2008; Asbee *et al.* 2009; Thornton *et al.* 2009; Luoto *et al.* 2011; Vinter *et al.* 2011)	1577	1526	39.60 ± 1.40	39.49 ± 1.59	0.06 (−0.05 to 0.16)	0.08	0.29	49
Preterm delivery	7 (Kafatos *et al.* 1989; Van Buul *et al.* 1997; Briley *et al.* 2002; Khoury *et al.* 2005; Thornton *et al.* 2009; Quinlivan *et al.* 2011; Vinter *et al.* 2011)	884	875	33	56	−0.25 (−0.56 to 0.05)	0.67	0.10	19
Post-term delivery	1 (Thornton *et al.* 2009)	116	116	15	16	−0.04 (−0.46 to 0.38)	0.95	0.85	NA
Caesarean section	6 (Knuist *et al.* 1998; Bonomo *et al.* 2005; Wolff *et al.* 2008; Asbee *et al.* 2009; Thornton *et al.* 2009; Vinter *et al.* 2011)	680	667	206	206	−0.02 (−0.17 to 0.14)	0.99	0.85	12
Food and food products									
Hypertensive disorders	2 (Smuts *et al.* 2003a,b)	160	184	6	10	−0.29 (−0.86 to 0.29)	1.04	0.33	0
Gestational diabetes	3 (Smuts *et al.* 2003a,b; de Groot *et al.* 2004)	200	223	4	7	0.20 (−0.50 to 0.89)	0.86	0.58	0
Length of gestation (weeks)	7 (Ross *et al.* 1985; Mardones-Santander *et al.* 1988; Smuts *et al.* 2003a,b; de Groot *et al.* 2004; Bech *et al.* 2007; Courville *et al.* 2011)	1399§	895§	39.68 ± 1.09	39.65 ± 1.37	0.09 (−0.13 to 0.25)	0.25	0.27	48
Preterm delivery	7 (Mora *et al.* 1978; Rush *et al.* 1980; McDonald *et al.* 1981; Smuts *et al.* 2003a,b; de Groot *et al.* 2004; Bech *et al.* 2007)	1620	1349	170	145	−0.10 (−0.23 to 0.04)	0.88	0.16	0
Post-term delivery	2 (Mora *et al.* 1978; McDonald *et al.* 1981)	315	310	18	26	−0.16 (−0.68 to 0.36)	0.76	0.54	43
Caesarean section	3 (Smuts *et al.* 2003a,b; de Groot *et al.* 2004)	189	213	23	35	−0.15 (−0.48 to 0.17)	0.82	0.35	0

Table 2. Continued

	Number of trials (references)	Number of participants		Intervention Number or Mean ± SD	Control Number or Mean ± SD	SMD† (95% CI)	RMD‡	*P*-value	I^2 (%)
		Int	Con						
Combination									
Hypertensive disorders	1 (Aaltonen *et al.* 2008)	86	85	5	6	−0.11 (−0.79 to 0.56)	0.83	0.74	NA
Systolic BP (mmHg)	1 (Aaltonen *et al.* 2008)	86	85	116 ± 14.10	112 ± 11	0.32 (0.01 to 0.62)	4.00	0.04	NA
Diastolic BP (mmHg)	1 (Aaltonen *et al.* 2008)	86	85	70 ± 9.60	69.00 ± 9.20	0.11 (−0.20 to 0.41)	1.00	0.49	NA
Gestational diabetes	1 (Aaltonen *et al.* 2008)	86	85	8	6	0.17 (−0.44 to 0.77)	1.29	0.59	NA
Length of gestation (weeks)	1 (Aaltonen *et al.* 2008)	86	85	38.25 ± 1.98	39.69 ± 1.4	−0.55 (−0.84 to −0.26)	−1.43	<0.001	NA
Preterm delivery	1 (Aaltonen *et al.* 2008)	86	85	4	1	0.78 (−0.44 to 2.00)	3.82	0.21	NA
Post-term delivery	1 (Aaltonen *et al.* 2008)	86	85	0	1	−0.62 (−2.40 to 1.16)	0.33	0.50	NA
Caesarean section	1 (Luoto *et al.* 2010)	77	76	12	11	0.05 (−0.44 to 0.54)	1.07	0.85	NA
All dietary interventions									
Hypertensive disorders	10	1057	1060	88	112	−0.13 (−0.30 to 0.04)	0.84	0.12	0
Systolic BP (mmHg)	4	311	292	115.74 ± 7.87	116.35 ± 8.46	−0.12 (−0.45 to 0.21)	−0.35	0.48	71
Diastolic BP (mmHg)	4	311	292	71.36 ± 5.63	73.51 ± 7.18	−0.40 (−0.79 to −0.01)	−0.45	0.04	80
Gestational diabetes	10	910	890	203	185	−0.12 (−0.44 to 0.20)	0.82	0.47	52
Length of gestation (weeks)	20	3062	2506	39.60 ± 1.31	39.55 ± 1.51	0.04 (−0.06 to 0.14)	0.10	0.42	61
Preterm delivery	15	2590	2309	207	202	−0.13 (−0.27 to 0.02)	0.84	0.09	15
Post-term delivery	4	517	511	33	43	−0.16 (−0.42 to 0.11)	0.79	0.24	0
Caesarean section	10	946	956	241	252	−0.03 (−0.16 to 0.10)	0.98	0.65	0

BP, blood pressure; CI, confidence interval; Con, control; Int, intervention; NA, not applicable; RMD, raw mean difference; SD, standard deviation; SMD, standardised mean difference.

*The main measure of effect was SMD. The SMD was determined by taking the difference in the mean of an outcome between the intervention and control group in one publication, and dividing it by the pooled standard deviation for the outcome across the whole trial. †The trials by Metcoff *et al.* (1985) and Mora *et al.* (1979) were not included in the meta-analysed outcome: length of gestation, as authors did not present results. ‡The secondary measure of effect was RMD. Categorical outcomes are reported as odds ratios. §The total number of participants (Int + Con) was 3307. The trial by Metcoff *et al.* (1985) provided withdrawals for the trial ($n = 61$); however, it did not specify participant withdrawals per Int and Con group. Because authors did not provide this data, all enrolled participants were included in the number of participants ($n = 3368$).

meta-analysed as part of all dietary interventions only.

Pregnancy hypertensive disorders

Seven RCTs studied the effect of dietary counselling on pregnancy hypertensive disorders ($n = 1602$ women) (Van Buul *et al*. 1997; Khoury *et al*. 2005; Wolff *et al*. 2008; Asbee *et al*. 2009; Thornton *et al*. 2009; Luoto *et al*. 2010; Vinter *et al*. 2011), with two trials reporting more than one hypertensive outcome (Wolff *et al*. 2008; Thornton *et al*. 2009). Trials examining the effect of dietary counselling on pregnancy hypertensive disorders included: three trials on pregnancy-induced hypertension (Van Buul *et al*. 1997; Wolff *et al*. 2008; Thornton *et al*. 2009), four trials on preeclampsia/eclampsia (Wolff *et al*. 2008; Asbee *et al*. 2009; Thornton *et al*. 2009; Luoto *et al*. 2010), and two trials combining pregnancy-induced hypertension and preeclampsia (Khoury *et al*. 2005; Vinter *et al*. 2011). Two RCTs studied the effect of food and fortified food products on pregnancy hypertensive disorders ($n = 344$ women) (Smuts *et al*. 2003a,b). Meta-analysis demonstrated no effect of dietary intervention components or all dietary interventions combined on pregnancy hypertensive disorders, with no evidence of heterogeneity or bias.

Maternal BP

Three RCTs studied the effect of dietary counselling on maternal BP (systolic and diastolic) ($n = 432$ women) (Chan *et al*. 2006; Wolff *et al*. 2008; Vinter *et al*. 2011). Meta-analysis demonstrated significant effects for a reduction in both systolic (SMD −0.26, 95% CI −0.45 to −0.07; $P < 0.001$; $I^2 = 0\%$) (Fig. 2) and diastolic BP (SMD −0.57, 95% CI −0.75 to −0.38; $P < 0.001$; $I^2 = 0\%$) for dietary counselling (Fig. 3). Using RMD, this roughly translates to a mean change in systolic and diastolic BP of −0.66 mmHg and −2.76 mmHg, respectively, although, this could vary by population. Meta-analysis demonstrated an effect for all dietary interventions combined on diastolic BP, with very high heterogeneity ($I^2 = 80\%$), and no evidence of bias. The four trials contributing to this analysis all commenced in the second trimester. Therefore, any effect from earlier or later intervention during pregnancy could not be determined.

GDM

Six RCTs studied the effect of dietary counselling on GDM ($n = 1206$ women) (Wolff *et al*. 2008; Asbee *et al*. 2009; Thornton *et al*. 2009; Luoto *et al*. 2010; Quinlivan *et al*. 2011; Vinter *et al*. 2011). Three RCTs studied the

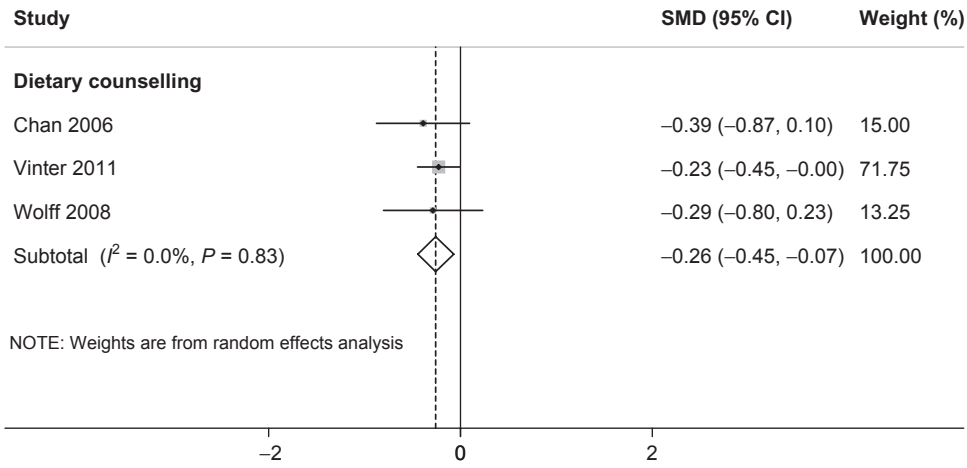

Fig. 2. Standardised mean difference (SMD) for systolic blood pressure and dietary counselling in pregnancy. The overall effect size was estimated by SMD. The black dot represents the point estimate, and square size the weight of each study in the meta-analysis and the horizontal lines represent the 95% confidence interval (CI). The vertical solid line represents the line of no effect. The diamond represents the overall pooled estimate effect of dietary counselling on systolic blood pressure.

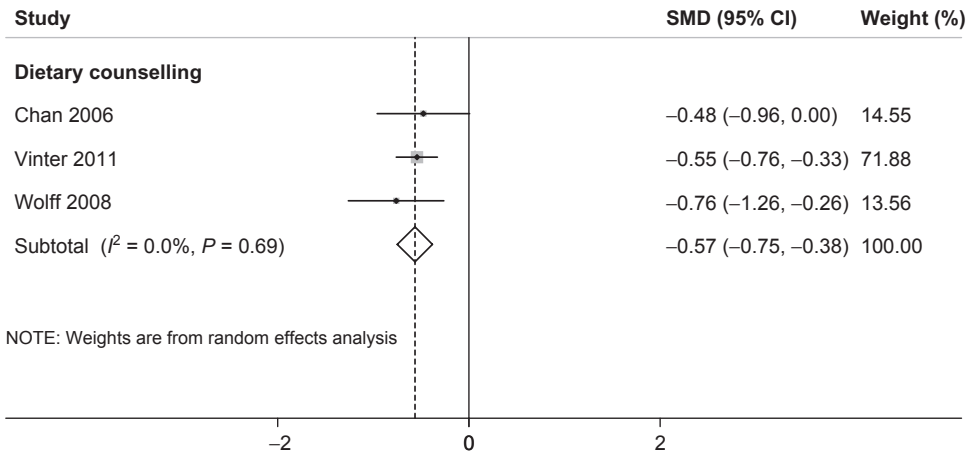

Fig. 3. Standardised mean difference (SMD) for diastolic blood pressure and dietary counselling in pregnancy. The overall effect size was estimated by SMD. The black dot represents the point estimate, and square size the weight of each study in the meta-analysis and the horizontal lines represent the 95% confidence interval (CI). The vertical solid line represents the line of no effect. The diamond represents the overall pooled estimate effect of dietary counselling on diastolic blood pressure.

effect of food and fortified food products on GDM (n = 423 women) (Smuts *et al*. 2003a,b; de Groot *et al*. 2004). Meta-analysis demonstrated no effect of dietary intervention components or all dietary interventions combined on GDM. The data showed heterogeneity (I^2 = 52%), with no presence of bias.

Caesarean section

Six RCTs studied the effect of dietary counselling on caesarean section (n = 1347 women) (Knuist *et al*. 1998; Bonomo *et al*. 2005; Wolff *et al*. 2008; Asbee *et al*. 2009; Thornton *et al*. 2009; Vinter *et al*. 2011). Three RCTs studied the effect of food and fortified food products on caesarean section (n = 402 women) (Smuts *et al*. 2003a,b; de Groot *et al*. 2004). Meta-analysis demonstrated no effect of dietary intervention components or all dietary interventions combined on caesarean section, with no evidence of heterogeneity or bias.

Length of gestation

Twelve RCTs studied the effect of dietary counselling on length of gestation (n = 3103 women) (Kafatos *et al*. 1989; Van Buul *et al*. 1997; Knuist *et al*. 1998; Bonomo *et al*. 2005; Khoury *et al*. 2005; Chan *et al*. 2006; O'Connor & Whaley 2007; Wolff *et al*. 2008; Asbee *et al*. 2009; Thornton *et al*. 2009; Luoto *et al*. 2010; Vinter *et al*. 2011). Nine RCTs studied the effect of food and fortified food products on length of gestation (n = 3307 women) (Mora *et al*. 1979; Metcoff *et al*. 1985; Ross *et al*. 1985; Mardones-Santander *et al*. 1988; Smuts *et al*. 2003a,b; de Groot *et al*. 2004; Bech *et al*. 2007; Courville *et al*. 2011), with seven of these trials meta-analysed (n = 2294). There were no effects of dietary intervention components or all dietary interventions combined on length of gestation. The data showed heterogeneity (I^2 = 61%), with no evidence of bias.

Fourteen RCTs (n = 4728) studied the effect of dietary intervention on preterm delivery (Mora *et al*. 1978; Rush *et al*. 1980; McDonald *et al*. 1981; Kafatos *et al*. 1989; Van Buul *et al*. 1997; Briley *et al*. 2002; Smuts *et al*. 2003a,b; de Groot *et al*. 2004; Khoury *et al*. 2005; Bech *et al*. 2007; Thornton *et al*. 2009; Quinlivan *et al*. 2011; Vinter *et al*. 2011). Seven RCTs studied the effect of dietary counselling on preterm delivery (n = 1759 women) (Kafatos *et al*. 1989; Van Buul *et al*. 1997; Briley *et al*. 2002; Khoury *et al*. 2005; Thornton *et al*. 2009; Quinlivan *et al*. 2011; Vinter *et al*. 2011): the other seven trials provided food and fortified food products (n = 2969) (Mora *et al*. 1978; Rush *et al*. 1980; McDonald *et al*. 1981; Smuts *et al*.

2003a,b; de Groot *et al*. 2004; Bech *et al*. 2007). Meta-analysis demonstrated no effect of dietary intervention components or all dietary interventions combined, with no evidence of heterogeneity or bias.

One RCT studied the effect of dietary counselling on post-term delivery ($n = 232$ women) (Thornton *et al*. 2009). Two RCTs studied the effect of food and fortified food products on post-term delivery ($n = 625$ women) (Mora *et al*. 1978; McDonald *et al*. 1981). Meta-analysis found no effect of dietary intervention components or all dietary interventions combined on post-term delivery, with no evidence of heterogeneity or bias.

Sub-analysis: trimester when the intervention commenced, nutrient of interest, BMI and country income

There were very few effects of diet on pregnancy outcome by trimester when the intervention commenced, nutrient of interest, BMI and country income subgroupings (Table 3). This was largely due to the small number of outcomes where three or more trials contributed data for each group. The trials for trimester when the intervention commenced on diastolic BP, and high-income country on hypertensive disorders, systolic and diastolic BP, GDM and caesarean section did not differ from the results presented in Table 2, therefore have not been repeated.

Trials altering the macronutrient composition of dietary interventions demonstrated a reduction in the incidence of preterm delivery ($P = 0.01$). Using RMD, this translates to a 21% reduction (0.79 odds ratio) in the incidence of preterm birth.

Discussion

Summary of main findings

To develop appropriate dietary guidelines for pregnancy we need to understand the effects of diet and dietary modification on a range of pregnancy outcomes. This systematic review summarises the best available evidence of dietary intervention during pregnancy on pregnancy outcomes. Results indicate that dietary interventions during pregnancy, particularly dietary counselling, slightly reduce BP (0.66 mmHg systolic and 2.76 mmHg diastolic), but not hypertensive disorders. Dietary interventions focusing on macronutrient intake reduce the incidence of preterm delivery (21% decrease in the odds), while interventions commencing in the second trimester reduce diastolic BP (0.45 mmHg). No other significant effects were observed for the other pregnancy outcomes.

Interpretation

The effect on BP was not consistent across trial populations. There were variations in the types of dietary intervention and the effect of specific nutrient components. There was no heterogeneity for dietary intervention components, but considerate heterogeneity for all dietary interventions combined. Dietary counselling interventions shown to lower BP varied in frequency from one to 10 sessions. Target populations ranged from adolescent girls (Chan *et al*. 2006) to obese women (Wolff *et al*. 2008; Vinter *et al*. 2011), limiting the generalisability of the findings to a broader group of women of child-bearing age. Those interventions shown to be effective in reducing BP included a balanced diet complying with National recommendations (Vinter *et al*. 2011), energy intake individualised for the needs of the mother (Wolff *et al*. 2008) and modifying calcium intake (Chan *et al*. 2006). Identifying specific components of successful interventions can assist in understanding how the intervention exerts its effect (Michie *et al*. 2011). Calcium has been studied in large supplemental trials (Palacios & Pena-Rosas 2011), demonstrating a reduction in the risk of hypertensive disorders during pregnancy, particularly for women at high-risk or with low calcium intakes (Hofmeyr *et al*. 2010; Palacios & Pena-Rosas 2011; Imdad & Bhutta 2012). Our review identified no effect of dietary intervention on hypertensive disorders. However, the pooled trials involved whole diet recommendations, or the modification of fat intake, and there were no trials on hypertensive disorders specifically targeting calcium intake.

Like the results in our review, calcium intake has been shown to lower BP among pregnant women

Table 3. Subgroup analyses for pregnancy outcomes in evaluation of dietary intervention during pregnancy*

	Number of trials	SMD (95% CI)	RMD†	*P*-value
Hypertensive disorders				
Trimester when the intervention commenced				
One	1	−0.16 (−1.70 to 1.39)	0.76	0.84
Two	9	−0.13 (−0.30 to 0.04)	0.84	0.13
Nutrient of interest				
Macronutrients	9	−0.16 (−0.34 to 0.02)	0.80	0.08
Micronutrient‡	1	0.04 (−0.37 to 0.45)	1.05	0.86
BMI				
Overweight and obese	3	−0.30 (−0.63 to 0.02)	0.69	0.07
All weight categories§	7	−0.03 (−0.25 to 0.18)	0.96	0.76
Systolic BP (mmHg)				
Nutrient of interest				
Macronutrients	3	−0.05 (−0.45 to 0.35)	−0.28	0.81
Micronutrient§	1	−0.39 (−0.87 to 0.10)	−3.51	0.12
BMI				
Overweight and obese	2	−0.24 (−0.44 to −0.03)	−0.51	0.02
All weight categories	2	−0.01 (−0.07 to 0.68)	−0.43	0.98
Diastolic BP (mmHg)				
Nutrient of interest				
Macronutrients	3	−0.38 (−0.88 to 0.12)	−1.43	0.13
Micronutrient¶	1	−0.48 (−0.96 to 0.00)	−3.50	0.05
BMI				
Overweight and obese	2	−0.58 (−0.78 to −0.38)	−2.69	<0.001
All weight categories	2	−0.15 (−0.72 to 0.41)	−0.92	0.60
Gestational diabetes				
Trimester when the intervention commenced				
One	1	−0.77 (−2.57 to 1.02)	0.26	0.40
Two	9	−0.10 (−0.43 to 0.23)	0.83	0.55
Nutrient of interest				
Macronutrients	10	−0.12 (−0.44 to 0.20)	0.82	0.47
BMI				
Overweight and obese	4	−0.42 (−0.91 to 0.06)	0.57	0.09
All weight categories	6	0.22 (−0.00 to 0.43)	1.11	0.05
Length of gestation (weeks)**				
Trimester when the intervention commenced				
One	2	0.11 (−1.31 to 1.53)	−0.06	0.88
Two	17	0.06 (−0.05 to 0.17)	0.14	0.30
Three	1	−1.44 (−1.64 to −1.23)	−0.20	<0.001
Nutrient of interest				
Macronutrients	15	0.01 (−0.10 to 0.13)	0.03	0.83
Micronutrients	5	0.11 (−0.08 to 0.31)	0.31	0.27
BMI				
Underweight/nutritional risk	2	0.00 (-0.21 to 0.21)	0.00	1.00
Overweight and obese	3	0.02 (−0.14 to 0.18)	0.00	0.83
All weight categories	15	0.05 (−0.07 to 0.18)	0.13	0.41
Country income				
Low	2	0.00 (−0.21 to 0.21)	0.00	1.00
High	18	0.05 (−0.06 to 0.15)	0.11	0.40
Preterm delivery				
Trimester when the intervention commenced				
One	1	−0.44 (−1.22 to 0.34)	0.49	0.27
Two	13	−0.13 (−0.30 to 0.05)	0.84	0.17
Three	1	−0.08 (−0.41 to 0.26)	0.89	0.66

Table 3. Continued

	Number of trials	SMD (95% CI)	RMD†	*P*-value
Nutrient of interest				
Macronutrients	13	–0.19 (–0.34 to –0.04)	0.79	0.01
Micronutrients	2	0.10 (–0.16 to 0.37)	1.18	0.44
BMI				
Underweight/nutritional risk	3	–0.15 (–0.32 to 0.01)	0.83	0.07
Overweight and obese	3	–0.04 (–0.48 to 0.41)	0.94	0.88
All weight categories	9	–0.16 (–0.46 to 0.14)	0.80	0.32
Country income				
Low	2	–0.13 (–0.44 to 0.17)	0.83	0.40
High	13	–0.13 (–0.30 to 0.05)	0.82	0.17
Post-term delivery				
Trimester when the intervention commenced				
One	1	0.20 (–0.52 to 0.91)	1.38	0.59
Two	2	–0.07 (–0.48 to 0.34)	0.91	0.73
Three	1	–0.36 (–0.76 to 0.04)	0.57	0.08
Nutrient of interest				
Macronutrients	4	–0.16 (–0.42 to 0.11)	0.79	0.24
BMI				
Underweight/nutritional risk	2	–0.16 (–0.68 to 0.36)	0.76	0.54
Overweight and obese	1	–0.04 (–0.46 to 0.38)	0.95	0.85
All weight categories	1	–0.62 (–2.40 to 1.16)	0.33	0.50
Country income				
Low	2	–0.16 (–0.68 to 0.36)	0.76	0.54
High	2	–0.07 (–0.48 to 0.34)	0.91	0.73
Caesarean section				
Trimester when the intervention commenced				
One	1	–0.48 (–1.03 to 0.08)	0.56	0.09
Two	8	–0.02 (–0.16 to 0.13)	0.99	0.84
Three	1	0.04 (–0.24 to 0.31)	1.04	0.80
Nutrient of interest				
Macronutrients	9	–0.00 (–0.14 to 0.13)	1.00	0.96
Micronutrients	1	–0.18 (–0.52 to 0.15)	0.77	0.28
BMI				
Overweight and obese	3	0.10 (–0.11 to 0.31)	1.05	0.36
All weight categories	7	–0.10 (–0.26 to 0.06)	0.89	0.41

BMI, body mass index; BP, blood pressure; CI, confidence interval; RMD, raw mean difference; SMD, standardised mean difference. *The main measure of effect was SMD. Meta-analysis focused on outcomes with three or more trials contributing data to pooled results. There were no studies for subgroup analyses for trimester commencement: one on systolic BP and diastolic BP, and three on hypertensive disorders, systolic BP, diastolic BP and gestational diabetes; or nutrient of interest: micronutrients on gestational diabetes and post-term delivery; BMI: underweight/nutritional risk on hypertensive disorders, systolic BP, diastolic BP and caesarean section; or country income: low on hypertensive disorders, systolic BP, diastolic BP, gestational diabetes and caesarean section. †The secondary measure of effect was RMD. Categorical outcomes are reported as odds ratios. ‡Micronutrient of interest is sodium. §All weight categories included those trials not restricting BMI within the target population. ¶Micronutrient of interest is calcium. **The trials by Metcoff *et al.* (1985) and Mora *et al.* (1979) were not included in the meta-analysed outcome: length of gestation, as authors did not present results.

(Carroli *et al*. 1994; Van Mierlo *et al*. 2006; Hofmeyr *et al*. 2010; Palacios & Pena-Rosas 2011), and the effects appear stronger in women with low calcium intakes prior to intervention (Carroli *et al*. 1994; Van Mierlo *et al*. 2006; Hofmeyr *et al*. 2010; Palacios & Pena-Rosas 2011). Calcium intakes were not analysed for each included study as part of this review. The effect of low calcium during pregnancy is thought to exert its effect via an increase in parathyroid hormone secretion, which increases intracellular calcium,

smooth muscle contractibility and/or releases renin from the kidney, leading to vasoconstriction and retention of sodium (Hacker *et al.* 2012). These physiological changes (from low calcium intakes during pregnancy) increase BP and potentially contribute to the development of hypertensive disorders (Hacker *et al.* 2012). Women beginning pregnancy with adequate intakes of at least 1000 mg calcium per day may not need additional amounts, while those with suboptimal intakes (<500 mg per day) may benefit from intervention (Hacker *et al.* 2012).

Our review also found that women consuming a balanced diet, including enough energy based on their individual requirements, had lower BP, paralleling current lifestyle recommendations for individuals with high BP (National Institutes of Health 2003). The effect size of 1–3 mmHg was very small compared with using antihypertensive agents (Patel *et al.* 2012). However, the effect was evident in normotensive rather than hypertensive women. Furthermore, treatment with antihypertensive drugs during pregnancy carries known and unknown risks to the fetus, because these drugs cross the placenta (e.g. nifedapine is Category C) (Department of Health, 2014). Therefore, any reductions in BP gained from dietary intervention offers clear advantages when BP is of clinical concern.

Macronutrient interventions demonstrating a reduction in the incidence of preterm delivery varied in frequency and included supplemental beverages (Rush *et al.* 1980; McDonald *et al.* 1981), dietary counselling on the nutritional needs during pregnancy (Kafatos *et al.* 1989; Briley *et al.* 2002; Quinlivan *et al.* 2011), limiting cholesterol and reducing saturated fat (Khoury *et al.* 2005), GDM-specific dietary recommendations (Thornton *et al.* 2009), providing DHA-fortified eggs (Smuts *et al.* 2003a,b), and a range of energy- and protein-based foods (Mora *et al.* 1978). Observational studies (Kramer 1987; Institute of Medicine 1990; Rush 2001) have reported that energy intake may be strongly and positively associated with a reduced risk of preterm birth. Our review confirms these findings, demonstrating a reduction in the incidence of preterm birth with macronutrient or whole diet intervention.

The pooled studies on preterm delivery showed little evidence of heterogeneity, with a narrow spread of data and overlapping CIs. Six of the macronutrient dietary interventions were conducted in low-income populations (Mora *et al.* 1978; Rush *et al.* 1980; McDonald *et al.* 1981; Kafatos *et al.* 1989; Briley *et al.* 2002; Quinlivan *et al.* 2011). Four interventions contained small sample sizes of less than 125 pregnant women (Briley *et al.* 2002; Smuts *et al.* 2003a; de Groot *et al.* 2004; Quinlivan *et al.* 2011) meaning these trials were individually underpowered. Of the 13 RCTs contributing data, only two trials (Kafatos *et al.* 1989; Khoury *et al.* 2005) demonstrated statistically significant effects in their respective publications. Based on the pooled dietary interventions, there were particular nutrients impacting on the result rather than the type of intervention. Modifying fat intake, particularly long-chain polyunsaturated fatty acids (LC-PUFA) were shown to reduce the incidence of preterm delivery (Smuts *et al.* 2003a,b; Khoury *et al.* 2005). Paralleling these findings, Horvath *et al.* (2007), Szajewska *et al.* (2006) and a recent Cochrane Review (Makrides *et al.* 2012) concluded that women allocated to LC-PUFA supplementation had longer gestation than women receiving placebo or no treatment, which remained true for both low- and high-risk pregnancies. Observational studies, mainly in populations with high consumption of seafood, have also suggested that marine LC-PUFA intake during pregnancy promotes longer gestation (Olsen *et al.* 1986, 1990, 1993). DHA and arachidonic acid are essential nutrients that are supplied during pregnancy to the fetus by preferential placental transfer (Al *et al.* 1995; Otto *et al.* 1997; Berghaus *et al.* 2000; Larquè *et al.* 2003). The mechanism behind their role in increasing gestational length may be an imbalance between DHA and arachidonic acid, which is associated with disturbances in the production of prostacyclin and thromboxane involved in the initiation of labour (Herrera 2002). Therefore, adequate dietary intake or supplementation of LC-PUFA may prolong gestational length, and in turn, decrease the risk of preterm delivery (Horvath *et al.* 2007).

The conversion of each publication's results to SMD for statistical inference effectively creates a common scale that would otherwise not be possible

because of the differences in variance between publications. The SMD express differences as units of standard deviations, and thus cannot be used to interpret absolute or relative differences in clinically meaningful outcomes (e.g. for BP, the absolute reduction in mmHg). Therefore, we have also provided the RMD to give some indication of effect size in the standard units for each outcome, including odds ratios for outcomes like pre- and post-term delivery.

Implications for practice and research

Dietary intervention during pregnancy slightly reduces maternal BP and the incidence of preterm delivery. No strong evidence was found for any effect of dietary intervention during pregnancy on the other outcomes. To develop appropriate dietary guidelines for pregnancy, we need to understand the role of everyday diet on pregnancy. This review advances our understanding of the role of nutrition for a healthy pregnancy by observing small reductions in BP and slight increases in the length of gestation. Further research, with larger sample sizes and robust methodology is required to better understand. Quantifying dietary intakes before, during and after an intervention would provide an important measure of compliance with the dietary intervention regime, which was lacking from most of the included trials ($n = 14$).

Limitations

This systematic review is broader in scope when compared with other published systematic reviews and meta-analyses (Dodd *et al*. 2010; Streuling *et al*. 2010; Tanentsapf *et al*. 2011; Oteng-Ntim *et al*. 2012; Thangaratinam *et al*. 2012), and the quality of the included studies was mostly positive. Gestational weight gain was not included as an outcome in this review, as others have focused on this (Dodd *et al*. 2010; Streuling *et al*. 2010; Tanentsapf *et al*. 2011; Oteng-Ntim *et al*. 2012; Thangaratinam *et al*. 2012). Despite the broad scope of this review, very few trials contributed data to each pregnancy outcome, with the exception of length of gestation ($n = 12$). For this reason, some of the outcomes were not reported and others are underpowered, particularly with the subgroup analysis. Dietary intervention trials should measure and report on a range of pregnancy outcomes so the effects of diet on pregnancy outcomes can be determined. There was heterogeneity for BP, GDM and length of gestation only, which was not explained by subgroup analyses, but may be due to the varying intensity and duration of included trials.

Conclusion

There is evidence that dietary intervention during pregnancy can reduce maternal BP and the incidence of preterm delivery. Interventions focusing on national recommendations and modifying calcium, saturated fat and cholesterol are the most promising dietary interventions to reduce BP and the incidence of preterm delivery. Further large high-quality RCTs investigating combination dietary intervention and micronutrient provision from food are needed. Future trials beginning in preconception and spanning for the duration of pregnancy, as well as between pregnancies are needed to advance our understanding of optimal nutrition for maternal–child health.

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Conflict of interest

The authors declare that they have no conflicts of interest.

Contributions

EG, AB, JEB, AJH designed research; EG and AJH conducted research; EG and AB analysed data; EG

wrote the paper and had primary responsibility for final content. All authors read and approved the final paper.

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Supporting Information

Additional Supporting Information may be found in the online version of this article at the publisher's web-site:

Table S1. Characteristics of included studies in a systematic review of dietary interventions during pregnancy on pregnancy outcomes