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## Dietary advice interventions in pregnancy for preventing gestational diabetes mellitus (Review)

Tieu J, Shepherd E, Middleton P, Crowther CA

Tieu J, Shepherd E, Middleton P, Crowther CA.

Dietary advice interventions in pregnancy for preventing gestational diabetes mellitus.

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[Intervention Review]

# Dietary advice interventions in pregnancy for preventing gestational diabetes mellitus

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## ABSTRACT

### Background

Gestational diabetes mellitus (GDM) is a form of diabetes occurring during pregnancy which can result in short- and long-term adverse outcomes for women and babies. With an increasing prevalence worldwide, there is a need to assess strategies, including dietary advice interventions, that might prevent GDM.

### Objectives

To assess the effects of dietary advice interventions for preventing GDM and associated adverse health outcomes for women and their babies.

### Search methods

We searched Cochrane Pregnancy and Childbirth's Trials Register (3 January 2016) and reference lists of retrieved studies.

### Selection criteria

Randomised controlled trials (RCTs) and quasi-RCTs assessing the effects of dietary advice interventions compared with no intervention (standard care), or to different dietary advice interventions. Cluster-RCTs were eligible for inclusion but none were identified.

### Data collection and analysis

Two review authors independently assessed study eligibility, extracted data and assessed the risk of bias of the included studies. Data were checked for accuracy. The quality of the evidence was assessed using the GRADE approach.

### Main results

We included 11 trials involving 2786 women and their babies, with an overall unclear to moderate risk of bias. Six trials compared dietary advice interventions with standard care; four compared low glycaemic index (GI) with moderate- to high-GI dietary advice; one compared specific (high-fibre focused) with standard dietary advice.

### *Dietary advice interventions versus standard care (six trials)*

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**Dietary advice interventions in pregnancy for preventing gestational diabetes mellitus (Review)**  
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Considering primary outcomes, a trend towards a reduction in GDM was observed for women receiving dietary advice compared with standard care (average risk ratio (RR) 0.60, 95% confidence interval (CI) 0.35 to 1.04; five trials, 1279 women;  $Tau^2 = 0.20$ ;  $I^2 = 56\%$ ;  $P = 0.07$ ; GRADE: *very low-quality evidence*); subgroup analysis suggested a greater treatment effect for overweight and obese women receiving dietary advice. While no clear difference was observed for pre-eclampsia (RR 0.61, 95% CI 0.25 to 1.46; two trials, 282 women; GRADE: *low-quality evidence*) a reduction in pregnancy-induced hypertension was observed for women receiving dietary advice (RR 0.30, 95% CI 0.10 to 0.88; two trials, 282 women; GRADE: *low-quality evidence*). One trial reported on perinatal mortality, and no deaths were observed (GRADE: *very low-quality evidence*). None of the trials reported on large-for-gestational age or neonatal mortality and morbidity.

For secondary outcomes, no clear differences were seen for caesarean section (average RR 0.98, 95% CI 0.78 to 1.24; four trials, 1194 women;  $Tau^2 = 0.02$ ;  $I^2 = 36\%$ ; GRADE: *low-quality evidence*) or perineal trauma (RR 0.83, 95% CI 0.23 to 3.08; one trial, 759 women; GRADE: *very low-quality evidence*). Women who received dietary advice gained less weight during pregnancy (mean difference (MD) -4.70 kg, 95% CI -8.07 to -1.34; five trials, 1336 women;  $Tau^2 = 13.64$ ;  $I^2 = 96\%$ ; GRADE: *low-quality evidence*); the result should be interpreted with some caution due to considerable heterogeneity. No clear differences were seen for the majority of secondary outcomes reported, including childhood/adulthood adiposity (skin-fold thickness at six months) (MD -0.10 mm, 95% CI -0.71 to 0.51; one trial, 132 children; GRADE: *low-quality evidence*). Women receiving dietary advice had a lower well-being score between 14 and 28 weeks, more weight loss at three months, and were less likely to have glucose intolerance (one trial).

The trials did not report on other secondary outcomes, particularly those related to long-term health and health service use and costs. We were not able to assess the following outcomes using GRADE: postnatal depression; maternal type 2 diabetes; neonatal hypoglycaemia; childhood/adulthood type 2 diabetes; and neurosensory disability.

#### ***Low-GI dietary advice versus moderate- to high-GI dietary advice (four trials)***

Considering primary outcomes, no clear differences were shown in the risks of GDM (RR 0.91, 95% CI 0.63 to 1.31; four trials, 912 women; GRADE: *low-quality evidence*) or large-for-gestational age (average RR 0.60, 95% CI 0.19 to 1.86; three trials, 777 babies;  $Tau^2 = 0.61$ ;  $P = 0.07$ ;  $I^2 = 62\%$ ; GRADE: *very low-quality evidence*) between the low-GI and moderate- to high-GI dietary advice groups. The trials did not report on: hypertensive disorders of pregnancy; perinatal mortality; neonatal mortality and morbidity.

No clear differences were shown for caesarean birth (RR 1.27, 95% CI 0.79 to 2.04; two trials, 201 women; GRADE: *very low-quality evidence*) and gestational weight gain (MD -1.23 kg, 95% CI -4.08 to 1.61; four trials, 787 women;  $Tau^2 = 7.31$ ;  $I^2 = 90\%$ ; GRADE: *very low-quality evidence*), or for other reported secondary outcomes.

The trials did not report the majority of secondary outcomes including those related to long-term health and health service use and costs. We were not able to assess the following outcomes using GRADE: perineal trauma; postnatal depression; maternal type 2 diabetes; neonatal hypoglycaemia; childhood/adulthood adiposity; type 2 diabetes; and neurosensory disability.

#### ***High-fibre dietary advice versus standard dietary advice (one trial)***

The one trial in this comparison reported on two secondary outcomes. No clear difference between the high-fibre and standard dietary advice groups observed for mean blood glucose (following an oral glucose tolerance test at 35 weeks), and birthweight.

#### **Authors' conclusions**

Very low-quality evidence from five trials suggests a possible reduction in GDM risk for women receiving dietary advice versus standard care, and low-quality evidence from four trials suggests no clear difference for women receiving low- versus moderate- to high-GI dietary advice. A possible reduction in pregnancy-induced hypertension for women receiving dietary advice was observed and no clear differences were seen for other reported primary outcomes. There were few outcome data for secondary outcomes.

For outcomes assessed using GRADE, evidence was considered to be low to very low quality, with downgrading based on study limitations (risk of bias), imprecision, and inconsistency.

More high-quality evidence is needed to determine the effects of dietary advice interventions in pregnancy. Future trials should be designed to monitor adherence, women's views and preferences, and powered to evaluate effects on short- and long-term outcomes; there is a need for such trials to collect and report on core outcomes for GDM research. We have identified five ongoing studies and four are awaiting classification. We will consider these in the next review update.

## **PLAIN LANGUAGE SUMMARY**

## Dietary advice during pregnancy to prevent gestational diabetes

### What is the issue?

Can dietary advice for pregnant women prevent the development of diabetes in pregnancy, known as gestational diabetes mellitus (GDM), which can cause health complications for women and their babies?

### Why is this important?

Women with GDM have an increased risk of developing high blood pressure and protein in their urine during pregnancy (pre-eclampsia), and of having a caesarean section birth. Their babies may grow large and, as a result, be injured at birth, or cause injury to their mothers during birth. Additionally, there can be long-term health problems for women and their babies, including an increased risk of cardiovascular disease or type 2 diabetes. The number of women being diagnosed with GDM is increasing around the world, so finding simple and cost-effective ways to prevent women developing GDM is important.

Carbohydrates are the main nutrient affecting blood glucose after meals. The glycaemic index (GI) can be used to characterise the capability of carbohydrate-based foods to raise these levels. Some diets, for example, those with low-fibre and high-GI foods, can increase the risk of developing GDM. It has been suggested that dietary advice interventions in pregnancy may help to prevent women developing GDM.

### What evidence did we find?

We searched for studies on 3 January 2016, and included 11 randomised controlled trials involving 2786 pregnant women and their babies. The quality of the evidence was assessed as low or very low and the overall risk of bias of the trials was unclear to moderate. Six trials compared dietary advice with standard care, four compared advice focused on a low-GI diet with advice for a moderate- to high-GI diet, and one compared dietary advice focused on a high-fibre diet with standard advice.

There was a possible reduction in the development of GDM for women who received dietary advice versus standard care across five trials (1279 women, *very low-quality evidence*), though no clear difference for GDM was seen between women who received low- versus moderate- to high-GI diet advice across four trials (912 women, *low-quality evidence*). Two trials (282 women) reported no clear difference between women who received dietary advice versus standard care for pre-eclampsia (*low-quality evidence*), though fewer women who received dietary advice developed pregnancy-induced high blood pressure (*low-quality evidence*). There was no clear difference between the groups of women who received low-GI and moderate- to high-GI diet advice, in the number of babies born large-for-gestational age across three trials (777 babies, *very low-quality evidence*). Only one trial comparing dietary advice with standard care reported on the number of babies who died (either before birth or shortly afterwards), with no deaths in this trial.

There were no clear differences for most of the other outcomes assessed in the trials comparing dietary advice with standard care, including caesarean section, perineal trauma, and child skin-fold thickness at six months. However, women who received dietary advice gained less weight during their pregnancy across five trials (1336 women) (*low-quality evidence*).

Similarly, there were no clear differences for other outcomes assessed in the trials comparing low- and moderate- to high-GI diet advice, including for caesarean birth and weight gain in pregnancy. The trial comparing dietary advice focused on a high-fibre diet with standard advice found no clear differences for any outcomes.

The included trials did not report on a large number of outcomes listed in this review, including outcomes relating to longer-term health for the women and their babies (as children and adults), and the use and cost of health services.

### What does this mean?

Dietary advice interventions for pregnant women may be able to prevent GDM. Based on current trials, however, conclusive evidence is not yet available to guide practice. Further large, well-designed, randomised controlled trials are required to assess the effects of dietary interventions in pregnancy for preventing GDM and improving other health outcomes for mothers and their babies in the short and long term. Five trials are ongoing, and four await classification (pending availability of more information) and will be considered in the next update of this review.



## SUMMARY OF FINDINGS FOR THE MAIN COMPARISON *[Explanation]*

Dietary advice interventions versus standard care (maternal outcomes)						
<b>Population:</b> pregnant women <b>Setting:</b> 6 studies carried out in Australia, the USA, Brazil, Denmark, Ireland and Finland <b>Intervention:</b> dietary advice interventions <b>Comparison:</b> standard care						
Outcomes	Anticipated absolute effects* (95% CI)		Relative effect (95% CI)	No of participants (studies)	Quality of the evidence (GRADE)	Comments
	Risk with standard care	Risk with dietary advice interventions				
Gestational diabetes	Study population		RR 0.60 (0.35 to 1.04)	1279 (5 RCTs)	⊕○○○ VERY LOW <sup>1,2,3</sup>	
	126 per 1000	76 per 1000 (44 to 131)				
Hypertensive disorders of pregnancy (pregnancy-induced hypertension)	Study population		RR 0.30 (0.10 to 0.88)	282 (2 RCTs)	⊕⊕○○ LOW <sup>1,4</sup>	Anticipated absolute effects based on only 2 trials contributing data
	98 per 1000	29 per 1000 (10 to 86)				
Hypertensive disorders of pregnancy (pre-eclampsia)	Study population		RR 0.61 (0.25 to 1.46)	282 (2 RCTs)	⊕⊕○○ LOW <sup>1,5</sup>	Anticipated absolute effects based on only 2 trials contributing data
	84 per 1000	51 per 1000 (21 to 123)				
Caesarean section	Study population		RR 0.98 (0.78 to 1.24)	1194 (4 RCTs)	⊕⊕○○ LOW <sup>1,3</sup>	
	300 per 1000	294 per 1000 (234 to 372)				
Perineal trauma (anal sphincter injury)	Study population		RR 0.83 (0.23 to 3.08)	759 (1 RCT)	⊕○○○ VERY LOW <sup>6,7</sup>	Anticipated absolute effect based on findings from a single study

	13 per 1000	11 per 1000 (3 to 40)				
Gestational weight gain (kg)	The mean gestational weight gain in the intervention group was 4.7 kg less (8.07 kg less to 1.34 kg less)		MD -4.70 (-8.07 to -1.34)	1336 (5 RCTs)	⊕⊕○○ LOW <sup>1,8</sup>	There was high heterogeneity for this outcome
Postnatal depression			Not estimable	(0 studies)	No data reported for postnatal depression in any of the included studies	

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

**CI:** confidence interval; **kg:** kilogram; **MD:** mean difference; **RCT:** randomised controlled trial; **RR:** risk ratio

#### GRADE Working Group grades of evidence

**High quality:** We are very confident that the true effect lies close to that of the estimate of the effect

**Moderate quality:** We are moderately confident in the effect estimate: The true effect is likely to be close to the estimate of the effect, but there is a possibility that it is substantially different

**Low quality:** Our confidence in the effect estimate is limited: The true effect may be substantially different from the estimate of the effect

**Very low quality:** We have very little confidence in the effect estimate: The true effect is likely to be substantially different from the estimate of effect

<sup>1</sup>The studies contributing data had design limitations

<sup>2</sup>There was considerable variation in the size of the effect in different studies

<sup>3</sup>Wide 95% CI crossing the line of no effect

<sup>4</sup>Estimate based on studies with small sample sizes

<sup>5</sup>Estimate based on studies with small sample sizes, low event rates and 95% CI crossing the line of no effect

<sup>6</sup>Single study with design limitations

<sup>7</sup>Single study contributing data, low event rate and wide 95% CI crossing the line of no effect

<sup>8</sup>Very substantial heterogeneity ( $I^2 = 96\%$ )

## BACKGROUND

### Description of the condition

#### Gestational diabetes mellitus

Gestational diabetes mellitus (GDM) is defined as glucose intolerance or hyperglycaemia (high blood glucose concentration) that begins or is first detected during pregnancy (WHO 1999). In pregnancy, placental hormones (including oestrogen, progesterone, cortisol, placental lactogen, prolactin and growth hormone), create an insulin-resistant state in order to direct sufficient nutrition to the fetus (Setji 2005). For women with GDM, increased insulin resistance accompanied by an insufficient compensatory insulin release limits glucose transport into cells, resulting in maternal hyperglycaemia (Setji 2005). This in turn can result in fetal hyperglycaemia, which stimulates insulin production, thus allowing increased glucose and amino acid entry into cells, increasing metabolism and, ultimately, over-nourishing the fetus (Setji 2005).

A variety of risk factors for the development of GDM have been identified. These include non-modifiable risk factors, such as ethnicity (e.g. African, Hispanic, South or East Asian, Native American and Pacific Islander), advanced maternal age, maternal high or low birthweight, high parity (Petry 2010), polycystic ovarian syndrome (Toulis 2009), a past history of a macrosomic (large) baby or a stillbirth (Petry 2010), a past history of GDM (Kim 2007), and family history of first-degree relatives with GDM or type 2 diabetes (Petry 2010). Risk factors considered modifiable include maternal overweight or obesity (body mass index (BMI) equal to or greater than 25 kg/m<sup>2</sup> or 30 kg/m<sup>2</sup>, respectively) (Morisset 2010; Torloni 2009), certain dietary factors (Morisset 2010; Zhang 2011; *discussed further below*), physical inactivity before or in early pregnancy (Tobias 2011), and weight gain during pregnancy (Morisset 2010), including for those women who are overweight or obese. Selective (risk factor) or universal screening for GDM is usually performed between 24 and 28 weeks' gestation, with the use of a 50 g oral glucose challenge test (OGCT). Diagnosis is then made following a 75 g two-hour or 100 g three-hour oral glucose tolerance test (OGTT). Alternatively, a diagnostic OGTT may be used without prior OGCT. Diagnostic criteria for GDM have changed over time, and vary nationally and internationally (ACOG 2013; ADA 2013; IADPSG 2010; Nankervis 2014; New Zealand Ministry of Health 2014; NICE 2015; WHO 1999). Accordingly, GDM prevalence estimates vary depending on both the diagnostic criteria used, and the population(s) assessed; for example, a recent study identified GDM prevalence estimates to range from less than 1% to 28% (with data derived from expert estimates, single/multi-site and national prevalence assessments across 30 countries) (Jiwani 2012). Despite variation in diagnostic criteria, there is widespread agreement that the prevalence of diabetes,

including GDM, is increasing across the world, in line with the escalating prevalence of obesity. A recent estimate of the global prevalence of hyperglycaemia in pregnancy (including GDM and 'total diabetes' (known/unknown pre-existing diabetes) was 17% (Guariguata 2014).

GDM is associated with adverse consequences for women and their babies, in the short and long term. Babies of women with GDM are more likely to be macrosomic (with a birthweight exceeding 4000 g or 4500 g), or be born large for their gestational age (Reece 2009; Reece 2010). These babies are thus at an increased risk of injury at the time of birth, including birth asphyxia, shoulder dystocia, bone fracture or nerve palsy (Reece 2009; Reece 2010). Additional risks for babies in the short term include respiratory distress syndrome, hypoglycaemia, hyperbilirubinaemia, hypocalcaemia, hypomagnesaemia, and polycythaemia (Reece 2009; Reece 2010); such health consequences together contribute to an increased risk of admission to the neonatal nursery, and perinatal mortality. There is increasing evidence to suggest that infants born to women with GDM are at risk of being obese in childhood or adulthood, and at increased risk of developing metabolic syndrome, and type 2 diabetes later in life (Reece 2009; Reece 2010). Women with GDM are at additional risk in the short term of developing pre-eclampsia, having a caesarean section birth (including due to their babies being large-for-gestational age) and perineal trauma; in the longer term, women are at increased risk of developing GDM in a subsequent pregnancy, and of developing later cardiovascular disease, metabolic syndrome and type 2 diabetes (Reece 2009; Reece 2010).

### Description of the intervention

#### Dietary advice in pregnancy for preventing gestational diabetes mellitus

Dietary interventions in pregnancy to prevent GDM may incorporate varied advice, based on addressing potential risk factors, with the aim of preventing maternal hyperglycaemia. Dietary interventions to treat pregnancy hyperglycaemia have a similar aim of optimising glycaemic control, and thus improving outcomes for women and their babies, and are widely used as a primary management strategy. Three Cochrane reviews have assessed such interventions (incorporating dietary advice), in pregnancy for the treatment of hyperglycaemia (Han 2012b) or GDM (Alwan 2009; Han 2013), and have showed some benefits. Han 2012b included four trials (involving 543 women and their babies) assessing dietary advice and blood glucose concentration monitoring for women with pregnancy hyperglycaemia not meeting GDM and type 2 diagnostic criteria. The review revealed a reduced risk of babies being born large-for-gestational age and macrosomic with such interventions; however Han 2012b highlighted that the results were based on small, low-quality randomised trials and thus recognised

a need for further research (Han 2012b). Alwan 2009 included eight randomised trials (involving 1418 women and their babies) assessing a range of interventions for treating GDM, with five incorporating dietary advice. This review revealed that these interventions, incorporating dietary advice (and insulin therapy, where indicated), reduced the risk of adverse consequences for women and their babies (including pre-eclampsia, a composite outcome of perinatal death, shoulder dystocia, bone fracture and nerve palsy, macrosomia and large-for-gestational age), and concluded that further research should focus on the impact of different types of intensive treatment on short- and long-term outcomes (Alwan 2009). Han 2013 was specifically conducted to assess the effects of different types of dietary advice for women with GDM, and identified nine randomised trials (involving 437 women and their babies). These trials compared dietary advice focused on: low- to moderate-GI food versus high- to moderate-GI food; low-GI diet versus high-fibre moderate-GI diet; energy-restricted diet versus no energy restriction diet; low-carbohydrate diet versus high-carbohydrate diet; high-monounsaturated fat diet versus high-carbohydrate diet; and standard-fibre diet versus fibre-enriched diet. The types of dietary advice that are most effective for women with GDM remains uncertain (Han 2013).

Two further Cochrane reviews have assessed the effects of dietary advice interventions (Nield 2008), and combined dietary advice and exercise advice interventions (Orozco 2008) for preventing type 2 diabetes in adults. Nield 2008 included two randomised trials involving 328 people. A 33% reduction ( $P < 0.03$ ) in the incidence of diabetes with dietary advice in one trial at six-year follow-up and beneficial effects on markers of metabolic control (reductions in insulin resistance, fasting insulin and blood glucose) in the other included trial at one-year follow-up were shown. Nield 2008 recognised that “*more well-designed, long-term studies, providing well-reported, high-quality data are required before proper conclusions can be made into the best dietary advice for the prevention of diabetes mellitus in adults*” (Nield 2008). The Nield 2008 review has been withdrawn as it will be superseded by a new review, with broader scope, focused on ‘Diet, physical activity or both for the prevention or delay of type 2 diabetes mellitus and its associated complications in persons at increased risk’ (Nield 2016). Orozco 2008 included eight trials (involving 5956 people) assessing exercise plus diet interventions for prevention of type 2 diabetes, and overall showed a reduction in the risk of diabetes with such interventions (risk ratio (RR) 0.63, 95% confidence interval (CI) 0.49 to 0.79). The authors of the review concluded benefit for the high-risk groups assessed (people with impaired glucose tolerance or the metabolic syndrome), and highlighted a need for further research assessing impact on other outcomes including quality of life, morbidity and mortality, with a special focus on cardiovascular outcomes (Orozco 2008).

While the benefits of dietary advice intervention have been observed in randomised trials and systematic reviews for women with GDM (Alwan 2009), in relation to improved outcomes for women

and their babies, and for adults at risk of type 2 diabetes (Nield 2008; Orozco 2008), in regards to prevention of diabetes development, to date, the effects of such interventions for pregnant women for the prevention of GDM are unclear.

## How the intervention might work

### Dietary advice in pregnancy for preventing gestational diabetes mellitus

A large and increasing number of observational studies have suggested various components of women’s diets or dietary patterns before and during pregnancy which may influence GDM risk (Zhang 2011). Some of the most recent published evidence relates to the Nurses’ Health Study II, a longitudinal cohort of 116,000 nurses between the ages of 25 and 42 in the USA followed since 1989. Studies from this cohort indicate that pre-pregnancy, higher consumption of sugar-sweetened cola (Chen 2009); frequent fried food consumption, particularly away from home (Bao 2014); higher levels of potato consumption (Bao 2016); higher intakes of animal fat and dietary cholesterol (Bowers 2012); a high-glycaemic load and low-cereal fibre diet (Zhang 2006b); higher intake of dietary heme iron (Bowers 2011); higher intake of animal protein, in particular red and processed meat (Zhang 2006); and a low-carbohydrate dietary pattern with high protein and fat from animal-food sources (Bao 2014b) are associated with a higher risk of GDM (Bao 2013). Conversely, pre-pregnancy, a high-fibre diet (Zhang 2006b); higher intake of vegetable protein (specifically nuts); the substitution of red meat with poultry, fish, nuts or legumes (Bao 2013); the substitution of potatoes with other vegetables, legumes or whole grain foods (Bao 2016); and adherence to “*healthful*” dietary patterns (alternate Mediterranean (aMED), Dietary Approaches to Stop Hypertension (DASH), and the alternate Healthy Eating Index (aHEI) dietary patterns) (Tobias 2012) are associated with a lower risk of GDM.

Additional observational studies have shown numerous, and often similar, findings; with high egg and cholesterol intakes before and during pregnancy (Qiu 2011b); high levels of dietary heme iron intake before and during early pregnancy (Qiu 2011); a ‘Meats, snacks and sweets’ dietary pattern before pregnancy (Schoenaker 2015); lower carbohydrate and higher total fat intakes as a percentage of energy in pregnancy (Ley 2011); and high consumption of refined grains, fat, added sugars and low intake of fruits and vegetables during pregnancy (Shin 2015) shown to be associated with a higher risk of GDM. A ‘Mediterranean-style’ dietary pattern before pregnancy (Schoenaker 2015); a healthy “*prudent*” dietary pattern (with high intakes of seafood, eggs, vegetables, fruits and berries, vegetable oils, nuts and seeds, pasta, breakfast cereals; and low intakes of soft drinks and French fries) during pregnancy especially among women who were overweight or obese (Tryggvadottir 2016); and improved quality of dietary fat incorporating increased

n-3 polyunsaturated fatty acid intake during pregnancy (Barbieri 2015) have been associated with a lower risk of GDM.

Thus, as a number of dietary components/patterns have been shown to influence GDM risk, specific dietary advice interventions aimed at preventing GDM may be varied, and as such, may influence maternal glycaemic control through multiple mechanisms.

Advice can focus on both the quantity and type of carbohydrates consumed, with carbohydrates being the main nutrient affecting post-prandial glucose concentration (Reader 2007). Dietary GI can be used to characterise the capability of carbohydrate-based foods to induce post-prandial glycaemia (Jenkins 1981); foods with a low GI (such as whole-grain foods and many fruits and vegetables) produce a lower post-prandial glucose elevation, while foods with a high GI (highly processed foods such as white bread and some breakfast cereals) produce a rapid increase in post-prandial blood glucose concentration (Jenkins 1981). Dietary fibre may influence glucose homeostasis through a number of mechanisms (Zhang 2006b). For example, a high-fibre diet may reduce appetite and lower total energy intake, thus reducing adiposity and improving insulin sensitivity, or increased fibre may delay gastric emptying and slow digestion, subsequently reducing glucose absorption and insulin secretion (Zhang 2006b). Red and processed meat intake is postulated to influence glycaemic control through a variety of pathways (Zhang 2006), including through the adverse effects of components including saturated fat and cholesterol, and nitrites, used as a preservative agent, influencing insulin sensitivity and/or pancreatic beta-cell function, or through the toxic effects of advanced glycation end products (AGEs), which can be formed in meat following heating and processing (Zhang 2006). Restricting calorie intake could also influence glycaemic status and insulin sensitivity, through promoting weight loss and reduced fat mass (Knopp 1991). Excessive calorie restriction can however lead to ketonuria and ketonaemia through ketosis by accelerated fat catabolism, which has been associated with adverse psychomotor development for the child (Rizzo 1995).

### Why it is important to do this review

GDM is associated with a wide range of adverse health consequences for women and their babies in the short and long term. With potential for adverse consequences, and the increasing prevalence worldwide, there is an urgent need for effective strategies to prevent GDM.

This review will complement the existing reviews assessing interventions for preventing GDM, including: 'Diet and exercise interventions for preventing gestational diabetes mellitus' (Bain 2015); 'Exercise for pregnant women for preventing gestational diabetes mellitus' (Han 2012); 'Antenatal dietary supplementation with myo-inositol in women during pregnancy for preventing gestational diabetes' (Crawford 2015); and 'Probiotics for preventing gestational diabetes'

(Barrett 2014); and will assess dietary advice interventions for preventing GDM.

## OBJECTIVES

To assess the effects of dietary advice interventions for preventing gestational diabetes mellitus (GDM) and associated adverse health outcomes for women and their babies (as neonates, infants, children and adults).

## METHODS

### Criteria for considering studies for this review

#### Types of studies

We included all randomised and quasi-randomised controlled trials assessing the effects of dietary advice interventions for preventing gestational diabetes mellitus (GDM). We planned to include cluster-randomised trials, and we excluded cross-over trials.

We did not include trials presented only as abstracts (unless sufficient information was available to assess risk of bias and obtain data on primary/secondary outcomes); we plan to reconsider such trials for inclusion once they are published in full-text manuscript format.

#### Types of participants

We included studies involving pregnant women regardless of age, gestation, parity or plurality. We excluded studies involving women with pre-existing type 1 or type 2 diabetes.

#### Types of interventions

We included interventions that assessed any type of dietary advice before testing for GDM. We included studies where such interventions were compared with no dietary advice intervention (i.e. standard care), and to different types of dietary advice.

We did not include interventions assessing combined dietary advice and exercise interventions, as these are assessed in the Bain 2015 Cochrane review.

#### Types of outcome measures

##### Primary outcomes

For this update, we used the core outcome set agreed by consensus between review authors of Cochrane Pregnancy and Childbirth

systematic reviews for prevention and treatment of gestational diabetes mellitus (GDM) and pre-existing diabetes.

### **Perinatal outcomes**

- GDM
- Hypertensive disorders of pregnancy (e.g. pre-eclampsia, pregnancy-induced hypertension, eclampsia)

### **Fetal/neonatal outcomes**

- Perinatal mortality (stillbirth or neonatal mortality)
- Large-for-gestational age
- Mortality or morbidity composite (e.g. death, shoulder dystocia, bone fracture or nerve palsy)

### **Secondary outcomes**

### **For the mother**

#### *Perinatal outcomes*

- Caesarean section birth
- Operative vaginal birth
- Induction of labour
- Perineal trauma
- Placental abruption
- Postpartum haemorrhage
- Postpartum infection
- Breastfeeding (e.g. at discharge, six weeks postpartum)
- Gestational weight gain
- Adherence with the intervention
- Behaviour changes associated with the dietary intervention
- Relevant biomarker changes associated with the intervention
- Sense of well-being and quality of life
- Views of the intervention

#### *Longer-term maternal outcomes*

- Postnatal depression
- GDM in a subsequent pregnancy
- Type 1 diabetes mellitus
- Type 2 diabetes mellitus
- Impaired glucose tolerance
- Postnatal weight retention or return to pre-pregnancy weight
- Body mass index (BMI)
- Cardiovascular health (e.g. blood pressure, hypertension, cardiovascular disease, metabolic syndrome)

### **For the child**

#### *Fetal/neonatal outcomes*

- Stillbirth
- Neonatal mortality
- Preterm birth (before 37 weeks' gestation; before 34 weeks' gestation)
- Apgar score less than seven at five minutes
- Macrosomia
- Small-for-gestational age
- Shoulder dystocia
- Nerve palsy
- Bone fracture
- Respiratory distress syndrome
- Hypoglycaemia
- Hyperbilirubinemia
- Gestational age at birth
- Birthweight and z score
- Head circumference and z score
- Length and z score
- Ponderal index
- Adiposity (e.g. as measured by BMI or skinfold thickness)

#### *Childhood/adulthood outcomes*

- Weight and z scores
- Height and z scores
- Head circumference and z scores
- Adiposity (e.g. as measured by BMI, skinfold thickness)
- Cardiovascular health (e.g. blood pressure, hypertension, cardiovascular disease, metabolic syndrome)
- Employment, education and social status/achievement
- Type 1 diabetes mellitus
- Type 2 diabetes mellitus
- Impaired glucose tolerance
- Neurosensory disability

#### **Use of health services**

- Number of hospital or health professional visits (e.g. midwife, obstetrician, physician, dietitian, diabetic nurse)
- Number of antenatal visits or admissions
- Length of antenatal stay
- Neonatal intensive care unit admission
- Length of postnatal stay (mother)
- Length of postnatal stay (baby)
- Costs to families associated with the management provided
- Costs associated with the intervention
- Cost of maternal care
- Cost of infant care

## Search methods for identification of studies

The following methods section of this review is based on a standard template used by the Cochrane Pregnancy and Childbirth Group.

### Electronic searches

We searched Cochrane Pregnancy and Childbirth's Trials Register by contacting their Information Specialist (3 January 2016).

The Register is a database containing over 22,000 reports of controlled trials in the field of pregnancy and childbirth. For full search methods used to populate Pregnancy and Childbirth's Trials Register including the detailed search strategies for CENTRAL, MEDLINE, Embase and CINAHL; the list of handsearched journals and conference proceedings, and the list of journals reviewed via the current awareness service, please follow this link to the editorial information about the [Cochrane Pregnancy and Childbirth](#) in the Cochrane Library and select the '*Specialized Register*' section from the options on the left side of the screen.

Briefly, Cochrane Pregnancy and Childbirth's Trials Register is maintained by their Information Specialist and contains trials identified from:

1. monthly searches of the Cochrane Central Register of Controlled Trials (CENTRAL);
2. weekly searches of MEDLINE (Ovid);
3. weekly searches of Embase (Ovid);
4. monthly searches of CINAHL (EBSCO);
5. handsearches of 30 journals and the proceedings of major conferences;
6. weekly current awareness alerts for a further 44 journals plus monthly BioMed Central email alerts.

Search results are screened by two people and the full text of all relevant trial reports identified through the searching activities described above is reviewed. Based on the intervention described, each trial report is assigned a number that corresponds to a specific Pregnancy and Childbirth review topic (or topics), and is then added to the Register. The Information Specialist searches the Register for each review using this topic number rather than keywords. This results in a more specific search set which has been fully accounted for in the relevant review sections ([Included studies](#); [Excluded studies](#); [Studies awaiting classification](#); [Ongoing studies](#)).

### Searching other resources

We searched reference lists of retrieved articles. We did not apply any language or date restrictions.

### Data collection and analysis

For methods used in the previous version of this review, see [Tieu 2008](#).

For this update, the following methods were used for assessing the reports that were identified as a result of the updated search. Where required, information pertaining to the previously included studies was updated according to methods outlined in the *Cochrane Handbook for Systematic Reviews of Interventions* ([Higgins 2011](#)).

### Selection of studies

Two review authors independently assessed for inclusion all the potential studies identified as a result of the search strategy. We resolved any disagreement through discussion or, if required, we consulted the third review author.

### Data extraction and management

At least two review authors extracted the data using the agreed form. We resolved discrepancies through discussion or, if required, we consulted the third review author. Data were entered into Review Manager software ([RevMan 2014](#)) and checked for accuracy. When information regarding any of the above was unclear, we contacted authors of the original reports to provide further details.

### Assessment of risk of bias in included studies

Two review authors independently assessed risk of bias for each study using the criteria outlined in the *Cochrane Handbook for Systematic Reviews of Interventions* ([Higgins 2011](#)). Any disagreement was resolved by discussion or by involving a third assessor.

#### (1) Random sequence generation (checking for possible selection bias)

We described for each included study the method used to generate the allocation sequence in sufficient detail to allow an assessment of whether it should produce comparable groups.

We assessed the method as:

- low risk of bias (any truly random process, e.g. random number table; computer random number generator);
- high risk of bias (any non-random process, e.g. odd or even date of birth; hospital or clinic record number);
- unclear risk of bias.

#### (2) Allocation concealment (checking for possible selection bias)

We described for each included study the method used to conceal allocation to interventions prior to assignment and assessed whether intervention allocation could have been foreseen in advance of, or during recruitment, or changed after assignment.

We assessed the methods as:

- low risk of bias (e.g. telephone or central randomisation; consecutively numbered sealed opaque envelopes);
- high risk of bias (open random allocation; unsealed or non-opaque envelopes, alternation; date of birth);

- unclear risk of bias.

### **(3.1) Blinding of participants and personnel (checking for possible performance bias)**

We described for each included study the methods used, if any, to blind study participants and personnel from knowledge of which intervention a participant received. We considered that studies were at low risk of bias if they were blinded, or if we judged that the lack of blinding was unlikely to affect results. We assessed blinding separately for different outcomes or classes of outcomes.

We have assessed the methods as:

- low, high or unclear risk of bias for participants;
- low, high or unclear risk of bias for personnel.

### **(3.2) Blinding of outcome assessment (checking for possible detection bias)**

We described for each included study the methods used, if any, to blind outcome assessors from knowledge of which intervention a participant received. We assessed blinding separately for different outcomes or classes of outcomes.

We have assessed methods used to blind outcome assessment as:

- low, high or unclear risk of bias.

### **(4) Incomplete outcome data (checking for possible attrition bias due to the amount, nature and handling of incomplete outcome data)**

We described for each included study, and for each outcome or class of outcomes, the completeness of data including attrition and exclusions from the analysis. We stated whether attrition and exclusions were reported and the numbers included in the analysis at each stage (compared with the total randomised participants), reasons for attrition or exclusion where reported, and whether missing data were balanced across groups or were related to outcomes. Where sufficient information was reported, or could be supplied by the study authors, we planned to re-include missing data in the analyses which we undertook.

We assessed methods as:

- low risk of bias (e.g. no missing outcome data; missing outcome data balanced across groups);
- high risk of bias (e.g. numbers or reasons for missing data imbalanced across groups; 'as treated' analysis done with substantial departure of intervention received from that assigned at randomisation);
- unclear risk of bias.

### **(5) Selective reporting (checking for reporting bias)**

We described for each included study how we investigated the possibility of selective outcome reporting bias and what we found. We assessed the methods as:

- low risk of bias (where it was clear that all of the study's pre-specified outcomes and all expected outcomes of interest to the review were reported);

- high risk of bias (where not all the study's pre-specified outcomes were reported; one or more reported primary outcomes were not pre-specified; outcomes of interest were reported incompletely and so could not be used; study failed to include results of a key outcome that would have been expected to have been reported);

- unclear risk of bias.

### **(6) Other bias (checking for bias due to problems not covered by (1) to (6) above)**

We described for each included study any important concerns we had about other possible sources of bias.

### **(7) Overall risk of bias**

We made explicit judgements about whether studies were at high risk of bias, according to the criteria given in the *Handbook* (Higgins 2011). With reference to (1) to (6) above, we assessed the likely magnitude and direction of the bias and whether we considered it was likely to impact on the findings. We explored the impact of the level of bias through undertaking sensitivity analyses - see *Sensitivity analysis*.

### **Assessment of the quality of the evidence using the GRADE approach**

For this update, we evaluated the quality of the evidence for two of our three comparisons using the GRADE approach as outlined in the [GRADE handbook](#). The GRADE approach uses five considerations (study limitations, consistency of effect, imprecision, indirectness and publication bias) to assess the quality of the body of evidence for specific outcomes. The evidence can be downgraded from 'high quality' by one level for serious (or by two levels for very serious) limitations, depending on assessments for risk of bias, indirectness of evidence, inconsistency, imprecision of effect estimates or publication bias. In this review we planned to use the GRADE approach to assess the primary outcomes, as follows. We used the Cochrane Pregnancy and Childbirth GRADE core outcome set for reviews of prevention and treatment of gestational diabetes mellitus (GDM) and pre-existing diabetes in pregnancy.

#### **For the mother**

#### **Perinatal outcomes**

- GDM
- Hypertensive disorders of pregnancy (e.g. pre-eclampsia, pregnancy-induced hypertension, eclampsia)
- Caesarean section birth



- Perineal trauma
- Gestational weight gain

### Longer-term maternal outcomes

- Postnatal depression
- Type 2 diabetes mellitus

### For the child

#### Fetal/neonatal outcomes

- Perinatal mortality (stillbirth or neonatal mortality)
- Large-for-gestational age
- Mortality or morbidity composite (e.g. death, shoulder dystocia, bone fracture or nerve palsy)
- Neonatal hypoglycaemia

#### Childhood/adulthood outcomes

- Childhood/adulthood adiposity (e.g. as measured by BMI, skinfold thickness)
- Childhood/adulthood type 2 diabetes mellitus
- Childhood/adulthood neurosensory disability

### 'Summary of findings' table

We used [GRADEpro](#) Guideline Development Tool to import data from Review Manager 5.3 ([RevMan 2014](#)) in order to create 'Summary of findings' tables for two of the three comparisons in this review: dietary advice versus standard care, and low-GI dietary advice versus moderate- to high-GI dietary advice. We produced separate tables for maternal and child outcomes. Summaries of the intervention effect and measures of quality according to the GRADE approach are presented in the 'Summary of findings' tables.

### Measures of treatment effect

#### Dichotomous data

For dichotomous data, we presented results as summary risk ratio with 95% confidence intervals.

#### Continuous data

For continuous data, we used the mean difference. We planned to use the standardised mean difference to combine trials that measured the same outcome, but used different methods.

### Unit of analysis issues

#### Cluster-randomised trials

We did not identify any cluster-randomised trials for inclusion in this review.

If cluster-randomised trials are included in future reviews, we plan to include these trials in the analyses along with individually-randomised trials. Their sample sizes will be adjusted using the methods described in the *Handbook* ([Higgins 2011](#)) using an estimate of the intra-cluster correlation co-efficient (ICC) derived from the trial (if possible), or from another source. If ICCs from other sources are used, we will report this and conduct sensitivity analyses to investigate the effect of variation in the ICC. If we identify both cluster-randomised trials and individually-randomised trials, we plan to synthesise the relevant information. We consider it reasonable to combine the results from both types of studies if there is little heterogeneity between the study designs and the interaction between the effect of intervention and the choice of randomisation unit is considered to be unlikely. We plan also to acknowledge heterogeneity in the randomisation unit and perform a sensitivity analysis to investigate the effects of the randomisation units.

#### Other unit of analysis issues

#### Cross-over trials

We excluded trials with cross-over designs.

#### Multiple pregnancies

We did not identify any eligible studies that included a notable proportion of multiple pregnancies. [Laitinen 2009](#) reported one twin pregnancy in the dietary advice intervention group (any twin pairs were excluded from growth follow-up in this study). If studies with multiple pregnancies are included in trials included in future updates of this review, we will adjust for clustering in the analyses wherever possible, and use the inverse variance method for adjusted analyses, as described in the *Cochrane Handbook for Systematic Reviews of Interventions* ([Higgins 2011](#)) and in [Yelland 2011](#).

#### Multi-armed trials

We included one multi-armed trial ([Laitinen 2009](#)). We created a single pair-wise comparison, by including only the two treatment groups relevant to this review.

#### Dealing with missing data

For included studies, we noted levels of attrition. In future updates, if more eligible studies are included, the impact of including studies with high levels of missing data in the overall assessment of treatment effect will be explored by using sensitivity analyses.

For all outcomes, analyses were carried out, as far as possible, on an intention-to-treat basis, i.e. we attempted to include all participants randomised to each group in the analyses. The denominator for each outcome in each trial was the number randomised minus any participants whose outcomes were known to be missing.

### Assessment of heterogeneity

We assessed statistical heterogeneity in each meta-analysis using the Tau<sup>2</sup>, I<sup>2</sup> and Chi<sup>2</sup> statistics. We regarded heterogeneity as substantial if an I<sup>2</sup> was greater than 30% and either the Tau<sup>2</sup> was greater than zero, or there was a low P value (less than 0.10) in the Chi<sup>2</sup> test for heterogeneity. Where we identified substantial heterogeneity (above 30%), we aimed to explore it using pre-specified subgroup analyses.

### Assessment of reporting biases

Had there been 10 or more studies in a meta-analysis, we planned to investigate reporting biases (such as publication bias) using funnel plots. We planned to assess funnel plot asymmetry visually. If asymmetry was suggested by a visual assessment, we planned to perform exploratory analyses to investigate it.

### Data synthesis

We carried out statistical analysis using the Review Manager software (RevMan 2014). We used fixed-effect meta-analysis for combining data where it was reasonable to assume that studies were estimating the same underlying treatment effect: i.e. where studies were examining the same intervention, and the studies' populations and methods were judged sufficiently similar.

Where there was clinical heterogeneity sufficient to expect that the underlying treatment effects differed between trials, or where substantial statistical heterogeneity was detected, we used random-effects meta-analysis to produce an overall summary, if an average treatment effect across trials was considered clinically meaningful. The random-effects summary has been treated as the average of the range of possible treatment effects and we have discussed the clinical implications of treatment effects differing between trials. If the average treatment effect was not clinically meaningful, we did not combine trials. Where we used random-effects analyses, the results have been presented as the average treatment effect with 95% confidence intervals, and the estimates of Tau<sup>2</sup> and I<sup>2</sup>.

We performed separate comparisons for different types of interventions. We planned to consider separately, where possible: studies comparing dietary advice interventions with no dietary advice (i.e. standard care); and those studies comparing two different types of dietary advice interventions.

### Subgroup analysis and investigation of heterogeneity

Where we identified substantial heterogeneity, we planned to investigate it using subgroup and sensitivity analyses. We planned

to consider whether an overall summary was meaningful, and if it was, we used random-effects analysis to produce it. Maternal characteristics, and characteristics of the dietary advice interventions are likely to affect health outcomes. Therefore, we planned to carry out the following subgroup analyses for our primary outcomes.

#### Maternal characteristics

- Age at or before trial entry (e.g. < 35 years versus ≥ 35 years of age).
- BMI at or before trial entry (e.g. < 25 kg/m<sup>2</sup> versus ≥ 25 to < 30 kg/m<sup>2</sup> versus ≥ 30 kg/m<sup>2</sup>).
- Ethnicity (high-risk ethnicity versus low-risk ethnicity).

#### Characteristics of the interventions

- Frequency (e.g. frequent versus infrequent intervention).
- Duration (e.g. short versus long duration of intervention).
- Intensity (e.g. advice only versus more intensive support).

We planned to assess subgroup differences by interaction tests available within RevMan (RevMan 2014), and to report the results of subgroup analyses quoting the Chi<sup>2</sup> statistic and P value, and the interaction test I<sup>2</sup> value.

However, due to the paucity of data in this review, across three comparisons (only two with data for some of the primary outcomes), we were unable to conduct the majority of planned subgroup analyses in this version of the review, except for 'according to BMI at or before trial entry' for Comparison 1, where three of the six included trials included only overweight or obese women.

#### Sensitivity analysis

We carried out sensitivity analyses to explore the effects of trial quality assessed by sequence generation and allocation concealment, by omitting studies rated as 'high risk of bias' or 'unclear risk of bias' for these components. We restricted this to the primary outcomes.

## RESULTS

### Description of studies

#### Results of the search

In the previous version of the review (Tieu 2008), 15 reports, relating to nine studies were identified. We included three trials (eight reports) (Clapp 1998; Fraser 1983; Moses 2006), excluded one study (two reports) (Fraser 1988), one study (one report) was

awaiting further classification, and four studies (four reports) were ongoing.

The updated search of Cochrane Pregnancy and Childbirth's Trials Register in January 2016 identified a further 108 reports, and we identified a further three reports by contacting trial authors, and accessing reports cited on trial registrations web sites.

In total, we included 11 trials (54 records) in this update (Clapp 1998; Fraser 1983; Laitinen 2009; Markovic 2016; Moses 2006; Moses 2014; Quinlivan 2011; Thornton 2009; Vitolo 2011; Walsh 2012; Wolff 2008), and excluded 31 studies (62 records) (Althuisen 2013; Asbee 2009; Asemi 2013; Brand-Miller 2007; Dodd 2014; Facchinetti 2013; Fraser 1988; Hui 2006; King 2013; Korpi-Hyovalti 2012; Krummel 2009; Laitinen 2015; Lesser 2015; Lindsay 2014; Liu 2013; Luoto 2011; Maitland 2014; Matarrelli 2013; Mike O'Callaghan Federal Hospital 2011; Min 2014; Hellenes 2015; Moses 2009; Phelan 2011; Phelan 2016; Poston 2015; Reyes-Munoz 2014; Rhodes 2010; Taghizadeh 2014; Vesco 2014; Yap 2014; Zhou 2011).

Four studies (five records) are awaiting further classification (Angel 2011; Parat 2015; Simmons 2015; Zhang 2015), and five studies (five records) were identified as ongoing (NCT01056406; NCT01105455; NCT01628835; NCT01894139; NCT02218931).

The four studies awaiting classification have been published in abstract format only, with limited information regarding methods, intervention and outcomes provided to date; they assessed: a low glycaemic load diet versus a low fat diet in 64 overweight and obese pregnancy women in the USA (Angel 2011); individual and group dietary counselling sessions versus standard care in 268 overweight and obese pregnancy women in France (Parat 2015); face-to-face and telephone coaching sessions focused on healthy eating (versus coaching sessions focused on physical activity or

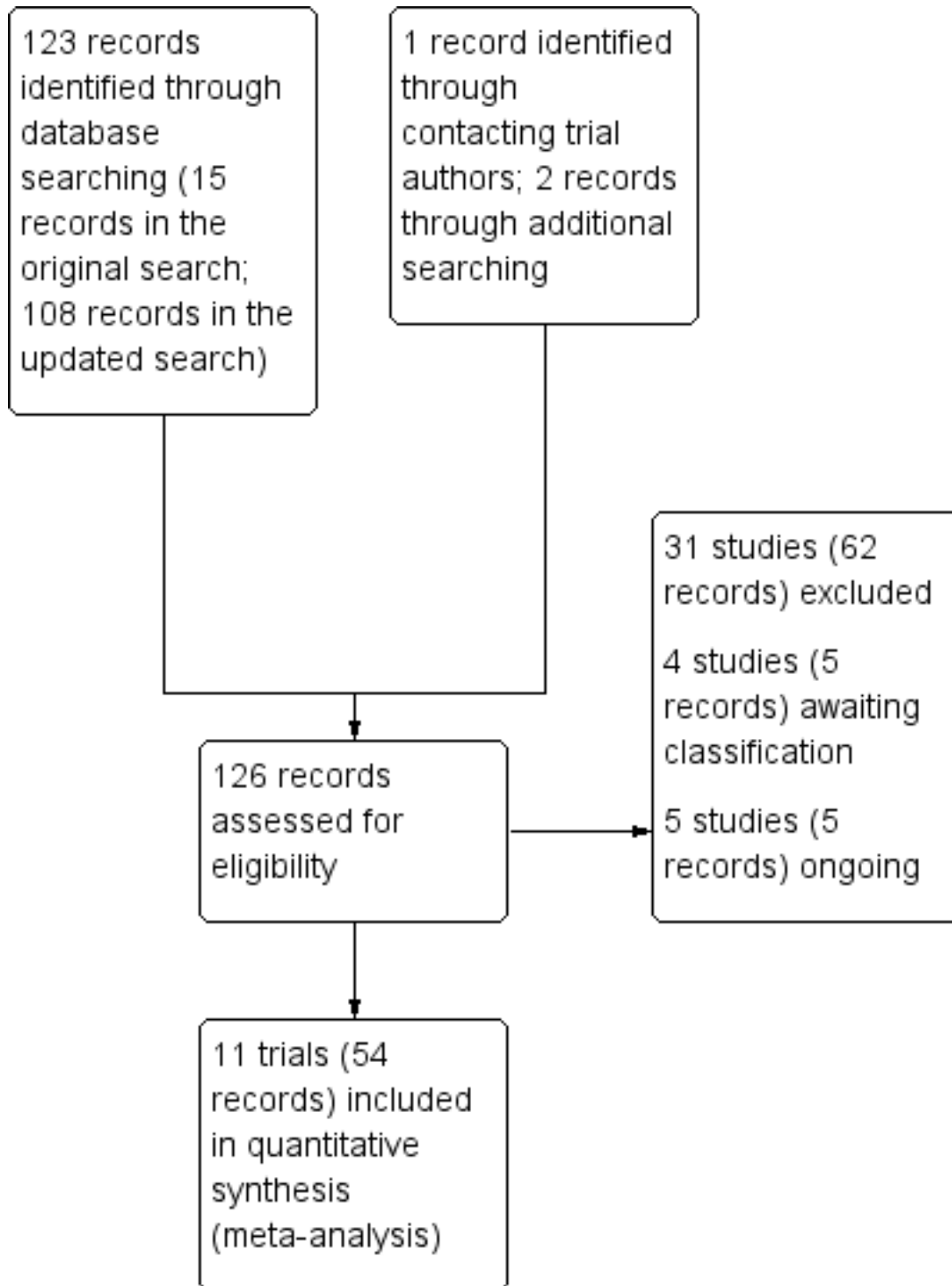
both healthy eating and physical activity) in 146 women at risk of gestational diabetes mellitus (GDM) in Europe (Simmons 2015); and 'medical nutrition guidance' in 261 pregnant women (country not reported) (Zhang 2015).

The five ongoing studies are being undertaken in the USA (NCT01056406), Canada (NCT01105455), China (NCT01628835), Denmark (NCT01894139) and the UK (NCT02218931), with interventions being assessed including:

- twice-monthly interaction with a registered dietitian from six to 16 weeks' gestation until six months postpartum versus no intervention (NCT01056406);
- group nutrition classes supplemented by handouts and provision of key study foods versus a leaflet with advice regarding a high-fibre diet (NCT01105455);
- four diet sessions at baseline, the end of the first trimester, the second trimester and the third trimester, including dietary assessments and consultations specifically recommending a low glycaemic index (GI) diet versus routine dietary advice (NCT01628835);
- a dietary and advice intervention recommending a high protein, especially marine and dairy protein and low-GI diet versus a diet according to the Nordic Nutritional Recommendations (NCT01894139);
- targeted advice intervention based on Mediterranean dietary pattern, including structured meal plans and grocery lists, recipes for healthy diet and appropriate choices at restaurants versus usual antenatal dietary advice (NCT02218931).

For further details see [Figure 1; Characteristics of included studies; Characteristics of excluded studies; Characteristics of ongoing studies; and Characteristics of studies awaiting classification](#) for further details.

Figure 1. Study flow diagram.



## Included studies

The 11 trials in this review involved a total of 2786 women and their babies, ranging from [Clapp 1998](#) and [Fraser 1983](#), with only 20 and 25 women respectively, to [Moses 2014](#) and [Walsh 2012](#) with 691 and 800 women respectively. These 11 trials were conducted across a variety of countries including four in Australia ([Markovic 2016](#); [Moses 2006](#); [Moses 2014](#); [Quinlivan 2011](#)), two in the USA ([Clapp 1998](#); [Thornton 2009](#)), and one each in Brazil ([Vitolo 2011](#)), Denmark ([Wolff 2008](#)), Finland ([Laitinen 2009](#)), Ireland ([Walsh 2012](#)), and the UK ([Fraser 1983](#)).

The included trials compared a variety of dietary interventions, and thus have been organised into three comparison: dietary advice interventions versus standard care; low-GI dietary advice versus moderate- to high-GI dietary advice; and high-fibre dietary advice versus standard dietary advice. Six trials compared a dietary advice intervention with standard care ([Laitinen 2009](#); [Quinlivan 2011](#); [Thornton 2009](#); [Vitolo 2011](#); [Walsh 2012](#); [Wolff 2008](#)); four trials compared low- and moderate- to high-GI dietary advice ([Clapp 1998](#); [Markovic 2016](#); [Moses 2006](#); [Moses 2014](#)); and one trial compared specific dietary advice (high-fibre focused) and standard dietary advice ([Fraser 1983](#)).

### 1) Dietary advice interventions versus standard care

As mentioned above, six trials were included in this arm of the review ([Laitinen 2009](#); [Quinlivan 2011](#); [Thornton 2009](#); [Vitolo 2011](#); [Walsh 2012](#); [Wolff 2008](#)).

### Participants

Three of the six trials included women who were overweight or obese: [Quinlivan 2011](#) included women with a singleton pregnancy, who were overweight (body mass index (BMI) 25 to 29.9) or obese (BMI > 29.9); [Thornton 2009](#) included 257 women (BMI ≥ 30). Women with pre-existing diabetes, hypertension, or chronic renal disease) with a singleton fetus between 12 and 28 weeks' gestation who were obese were excluded. [Wolff 2008](#) included 73 women with a singleton pregnancy, early in their pregnancy (mean: 15 + 3 weeks' gestation), who were obese (BMI ≥ 30), and excluded women who smoked or had any medical complications known to affect fetal growth adversely or to indicate limitation of weight gain.

The other three trials had varying inclusion criteria: [Laitinen 2009](#) included 256 women with singleton or twin pregnancies at less than 17 weeks' gestation with no metabolic or chronic diseases; [Vitolo 2011](#) included 315 women between 10 and 29 weeks' gestation (excluding those who had previously been diagnosed with diabetes, who had hypertension, anaemia or other conditions requiring a special diet); and the ROLO trial ([Walsh 2012](#)) included

800 women, who were secunda gravida with singleton pregnancies less than 18 weeks' gestation, with first infants weighing more than 4000 g at birth, and excluded women with any underlying medical disorders or history of GDM.

### Interventions

Although all six trials compared dietary advice with standard care, a range of specific dietary interventions were assessed. In general, women were counselled with regards to common healthy eating strategies, though resources used and protocols followed by each trial varied.

Women in the dietary advice intervention group of [Wolff 2008](#) received a total of 10, one-hour consultations with a dietitian. Advice was based on Danish dietary recommendations (fat intake: max 30 energy percent (E%), protein intake: 15 to 20 E%, carbohydrate intake: 50 to 55 E%), and individualised intake restrictions were based on each woman's energy requirements and the estimated energetic cost of fetal growth.

In [Laitinen 2009](#), women in the dietary advice intervention group received counselling by a dietitian at each study visit, which occurred three times during pregnancy (mean of 13, 24 and 34 weeks' gestation) and at one, six and 12 months postpartum, which aimed to modify dietary intake to conform with Nordic Nutrition Recommendations (with a particular focus on quality of dietary fat: saturated fatty acids providing 10% or less of energy intake, monounsaturated 10% to 15% and polyunsaturated fatty acids 5% to 10%). Women in the dietary advice intervention group were also provided with food products of favourable fat composition and fibre contents.

Women in the dietary advice intervention group of [Walsh 2012](#) attended a two-hour group dietary session (mean: 15.7 weeks' gestation) in groups of two to six with a research dietitian, where women received advice on general healthy eating guidelines for pregnancy; with women encouraged to choose as many low-GI foods as possible, but not to reduce their total caloric intake. Women then received two further dietary intervention sessions with the research dietitian at 28 and 34 weeks' gestation who reinforced the low-GI diet, and answered any questions.

[Quinlivan 2011](#) used Australian guidelines from the National Health and Medical Research Council for healthy eating as the basis of the dietary advice intervention which utilised a 'four-step multidisciplinary approach.' For women in the dietary advice intervention group, this approach, at a study-specific antenatal clinic, included 1) continuity of care by a single maternity provider; 2) assessment of weight gain at each antenatal visit; 3) a brief intervention (five minutes) by a food technologist before each visit; 4) an assessment by a clinical psychologist with intervention if required.

In [Thornton 2009](#), women in the dietary advice intervention group were 'nutritionally monitored'. They were prescribed a nutritionally balanced diet based on their weight at study entry and were asked to record all food and drink consumed each day in a food diary and these records were reviewed at each antenatal visit by the physician. Women in both groups were counselled at least once by a dietitian regarding conventional nutrition guidelines, however the intervention group received more detailed intake advice (recommendations: 18 to 24 kcal/kg (at least 2000 calories) consisting of 40% carbohydrates, 30% protein and 30% fat).

Women in the dietary advice intervention group in [Vitolo 2011](#) received dietary counselling according to their baseline nutritional status (assessed to be low weight, eutrophic or overweight), at an initial session, with reinforcement once a month thereafter. Advice in this trial aimed to improve the quality of food consumed in addition to augmenting the speed of weight gain during pregnancy. The control groups in the trials received standard care without additional dietary advice at the hospital/clinic they attended ([Quinlivan 2011](#); [Laitinen 2009](#); [Vitolo 2011](#); [Walsh 2012](#); [Wolff 2008](#)). In [Laitinen 2009](#) "All women attended communal dietary counselling provided by welfare clinics according to a national program, which consists of information of dietary guidelines through conversations and written material mediated by educated nurses," and in [Thornton 2009](#), all women in the standard care group were counselled at least once by a registered dietitian regarding conventional nutrition guidelines. Three of the trials ([Laitinen 2009](#), [Walsh 2012](#); [Wolff 2008](#)) asked women in both dietary advice intervention and standard care groups to provide information on their dietary intake through food diaries/records including at baseline, and in the second and/or third trimesters.

Both dietary advice intervention and standard care groups in [Wolff 2008](#) were given supplements to ensure adequate vitamin and trace element intake, particularly iron and folic acid.

[Laitinen 2009](#) also assessed the effects of probiotics, necessitating a third study group, not included in this review, who received dietary counselling in addition to a probiotic. Women in both the dietary advice and standard care groups of [Laitinen 2009](#), included in this review, received placebo capsules (containing microcrystalline cellulose and dextrose anhydrate), from their first study visit until the end of exclusive breastfeeding.

### GDM diagnosis

In [Laitinen 2009](#), a 75 g oral glucose tolerance test (OGTT) was used, and the diagnosis of GDM was based on modified criteria of the Fourth International Workshop Conference on Gestational Diabetes Mellitus. Specifically, GDM was diagnosed when one plasma glucose concentration exceeded  $\geq 4.8$  mmol/L at baseline,  $\geq 10.0$  mmol/L at one hour or  $\geq 8.7$  mmol/L at two hours.

[Quinlivan 2011](#) similarly used a 75 g OGTT, with the diagnosis of GDM based on World Health Organization Criteria; specifically, GDM was diagnosed when the two-hour plasma glucose

concentration was  $> 7.7$  mmol/L.

In [Walsh 2012](#), a 50 g oral glucose challenge test (OGCT) was used at 28 weeks' gestation, and women with a one-hour blood glucose concentration  $\geq 8.3$  mmol/L underwent an OGTT; GDM was diagnosed if two or more abnormal results were observed during a three-hour, 100 g OGTT diagnosed according to the Carpenter and Coustan criteria (fasting  $\geq 5.3$  mmol/L, one-hour  $\geq 10.0$  mmol/L, two-hour  $\geq 8.6$  mmol/L, three-hour  $\geq 7.8$  mmol/L). [Walsh 2012](#) reported GDM diagnosed according American Diabetes Association criteria (GDM diagnosed when any one of the following plasma glucose concentrations were exceeded: fasting  $\geq 5.1$  mmol/L, one-hour  $\geq 10.0$  mmol/L, two-hour  $\geq 8.5$  mmol/L).

[Wolff 2008](#) reported that "Fasting blood glucose and blood glucose 2 h postprandial to an oral glucose tolerance test of a 50-g glucose load were analyzed", however did not report specific criteria for GDM diagnosis.

[Thornton 2009](#) did not report on the criteria used for GDM diagnosis, only detailing: "Antepartum and intrapartum complications such as development of gestational diabetes, ketonuria, preeclampsia, and shoulder dystocia were identified from the medical record after the patient delivered".

Similarly, [Vitolo 2011](#) did not report on the criteria for GDM diagnosis, rather reporting only on a composite outcome 'clinical complications' (which included GDM, pre-eclampsia, low birth-weight, prematurity).

### 2) Low GI-dietary advice versus moderate- to high-GI dietary advice

Four trials, involving 928 women and their babies were included in this comparison of the review ([Clapp 1998](#); [Markovic 2016](#); [Moses 2006](#); [Moses 2014](#)). All trials were conducted in hospital settings, three in Australia ([Markovic 2016](#); [Moses 2006](#); [Moses 2014](#)), and one in the USA ([Clapp 1998](#)).

### Participants

[Clapp 1998](#) included 20 healthy women preconception, "who eventually completed an uncomplicated pregnancy", who commenced a regular, supervised exercise and weight-maintaining diet of predominantly carbohydrates until they were randomised at eight weeks' gestation. [Moses 2006](#) included 70 healthy "white" pregnant women with a singleton pregnancy at 12 to 16 weeks' gestation (and specifically excluded women with any problems that may have been associated with glucose metabolism or insulin resistance or could interfere with their ability to follow dietary instructions). The PREGGIO trial ([Moses 2014](#)) included 691 women with a singleton pregnancy at less than 20 weeks' gestation and excluded women with known diabetes or previous GDM, special dietary needs or any medical conditions that may affect metabolic status or use of medication likely to affect body weight.

The GI Baby 3 trial (Markovic 2016), however, included 147 women, between 12 and 20 weeks' gestation, specifically at high risk of GDM (with at least one of the risk factors: aged older than 35 years; first degree relative with type 2 diabetes; pre-pregnancy BMI 30 or over; past history of GDM or glucose intolerance; history of a previous baby with birthweight over 4000 g; high-risk ethnic group), with an otherwise healthy singleton pregnancy.

### Interventions

Women in Clapp 1998 were randomised at eight weeks' gestation, to either a high-GI/cafeteria diet (with carbohydrates from highly processed grains, root vegetables, and simple sugars) or a low-GI/Aboriginal diet (with carbohydrates from unprocessed whole grains, fruits, beans and vegetables, and dairy products).

Women in both groups of Clapp 1998 were recommended diets with a composition of 17% to 19% protein, 20% to 25% fat and 55% to 60% carbohydrate, with a total intake of 35 to 45 kcal/kg of lean body mass. Adherence was assessed by random, twice weekly 24-hour dietary recalls by a dietitian.

In Markovic 2016, women in the low-GI dietary advice group had a target GI of 50 or less, while those in the high-fibre, moderate-GI dietary advice group had a target GI of 60; though all women were recommended a similar macronutrient composition, of 15% to 25% protein, 25% to 39% fat, and 40% to 45% carbohydrate. All women attended a total of five individual dietary consultations with a dietitian (at 14 to 20, 18 to 24, 22 to 28, 26 to 32, and 34 to 36 weeks' gestation); with visit one using a three-day food record as the basis of dietary counselling, and written information regarding low-GI/high-fibre foods and pregnancy nutrition provided; and visits two to four using four-stage multiple-pass 24-hour recalls, and suitable alternative foods encouraged for non-compliant women. All women in Markovic 2016 were also provided with food samples (supplementary baskets) containing key foods for their assigned diet at all five consultations.

Women in both groups of Moses 2006 were also seen by a dietitian five times during their pregnancy (who was also available for telephone queries outside of scheduled visits); at visit one, a three-day food record and diet history was taken; at visit two (a week later), women received dietary education for either a low-GI diet (verified low-GI foods including pasta, brand-name breads and breakfast cereals with high-fibre content) or a moderate- to high-GI diet (high fibre, low sugar foods including potatoes, wheat-meal bread, and specific high-fibre breakfast cereals with moderate- to high-GI); at visits three (22 weeks' gestation) and four (30 weeks' gestation), 24-hour diet recalls were taken, and at visit five (36 weeks) a further three day food record and diet history were taken. All women in Moses 2006 were advised to maintain a diet of 33% fat and 55% carbohydrate (with only the recommended choice of carbohydrate foods varying), were provided with a booklet that outlined the carbohydrate choices and the food amounts that constituted one serving, and were provided key foods in a

monthly hamper.

In Moses 2014, all women received a set of booklets; for women in the low-GI dietary advice group, counselling focused on choices for and serving sizes of carbohydrate-rich foods, with specific information provided on low-GI alternatives for relevant food groups; while for women in the healthy eating dietary advice group, counselling focused on a conventional healthy diet with recommended foods and serving sizes as noted in the Australian Guide to Healthy Eating and no specific guidance on GI was given. There was no intended difference between groups in the macronutrient distribution of the diet, and for all women in Moses 2014 there were four contact points; at the first, a three-day food record was reviewed, and diet education was given specific to the assigned group by the dietitian; at the second, a phone call was made (four weeks after the first visit), to ensure adherence to the diet and goals set and identify any barriers or concerns; at the third, a dietitian reviewed the women face-to-face (at 28 weeks) before their obstetric appointment to monitor progress; at the fourth (as late as possible, and at a minimum of 34 weeks' gestation), the dietitian collected and reviewed the final three-day food records. For all women in Moses 2014, the research dietitian was available for telephone queries outside of scheduled visits, and an email with nutrition tips and recipes was sent monthly (five in total) to all women with content dependent on group.

### GDM diagnosis

Clapp 1998 focused on biochemical outcomes (including glucose and insulin responses), and reported data relating to GDM as a result of "glucose screens, which were conducted as part of their clinical care at approximately 28 weeks' gestation" (OGCT). No further information regarding GDM diagnosis criteria was provided.

In Markovic 2016, GDM diagnosis was based on modified Australasian Diabetes in Pregnancy Society 1998 criteria: following a 75 g OGTT, at either study entry (between 14 and 20 weeks' gestation) or at 26 to 28 weeks' gestation, with GDM diagnosed where fasting blood glucose was  $\geq 5.5$  mmol/L, one-hour  $\geq 10.0$  mmol/L, or two-hour  $\geq 8.0$  mmol/L.

Moses 2006 reported that a routine OGTT was performed at 28 weeks' gestation. Specific diagnostic criteria used were not provided (no further information regarding GDM diagnosis criteria were provided).

In Moses 2014, all women were tested for GDM between 24 and 28 weeks' gestation; at the beginning of the study, the test was based on the Australasian Diabetes in Pregnancy Society criteria (with GDM diagnosed following a 75 g OGTT if: fasting plasma glucose  $\geq 5.5$  mmol/L, or two-hour  $\geq 8.0$  mmol/L); and, later, was based on new International Association of Diabetes in Pregnancy Study Group criteria (with GDM diagnosed following a 75 g OGTT if any of: fasting plasma glucose  $\geq 5.1$  mmol/L, one-hour  $\geq 10.0$  mmol/L, or two-hour  $\geq 8.5$  mmol/L). Women diagnosed with GDM in Moses 2014 were "withdrawn from the study and treated

*conventionally*”; we have however included data on these women in the relevant meta-analysis for GDM.

### 3) High-fibre dietary advice versus “*standard*” dietary advice

One trial was included in this comparison (Fraser 1983).

#### Participants

Fraser 1983 was a trial (conducted in a hospital in Sheffield, UK) of 25 primigravid European women in their second half of pregnancy, of ‘normal’ weight, and with no family history of diabetes; thus considered to be at low risk for GDM.

#### Interventions

Women in the intervention (high-dietary fibre) group of Fraser 1983 saw a dietitian at 27 weeks’ gestation, and were advised to reduce intake of sucrose and white flour, and to make as many high-fibre substitutions as possible; aiming for a calorie intake of 2400. Women were also given diet and recipe sheets, and tokens for free wholemeal bread. Women in the control (standard dietary advice) group of Fraser 1983 were given “*standard*” advice at an interview with a dietitian at 27 weeks’ gestation, with a suggested calorie intake of 2400 (no further details were provided regarding the “*standard*” dietary advice). Women in both groups were seen by the dietitian at each of their antenatal attendances.

#### GDM diagnosis

Fraser 1983 focused on biochemical outcomes (including glucose and insulin profiles), and thus reported only a limited number of review outcomes, including results of a 75 g OGTT at 35 weeks; however, GDM diagnosis was not reported.

#### Excluded studies

We excluded 31 studies from this review (Althuisen 2013; Asbee 2009; Asemi 2013; Brand-Miller 2007; Dodd 2014; Facchinetti 2013; Fraser 1988; Hui 2006; King 2013; Korpi-Hyovalti 2012;

Krummel 2009; Laitinen 2015; Lesser 2015; Lindsay 2014; Liu 2013; Luoto 2011; Maitland 2014; Matarrelli 2013; Mike O’Callaghan Federal Hospital 2011; Min 2014; Hellenes 2015; Moses 2009; Phelan 2011; Phelan 2016; Poston 2015; Reyes-Munoz 2014; Rhodes 2010; Taghizadeh 2014; Vesco 2014; Yap 2014; Zhou 2011).

Ten trials were excluded as they assessed a combined diet and exercise intervention for preventing GDM (Althuisen 2013; Asbee 2009; Dodd 2014; Hui 2006; Korpi-Hyovalti 2012; Luoto 2011; Phelan 2011; Phelan 2016; Poston 2015; Vesco 2014), which is the focus of another Cochrane review (Bain 2015), and one trial assessed diet interventions in overweight and obese women and did not report on GDM (Rhodes 2010) (it is included in another Cochrane review (Muktabhant 2015)). Three trials were excluded for assessing a probiotic intervention (Asemi 2013; Laitinen 2015; Taghizadeh 2014), which is the focus of another Cochrane review (Barrett 2014); three for assessing myo-inositol (Facchinetti 2013; Lindsay 2014; Matarrelli 2013), which is the focus of another Cochrane review (Brown 2015); and three for assessing docosahexaenoic acid (DHA) (Min 2014; Krummel 2009; Zhou 2011), which is the focus of another Cochrane review (Makrides 2006). One trial each was excluded for assessing an exercise intervention (Hellenes 2015), which is the focus of another Cochrane review (Han 2012), magnesium supplementation (Liu 2013), which is the focus of another Cochrane review (Makrides 2014); metformin (Reyes-Munoz 2014); and vitamin D (Yap 2014).

One trial was excluded as the participants were women with GDM (Moses 2009). Four studies were excluded as they were cross-over trials (Fraser 1988; King 2013; Lesser 2015; Maitland 2014), and two were excluded as they were not conducted (one did not proceed due to lack of funding [personal correspondence] (Brand-Miller 2007); and one, designed to assess folic acid for prevention of GDM, appeared to have been withdrawn prior to enrolment (Mike O’Callaghan Federal Hospital 2011)).

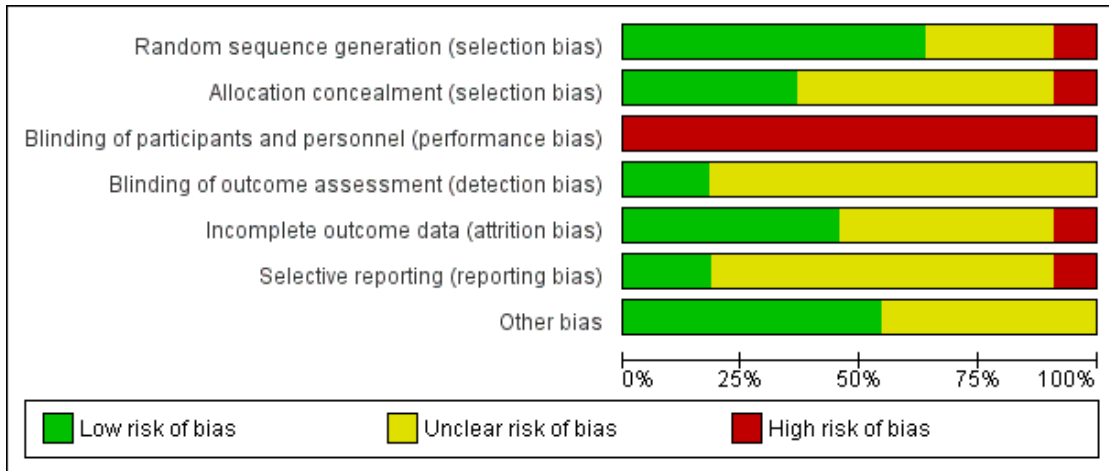
For further details see [Characteristics of excluded studies](#).

#### Risk of bias in included studies

Overall, the risk of bias was judged to be unclear to moderate - see [Characteristics of included studies](#) and [Figure 2](#) and [Figure 3](#) for further details.



**Figure 2. 'Risk of bias' graph: review authors' judgements about each risk of bias item presented as percentages across all included studies.**



**Figure 3. 'Risk of bias' summary: review authors' judgements about each risk of bias item for each included study.**

	Random sequence generation (selection bias)	Allocation concealment (selection bias)	Blinding of participants and personnel (performance bias)	Blinding of outcome assessment (detection bias)	Incomplete outcome data (attrition bias)	Selective reporting (reporting bias)	Other bias
Clapp 1998	?	?	-	?	+	?	?
Fraser 1983	?	?	-	?	?	-	?
Laitinen 2009	+	+	-	?	+	?	+
Markovic 2016	+	?	-	+	+	+	?
Moses 2006	-	-	-	?	?	?	?
Moses 2014	+	?	-	?	?	?	+
Quinlivan 2011	+	+	-	+	+	?	+
Thornton 2009	+	+	-	?	?	?	+
Vitolo 2011	?	?	-	?	?	?	?
Walsh 2012	+	+	-	?	+	+	+
Wolff 2008	+	?	-	?	-	?	+

## Allocation

Four trials were judged to be at a low risk of selection bias, with adequate methods for both random sequence generation and allocation concealment (Laitinen 2009; Quinlivan 2011; Thornton 2009; Walsh 2012). In three of these trials a computer-generated random number sequence was used (Laitinen 2009; Quinlivan 2011; Walsh 2012), and in the fourth one a random number table was used (Thornton 2009); and in each of the four trials, consecutively numbered, sealed, (opaque) envelopes were used. In three trials (Markovic 2016; Moses 2014; Wolff 2008), though adequate methods were reported for sequence generation (computer-generated random number sequences were used), methods for concealing allocation were not clearly specified.

For three trials (Clapp 1998; Fraser 1983; Vitolo 2011) the risk of selection bias was unclear; in Clapp 1998 no details regarding sequence generation or allocation concealment were provided, and in Fraser 1983 it was detailed only that sealed envelopes were opened after women had agreed to participate, however no detail of whether the envelopes were consecutively numbered or opaque was provided. In Vitolo 2011 (from the translation available) women “were randomized by means of a dark pouch with two equal sized cubes containing the term intervention in one and control in the other”.

The final trial, Moses 2006, was judged to be at a high risk of selection bias, as the trial was quasi-randomised (with allocation by alternation).

## Blinding

All 11 trials (Clapp 1998; Fraser 1983; Laitinen 2009; Markovic 2016; Moses 2006; Moses 2014; Quinlivan 2011; Thornton 2009; Vitolo 2011; Walsh 2012; Wolff 2008) were judged to be at a high risk of performance bias. While for a number of trials details on blinding were not explicit, due to the nature of the interventions it was presumed that it was unfeasible to blind participants and/or study personnel.

Two trials were judged to be low risk of detection bias (Markovic 2016; Quinlivan 2011). Markovic 2016 reported that “Apart from the study dietitian... who provided dietary education, all study personnel were blinded to dietary assignment;” and “A biostatistician blinded to the dietary allocation performed the statistical analysis”. In Quinlivan 2011 it was reported that outcome data for the mother and infant were ‘audited’ by a nurse, independent of clinical care, and blind to group allocation.

Nine of the 11 trials (Clapp 1998; Fraser 1983; Laitinen 2009; Moses 2006; Moses 2014; Thornton 2009; Vitolo 2011; Walsh 2012; Wolff 2008) were judged to be at an unclear risk of detection bias; while blinding of women and personnel was not possible due to the nature of the interventions, it was unclear in six studies as to whether it had been possible to blind outcome assessment

(Clapp 1998; Fraser 1983; Moses 2006; Moses 2014; Thornton 2009; Vitolo 2011). In Laitinen 2009, all personnel who handled or analysed blood samples were blind to the intervention, however no detail of whether other outcomes were assessed blinded was provided. Wolff 2008, reported that while women and dietitians were not blinded, women were asked not to reveal their group assignment to physicians and midwives, however it is unclear as to whether blinding of outcomes assessors would have been successfully achieved; and in Walsh 2012 it was reported only that “blinded sonographers made ultrasound measurements,” with no detail of blinding for other outcomes.

## Incomplete outcome data

Five trials were judged to be at low risk of attrition bias (Clapp 1998; Laitinen 2009; Markovic 2016; Quinlivan 2011; Walsh 2012). In Clapp 1998, there were no losses to follow-up and all women were analysed according to the group they were randomised, while in Quinlivan 2011, only four of 132 women withdrew from the study, and no other losses, attritions or exclusions were detailed. In Markovic 2016, only eight of the 147 women were excluded from the analyses (four in each group, for similar reasons), and in Walsh 2012, 41 of 800 women were excluded from the final analyses (with numbers and reasons for loss to follow-up/exclusions similar between groups). In Laitinen 2009, of the 256 mothers participating in the trial, 208 completed the one-year follow-up with reasons for discontinuation similar across the three groups.

Five trials were judged to be at unclear risk of attrition bias (Fraser 1983; Moses 2006; Moses 2014; Thornton 2009; Vitolo 2011), with no data provided regarding losses, or with unbalanced numbers/reasons for losses across groups. For example, Fraser 1983 did not report clearly on whether there were any missing data/losses; Thornton 2009 reported that 25 of 257 women were lost to follow-up, and there was some indication of higher loss in the control group (8/124 in the intervention group and 17/133 in the control group); and in Moses 2006, while data were provided for the 62/70 women who completed the study 19/62 women (30%) did not wish to participate in follow-up (those women had a similar BMI and age, but a higher parity than the 43 women who agreed to participate).

One trial, Wolff 2008, was judged to be at a high risk of attrition bias; of the 73 women “recruited to the study”, 50 (68%) were followed to birth, however additional data were missing for outcomes including weight measurement, without comment.

## Selective reporting

Only two of the 11 trials were judged to be at low risk of reporting bias (Markovic 2016; Walsh 2012), with no obvious risk of selective reporting.

For eight of the trials (Clapp 1998; Laitinen 2009; Moses 2006; Moses 2014; Quinlivan 2011; Thornton 2009; Vitolo 2011; Wolff 2008), the risk of reporting bias was judged to be unclear, largely due to insufficient information (such as access to trial registrations and/or trial protocols) available to confidently assess risk of selective reporting. In Clapp 1998, many results (such as for GDM and large-for-gestational age) were reported only narratively in text). It is not clear which outcomes were pre-specified. In Laitinen 2009, a number of outcomes not pre-specified in the manuscript methods were reported, such as breastfeeding and Apgar score at five minutes, and it is difficult to interpret and use infant data for outcomes such as birthweight, gestation, birth height, head circumference and Apgar at five minutes, as these data have been reported with 'ranges' of infants. In Moses 2006, some results were reported incompletely, quote: "The analysis of the diet histories produced similar findings (data not shown)". Moses 2014 included discrepancies between trial registration and published manuscript, with development of GDM listed as a primary outcome in the trial registration, but not reported as such in the manuscript. In Quinlivan 2011, data for all pre-specified outcomes (in the trial manuscript methods) were provided, however only three outcomes were pre-specified, and additional outcomes including changes in diet and serious adverse events were reported but not pre-specified in the methods. Thornton 2009 reported results for the majority of pre-specified outcomes, though there was no access to a trial protocol to assess selective reporting. In Vitolo 2011, the manuscript methods specify that data were obtained for outcomes such as newborn weight, length, gestational age, cephalic perimeter, Apgar scores at one and five minutes, mode of birth, gestational complications, diabetes and gestational hypertension; from the translation, data for these outcomes were not clearly reported. Wolff 2008 reported results for the majority of pre-specified outcomes in the trial manuscript methods, though no trial protocol or registration was available to assess selective reporting. Data were not provided for Apgar scores or 'methods of delivery' (data for caesarean birth were only reported).

One trial, Fraser 1983, was judged to be at high risk of reporting bias, with a general statement in text made for a number of outcomes and no data provided for these important outcomes; quote: "results of the antenatal monitoring (including maternal weight gain and serum ferritin) and fetal anthropometry showed no significant differences between the groups".

### Other potential sources of bias

Six trials were judged to be at a low risk of other bias, with no other obvious sources of bias identified (Laitinen 2009; Moses 2014; Quinlivan 2011; Thornton 2009; Walsh 2012; Wolff 2008).

The other five trials were judged to be at unclear risk of other

sources of bias (Clapp 1998; Fraser 1983; Markovic 2016; Moses 2006; Vitolo 2011). Clapp 1998 and Fraser 1983 provided limited methodological details to confidently assess other sources of bias, and Vitolo 2011 was assessed using a partial translation of the manuscript, making assessment of other sources of bias difficult; Markovic 2016 reported that while the majority of baseline characteristics were balanced across groups, family history of type 2 diabetes was more common in the low-GI group; in Moses 2006, it was reported that "Women who were assigned to the HGI diet group had a slightly higher BMI ( $P = 0.04$ ) and higher HOMA2-cell function ( $P = 0.07$ ) than did women in the LGI diet group".

### Effects of interventions

See: [Summary of findings for the main comparison](#) Dietary advice interventions versus standard care (maternal outcomes); [Summary of findings 2](#) Dietary advice interventions versus standard care (child outcomes); [Summary of findings 3](#) Low-GI dietary advice versus moderate- to high-GI dietary advice (maternal outcomes); [Summary of findings 4](#) Low-GI dietary advice versus moderate- to high-GI dietary advice (child outcomes)

### Comparison 1: Dietary advice interventions versus standard care

Six trials were included in this comparison (Laitinen 2009; Quinlivan 2011; Thornton 2009; Vitolo 2011; Walsh 2012; Wolff 2008).

### Primary outcomes

#### Gestational diabetes (GDM)

Overall, a trend towards a reduction in GDM was observed for women receiving dietary advice interventions compared with those receiving standard care (average risk ratio (RR) 0.60, 95% confidence interval (CI) 0.35 to 1.04; five trials, 1279 women;  $P = 0.07$ ; GRADE: *very low-quality evidence*) (Analysis 1.1). As there was substantial heterogeneity identified for this outcome ( $\text{Tau}^2 = 0.20$ ;  $I^2 = 56\%$ ), a random-effects meta-analysis was used.

Walsh 2012 reported on GDM according to two criteria (the Carpenter and Coustan criteria, and American Diabetes Association criteria). We have included the diagnoses according to the American Diabetes Association in this meta-analysis. Replacing these data with those relating to diagnoses according to the Carpenter and Coustan criteria (7/350 in the dietary advice intervention group and 9/371 in the standard care group) did not impact on the overall result.

### Hypertensive disorders of pregnancy

A reduction in pregnancy-induced hypertension was observed for women receiving dietary advice interventions compared with those receiving standard care (RR 0.30, 95% CI 0.10 to 0.88; two trials, 282 women; GRADE: *low-quality evidence*) (Analysis 1.2). No clear difference between groups was however observed for the risk of pre-eclampsia (RR 0.61, 95% CI 0.25 to 1.46; two trials, 282 women; GRADE: *low-quality evidence*) (Analysis 1.3).

### Perinatal mortality (stillbirth or neonatal mortality)

Only one trial (Laitinen 2009) reported on perinatal mortality, and there were no perinatal deaths in either the dietary advice interventions or standard care groups (GRADE: *very low-quality evidence*) (Analysis 1.4).

### Large-for-gestational age

None of the six trials in this comparison reported on the primary outcome: large-for-gestational age.

### Neonatal mortality or morbidity composite

None of the six trials in this comparison reported on the primary outcome: mortality or morbidity composite.

### Secondary outcomes

#### For the mother

#### Perinatal outcomes

No clear differences between the dietary advice interventions and standard care groups were seen for the following secondary outcomes: caesarean section (average RR 0.98, 95% CI 0.78 to 1.24; four trials, 1194;  $\text{Tau}^2 = 0.02$ ;  $I^2 = 36\%$ ; GRADE: *low-quality evidence*) (Analysis 1.5); induction of labour (average RR 1.10, 95% CI 0.48 to 2.51; two trials, 991 women;  $\text{Tau}^2 = 0.31$ ;  $I^2 = 87\%$ ) (Analysis 1.6); perineal trauma (RR 0.83, 95% CI 0.23 to 3.08; one trial, 759 women; GRADE: *very low-quality evidence*) (Analysis 1.7); postpartum haemorrhage (RR 0.71, 95% CI 0.28 to 1.86; two trials, 991 women) (Analysis 1.8); postpartum infection (RR 0.83, 95% CI 0.26 to 2.65; one trial, 232 women) (Analysis 1.9); breastfeeding at three months (RR 1.02, 95% CI 0.89 to 1.17; one trial, 452 women) (Analysis 1.10); or six months (RR 0.99, 95% CI 0.82 to 1.19; one trial, 146 women) (Analysis 1.11). Considerable statistical heterogeneity was observed in the meta-analysis for induction of labour, and thus the pooled result should be interpreted with some caution.

A reduction in gestational weight gain, was observed for women who received the dietary advice interventions (mean difference (MD) -4.70 kg, 95% CI -8.07 to -1.34; five trials, 1336 women;  $\text{Tau}^2 = 13.64$ ;  $I^2 = 96\%$ ; GRADE: *low-quality evidence*) (Analysis 1.12). Considerable statistical heterogeneity was observed in the meta-analysis for gestational weight gain, and thus the pooled result should be interpreted with some caution. Vitolo 2011 also reported on weekly gestational weight gain, however separately for women who were of 'low weight', were 'eutrophic' or were of 'excess weight.' Women of 'excess weight' in the dietary advice group had less weekly gestational weight gain, compared with women in the standard care group ( $P = 0.01$ ); no clear differences between groups were seen for the 'low weight' or 'eutrophic' women (Analysis 1.13). As the numbers in each group were not clearly reported, these data have not been included in the meta-analysis. Few behaviour changes were observed at three months postpartum for women in the dietary advice intervention group of one trial (Walsh 2012); more women were likely to be consuming a low-GI diet (RR 5.37, 95% CI 1.93 to 14.89; 197 women), and were reading food labels (RR 1.11, 95% CI 1.01 to 1.23; 453 women), specifically the nutrients (RR 1.21, 95% CI 1.03 to 1.41; 453 women) (Analysis 1.14). At three months postpartum, there were no clear differences between groups, however, for having a weight reducing diet (RR 0.95, 95% CI 0.66 to 1.38; 458 women), taking supplements (RR 1.12, 95% CI 0.98 to 1.28; 459 women), having made dietary changes since the study (RR 1.12, 95% CI 0.98 to 1.29; 420 women), and reading ingredients (RR 1.12, 95% CI 0.91 to 1.37; 453 women), calories (RR 1.08, 95% CI 0.89 to 1.31; 453 women), food weight (RR 1.51, 95% CI 0.90 to 2.54; 453 women), additives (RR 1.33, 95% CI 0.99 to 1.79; 453 women), or serving size (RR 1.39, 95% CI 0.83 to 2.34; 453 women), or in attending the gym (RR 0.98, 95% CI 0.65 to 1.49; 440 women) (Analysis 1.14).

A second trial (Quinlivan 2011) also reported on changes in diet associated with the intervention for the dietary advice intervention group only: "the intervention was associated with changes in diet, as recorded by the food technologist at every visit where women were asked to itemise their food consumption of the previous day... The intervention resulted in increased consumption of water, fresh fruit and vegetables and home-cooked meals. It was associated with a reduction in consumption of carbonated 'fizzy' drinks and juices and fast foods (frozen and fresh)".

While in one trial, no clear difference in fasting blood glucose concentrations (MD -0.06 mmol/L, 95% CI -0.13 to 0.01; 759 women) (Analysis 1.15) including fasting hyperglycaemia ( $\geq 5.1$  mmol/L) (RR 0.64, 95% CI 0.40 to 1.04; 673 women) (Analysis 1.16), and blood glucose concentrations following OGCT at 28 weeks' gestation were observed (MD -0.20 mmol/L, 95% CI -0.42 to 0.02; 759 women) (Analysis 1.17), there were fewer women with a OGCT  $> 7.8$  mmol/L (RR 0.72, 95% CI 0.53 to 0.99; 721 women) (Analysis 1.18), and fewer with a fasting glucose concentration  $\geq 5.1$  mmol/L or OGCT  $> 7.8$  mmol/L at 28 weeks'

gestation (RR 0.74, 95% CI 0.56 to 0.97; 672 women) (Analysis 1.19) in the same trial.

Wolff 2008 also reported that “The fasting glucose concentration in the control group had a small decrease with advancing gestational age,  $-0.04\text{mmol-1}$  ( $-0.2$  to  $0.2$ ) at 27 weeks of gestation, and  $-0.1\text{mmol-1}$  ( $-0.3$  to  $0.1$ ) at week 36. The intervention showed no reduction of the fasting glucose at 27 weeks, but at 36 weeks the fasting glucose was significantly reduced by 8%, group difference  $-0.3\text{ng ml-1}$  ( $-0.6$  to  $-0.0$ ,  $P=0.03$ ). No differences were obtained between intervention and control group in the 2-h glucose concentration after oral glucose tolerance test at 27 and 36 weeks of gestation,  $0.1\text{ng ml-1}$  ( $-0.6$  to  $0.8$ ,  $P=0.852$ ) and  $0.3\text{ng ml-1}$  ( $-0.4$  to  $1.0$ ,  $P=0.406$ )”.

In one trial, well-being was assessed using the World Health Organization well-being index (expressed as a percentage score), a five-item scale used to rate quality of life and psychological well-being, giving an overall score of nought to 25 which is converted to a percentage well-being score (Walsh 2012). Women in the dietary advice intervention group had a lower sense of well-being score, assessed by questionnaire between 14 and 28 weeks' gestation (MD  $-3.60$ , 95% CI  $-5.98$  to  $-1.22$ ; 618 women) (Analysis 1.20).

In regards to adherence with the intervention:

Laitinen 2009 reported for the dietary advice intervention group “According to the interviews, the proportion of women who consumed the food products provided for each 12-week period between study visits ranged from 68% to 100% depending on the product...However, as assessed by 3-day food records filled in immediately before the study visits, fewer women (39-81%) had, except for spreads, consumed the provided food products”.

Thornton 2009 reported that 77.6% (90/116) of women in the dietary advice intervention group were adherent with the prescribed nutritional regimen.

Walsh 2012 reported that “Almost 80% ( $n=294$ ) of the intervention arm reported following the low glycaemic index dietary advice either all or most of the time on the adherence questionnaire”.

Walsh 2012 reported on women's views, for women in the dietary advice intervention group: “Results from the compliance and acceptability questionnaires showed that 68% of women either agreed or strongly agreed that the diet was easy to follow. Sixty five percent of women agreed that they enjoyed making the changes to their diets, 72% of women reported that their family were happy with the changes they made to their diets, and 78% of women agreed/strongly agreed that they had enough energy while on the diet. Finally, over 80% of women reportedly enjoyed a wide variety of foods while following the diet”.

The trials in this comparison did not report on the outcomes: operative vaginal birth; placental abruption.

### Longer-term maternal outcomes

While in three trials, no clear differences were seen for postpartum weight loss at six weeks (MD  $-0.90\text{kg}$ , 95% CI  $-3.67$  to  $1.87$ ; one trial, 232 women) (Analysis 1.21), change in weight from late

pregnancy to three months postpartum (MD  $-0.35\text{kg}$ , 95% CI  $-1.84$  to  $1.14$ ; one trial, 165 women) (Analysis 1.22), and postpartum BMI (reported as mean and range in Laitinen 2009: Analysis 1.24), in one of the trials, women in the dietary intervention group had a greater reduction in weight from baseline to three months postpartum (MD  $-1.43\text{kg}$ , 95% CI  $-2.66$  to  $-0.20$ ; 414 women) (Analysis 1.23). Of interest, in Thornton 2009 the reported standard deviation for the standard care group for postpartum weight loss at six weeks (Analysis 1.21) was notably higher ( $14.84\text{kg}$  versus  $3.27\text{kg}$ ), indicating weight loss was more variable in the standard care compared with the dietary intervention group. Wolff 2008 also reported that “The intervention group ( $n=16$ ) retained  $6.9\text{kg}$  less weight than the control group ( $n=19$ ) 4 weeks postpartum ( $-4.5$  vs  $2.4\text{kg}$ , 95% CI of difference:  $2.5$ - $11.2$ ,  $P=0.003$ ) compared to the pregnancy weight”.

The trials in this comparison did not report on the outcomes: postnatal depression; GDM in a subsequent pregnancy; type 1 diabetes mellitus; type 2 diabetes mellitus; impaired glucose tolerance; cardiovascular health (e.g. blood pressure, hypertension, cardiovascular disease, metabolic syndrome).

## For the child

### Fetal/neonatal outcomes

Only two trials reported on stillbirth; in one trial there were no stillborn babies, and in the other, there was one stillbirth in the dietary intervention group (associated with trisomy 21) (RR 3.09, 95% CI 0.13 to 75.65; two trials, 959 babies) (Analysis 1.25). Only one of these trials reported on neonatal deaths, and none occurred in either group (Analysis 1.26).

There were no clear differences between the dietary advice intervention and standard care groups for the following outcomes: preterm birth (less than 37 weeks) (RR 0.51, 95% CI 0.21 to 1.25; three trials, 1149 babies) (Analysis 1.27); preterm birth (less than 32 weeks) (RR 1.70, 95% CI 0.23 to 12.88; two trials, 917 babies) (Analysis 1.28); Apgar score less than seven at five minutes (RR 3.00, 95% CI 0.12 to 72.89; one trial, 232 babies) (Analysis 1.29); macrosomia ( $<4000\text{g}$ ) (RR 0.99, 95% CI 0.86 to 1.14; one trial, 759 babies) (Analysis 1.30) and macrosomia ( $<4500\text{g}$ ) (RR 2.25, 95% CI 0.71 to 7.10; one trial, 232 babies) (Analysis 1.31); shoulder dystocia (RR 0.52, 95% CI 0.10 to 2.82; one trial, 759 babies) (Analysis 1.32); gestational age at birth (MD 0.05 weeks, 95% CI  $-0.31$  to  $0.40$ ; four trials, 1195 babies;  $\text{Tau}^2 = 0.05$ ;  $\text{I}^2 = 34\%$ ) (Analysis 1.33); birthweight (MD  $5.94\text{g}$ , 95% CI  $-51.11$  to  $62.99$ ; five trials, 1324 babies) (Analysis 1.34); head circumference at birth (MD  $-0.21\text{cm}$ , 95% CI  $-0.67$  to  $0.25$ ; three trials, 968 babies;  $\text{Tau}^2 = 0.11$ ;  $\text{I}^2 = 72\%$ ) (Analysis 1.35); length at birth (MD  $0.16\text{cm}$ , 95% CI  $-0.28$  to  $0.60$ ; three trials, 968 babies;  $\text{Tau}^2 = 0.05$ ;  $\text{I}^2 = 33\%$ ) (Analysis 1.36); ponderal index at birth (MD  $0.01$ , 95% CI  $-0.38$  to  $0.40$ ; one trial, 759 babies) (Analysis 1.37);

or adiposity (skin-fold thickness at birth) (MD -1.60 mm, 95% CI -4.77 to 1.57; one trial, 219 babies) (Analysis 1.38). In Walsh 2012 the reported standard deviation for the dietary intervention group for ponderal index at birth (Analysis 1.37) was considerably higher (3.8 versus 0.33), suggesting ponderal indices were more variable in the dietary intervention compared with the standard care group. Substantial statistical heterogeneity was observed in the meta-analysis for head circumference at birth, and thus the pooled result should be interpreted with some caution. The trials in this comparison did not report on the outcomes: small-for-gestational age; nerve palsy; bone fracture; respiratory distress syndrome; hypoglycaemia; hyperbilirubinaemia.

### **Childhood/adulthood outcomes**

There was no difference between the dietary advice intervention and standard care groups in Walsh 2012 for weight at three months (MD 0.23 kg, 95% CI -0.37 to 0.83; one trial, 422 children) (Analysis 1.39). In Walsh 2012, the reported standard deviation for the dietary intervention group for weight at three months (Analysis 1.39) was notably higher (4.36 kg versus 0.98 kg), indicating ponderal indices were more variable in the dietary intervention compared with the standard care group.

Similarly, there were no clear differences between the dietary advice intervention and standard care groups in Laitinen 2009 for the following outcomes: weight at six months (MD -0.03 kg, 95% CI -0.35 to 0.29; one trial, 143 children) (Analysis 1.40); length at six months (MD 0.00 cm, 95% CI -1.06 to 1.06; one trial, 143 children) (Analysis 1.41); head circumference at six months (MD -0.20 cm, 95% CI -0.61 to 0.21; one trial, 132 children) (Analysis 1.42); adiposity (skin-fold thickness at six months) (MD -0.10 mm, 95% CI -0.71 to 0.51; one trial, 132 children; GRADE: *low-quality evidence*) (Analysis 1.43); systolic (MD -1.00 mmHg, 95% CI -4.53 to 2.53; one trial, 113 children) (Analysis 1.44), diastolic (MD -1.00 mmHg, 95% CI -3.77 to 1.77; one trial, 113 children) (Analysis 1.45), or mean blood pressure (MD -1.00 mmHg, 95% CI -3.77 to 1.77; one trial, 113 children) at six months (Analysis 1.46); or heart rate at six months (MD 2.00 bpm, 95% CI -2.89 to 6.89; one trial, 113 children) (Analysis 1.47).

Laitinen 2009 also reported no clear differences in growth at follow up between groups: "Weight gain and growth in length during the periods 0-6 months, 6-12 months and 12-24 months showed no statistically significant differences between the diet/probiotics, diet/placebo and control groups. The respective mean weight gains (g/month) over 0-6 months were 759 (SD 160), 762 (SD 165) and 756 (SD 148); over 6-12 months were 323 (SD 80), 296 (SD 99) and 315 (SD 91); over 12-24 months were 211 (SD 76), 230 (SD 61) and 218 (SD 52) ( $P=0.983$  for group effect;  $P<0.001$  for time effect;  $P=0.520$  for group X time interaction; ANOVA for repeated measurements). The mean growths in length (cm/month) in the respective periods were 2.84 (SD 0.35), 2.89 (SD 0.29) and 2.93 (SD 0.35); 1.40 (SD 0.19), 1.38 (SD 0.21) and 1.36 (SD 0.24); 0.95 (SD 0.14), 0.94

(SD 0.15) and 0.93 (SD 0.12) in the diet/probiotics, diet/placebo and control groups, respectively ( $P=0.872$  for group effect;  $P<0.001$  for time effect;  $P=0.325$  for group X time interaction; ANOVA for repeated measurements)". We have not included these data in the meta-analyses as the N values for the two relevant groups (diet/placebo and control groups) were not clearly reported.

The trials in this comparison did not report on the outcomes: employment, education and social status/achievement; type 1 diabetes mellitus; type 2 diabetes mellitus; impaired glucose tolerance; neurosensory disability.

### **Use of health services**

The trials in this comparison did not report on any of the secondary outcomes relating to the use of health services.

### **Non pre-specified review outcomes**

Vitolo 2011 reported on a composite outcome 'clinical complications' (GDM, pre-eclampsia, low birthweight, prematurity), and showed a reduction in this outcome for women who received the dietary advice intervention (RR 0.37, 95% CI 0.21 to 0.66; 305 women/babies) (Analysis 1.48).

## **Comparison 2: Low-GI dietary advice versus moderate- to high-GI dietary advice**

Four trials were included in this arm of the review (Clapp 1998; Markovic 2016; Moses 2006; Moses 2014).

### **Primary outcomes**

#### **Gestational diabetes (GDM)**

No clear difference was shown in the risk of GDM between women in the low-GI dietary advice group and moderate- to high-GI dietary advice group (RR 0.91, 95% CI 0.63 to 1.31; four trials, 912 women; GRADE: *low-quality evidence*) (Analysis 2.1).

#### **Hypertensive disorders of pregnancy**

None of the four trials in this comparison reported on the primary outcome: hypertensive disorders of pregnancy.

#### **Perinatal mortality (stillbirth or neonatal mortality)**

None of the four trials in this comparison reported on the primary outcome: perinatal mortality.

### Large-for-gestational age

There was no clear difference in the risk of babies being born large-for-gestational age between the low-GI dietary advice and moderate- to high-GI dietary advice groups (average RR 0.60, 95% CI 0.19 to 1.86; three trials, 777 babies;  $\text{Tau}^2 = 0.61$ ;  $P = 0.07$ ;  $I^2 = 62\%$ ; GRADE: *very low-quality evidence*) (Analysis 2.2). Clapp 1998 reported that “The women who ate the low-glycemic diets were delivered of normal sized infants whose measurements fell between the 25th and 75th centiles. Those born of women eating the high-glycemic diet were symmetrically overgrown with average birth weights, crown-heel lengths, ponderal indices and lean body masses >90th centile without an excessive increase in either fat mass or the fetoplacental weight ratio”; however these data were not able to be included in the above meta-analysis.

### Neonatal mortality or morbidity composite

None of the four trials in this comparison reported on the primary outcome: mortality or morbidity composite.

### Secondary outcomes

#### For the mother

#### Perinatal outcomes

No clear differences were shown between the low-GI dietary advice and moderate- to high-GI dietary advice groups for the secondary outcomes: caesarean birth (RR 1.27, 95% CI 0.79 to 2.04; two trials, 201 women; GRADE: *very low-quality evidence*) (Analysis 2.3); operative vaginal birth (RR 1.25, 95% CI 0.49 to 3.18; one trial, 62 women) (Analysis 2.4); and gestational weight gain (MD -1.23 kg, 95% CI -4.08 to 1.61; four trials, 787 women;  $\text{Tau}^2 = 7.31$ ;  $I^2 = 90\%$ ; GRADE: *very low-quality evidence*) (Analysis 2.5). Considerable statistical heterogeneity was observed in the meta-analysis for gestational weight gain, and thus the pooled result should be interpreted with some caution.

In regards to adherence to the intervention, two trials (Moses 2006; Moses 2014) asked women to respond to the statement ‘I adhered well to the dietary instructions’ using a five-point Likert scale (with one being ‘all of the time’ and five being ‘none of the time’). No clear difference between the low GI-dietary advice and moderate- to high-GI dietary advice groups was seen in regards to adherence (MD 0.03, 95% CI -0.07 to 0.13; two trials, 636 women) (Analysis 2.6).

While no clear difference was seen between the low-GI dietary advice and moderate- to high-GI dietary advice groups for fasting blood glucose at 24 to 28 weeks in one trial (MD -0.17 mmol/L, 95% CI -0.57 to 0.23; 20 women) (Analysis 2.7); at 32 to

36 weeks, the low-GI dietary advice group in two trials had a significantly lower fasting blood glucose concentration (MD -0.27 mmol/L, 95% CI -0.52 to -0.03; 82 women) (Analysis 2.8).

In two trials (Moses 2006; Moses 2014), women were asked to respond to a number of statements relating to their views of the intervention, using a five-point Likert scale (with one being ‘strongly agree’ and five being ‘strongly disagree’). No clear difference between the low-GI dietary advice and moderate- to high-GI dietary advice groups were seen for the following statements: ‘It was easy to follow the diet recommended during this study’ (MD -0.09, 95% CI -0.45 to 0.27; two trials, 636 women;  $\text{Tau}^2 = 0.06$ ;  $I^2 = 82\%$ ); ‘I enjoyed the dietary changes that I made’ (MD -0.09, 95% CI -0.22 to 0.03; two trials, 636 women); ‘The changes recommended were affordable’ (MD 0.04, 95% CI -0.08 to 0.16; two trials, 636 women); ‘My family was accepting of the changes made to my eating habits’ (MD -0.02, 95% CI -0.15 to 0.10; two trials, 636 women); ‘The study diet helped me meet the physical challenges of pregnancy’ (MD 0.10, 95% CI -0.03 to 0.22; two trials, 636 women); and ‘I enjoyed a wide variety of foods in my eating plan’ (MD -0.05, 95% CI -0.15 to 0.05; two trials, 636 women) (Analysis 2.9).

The trials in this comparison did not report on the outcomes: induction of labour; perineal trauma; placental abruption; postpartum haemorrhage; postpartum infection; breastfeeding; behaviour changes associated with the intervention; sense of well-being and quality of life.

#### Longer-term maternal outcomes

The trials in this comparison did not report on any of the secondary longer-term maternal outcomes.

#### For the child

#### Fetal/neonatal outcomes

No clear differences were shown between the low-GI dietary advice and moderate- to high-GI dietary advice groups for the secondary outcomes: Apgar score less than seven at five minutes (RR 2.82, 95% CI 0.12 to 66.62; one trial, 62 babies) (Analysis 2.10); macrosomia (> 4000 g) (RR 0.73, 95% CI 0.49 to 1.09; two trials, 715 babies) (Analysis 2.11) and macrosomia (> 4500 g) (RR 0.32, 95% CI 0.06 to 1.55; one trial, 576 babies) (Analysis 2.12); small-for-gestational age (RR 0.88, 95% CI 0.53 to 1.45; three trials, 777 babies) (Analysis 2.13); gestational age at birth (MD 0.11 weeks, 95% CI -0.11 to 0.33; three trials, 777 babies) (Analysis 2.14); birthweight (MD -217.97 g, 95% CI -483.96 to 48.02; four trials, 797 babies;  $\text{Tau}^2 = 62689.88$ ;  $I^2 = 90\%$ ) (Analysis 2.15); birthweight z score (MD 0.07, 95% CI -0.26 to 0.40; one trial, 139 babies) (Analysis 2.16); head circumference at birth (MD -



1.20 cm, 95% CI -2.75 to 0.36; two trials, 82 babies;  $Tau^2 = 0.99$ ;  $I^2 = 78\%$ ) (Analysis 2.17); length at birth (MD -0.77 cm, 95% CI -1.98 to 0.45; three trials, 658 babies;  $Tau^2 = 0.89$ ;  $I^2 = 81\%$ ) (Analysis 2.18); ponderal index at birth (MD -0.06, 95% CI -0.16 to 0.04; four trials, 797 babies;  $Tau^2 = 0.01$ ;  $I^2 = 80\%$ ) (Analysis 2.19); or adiposity at birth (% body fat) (MD 0.02, 95% CI -1.43 to 1.47; two trials, 108 babies) (Analysis 2.20). Considerable statistical heterogeneity was observed in the meta-analyses for birthweight, head circumference, length at birth and ponderal index at birth, and thus the pooled results should be interpreted with some caution.

The trials in this comparison did not report on the secondary outcomes: stillbirth; neonatal mortality; preterm birth; shoulder dystocia; nerve palsy; bone fracture; respiratory distress syndrome; hypoglycaemia; hyperbilirubinaemia.

### Childhood/adulthood outcomes

Markovic 2016 reported that “At 3 months of age, there were no significant differences between diet groups in growth parameters or body composition”.

The four trials in this comparison did not report any other data regarding the secondary childhood/adulthood outcomes.

### Use of health services

No clear difference was shown between the low-GI dietary advice and moderate- to high-GI dietary advice groups for neonatal intensive care unit admission (RR 0.37, 95% CI 0.12 to 1.11; one trial, 138 babies) (Analysis 2.21).

The trials in this comparison did not report on any of the other secondary outcomes relating to the use of health services.

## Comparison 3: High-fibre dietary advice versus 'standard' dietary advice

One trial was included in this comparison (Fraser 1983).

### Primary outcomes

Fraser 1983 did not report on any of the primary outcomes: GDM; hypertensive disorders of pregnancy; perinatal mortality (stillbirth or neonatal mortality); large-for-gestational age; neonatal mortality or morbidity composite.

### Secondary outcomes

### For the mother

### Perinatal outcomes

Fraser 1983 reported on mean blood glucose concentrations following an OGTT at 35 weeks, and showed no clear difference between the high-fibre dietary advice and standard dietary advice groups (MD -0.36 mmol/L, 95% CI -0.90 to 0.18; 25 women) (Analysis 3.1). Fraser 1983 also reported that “Results of the antenatal monitoring (including maternal weight gain)... showed no significant differences between the groups”.

Fraser 1983 did not report on any of the other secondary perinatal outcomes.

### Longer-term maternal outcomes

Fraser 1983 did not report on any of the secondary longer-term maternal outcomes.

### For the child

### Fetal/neonatal outcomes

Fraser 1983 reported on birthweight centile, and showed no clear difference between the high-fibre dietary advice and standard dietary advice groups (MD -0.30, 95% CI -5.40 to 4.80; 25 babies) (Analysis 3.2). Fraser 1983 also reported that “Results of... fetal anthropometry showed no significant differences between the groups”.

Fraser 1983 did not report on any of the other secondary fetal/neonatal outcomes.

### Childhood/adulthood outcomes

Fraser 1983 did not report on any of the secondary childhood/adulthood outcomes.

### Use of health services

Fraser 1983 did not report on any of the secondary outcomes relating to the use of health services.

### Subgroup analyses

For Comparison 1: Dietary advice interventions versus standard care, we conducted a subgroup analysis for our primary outcome, GDM, based on BMI at trial entry. The test for subgroup differences was significant ( $Chi^2 = 4.57$ ,  $P = 0.03$ ,  $I^2 = 78.1\%$ ), indicating a possible difference in treatment effect based on BMI (Analysis 1.1). In particular, this subgroup analysis suggested a greater treatment effect (reduction in GDM risk for women who received dietary advice interventions) among trials that recruited

overweight and/or obese women (BMI > 25). We were not able to perform subgroup analyses based on BMI at trial entry for the other primary outcomes in this comparison, with only two trials (both recruiting overweight/obese women) reporting on hypertensive disorders of pregnancy, and only one trial reporting on perinatal mortality.

We were not able to perform the other pre-specified subgroup analyses in this version of the review, due to the paucity of data across the three comparisons.

### Sensitivity analyses

For Comparison 1: Dietary advice interventions versus standard care, we conducted sensitivity analyses for our primary outcomes with reported data, exploring the effects of trial quality assessed by sequence generation and allocation concealment by omitting studies rated as 'high risk of bias' or 'unclear risk of bias' for these components. As [Wolff 2008](#) was the only trial to have an 'unclear risk of bias' rating for allocation concealment, this was the only trial excluded from the GDM meta-analysis. Excluding this trial from the meta-analysis did not greatly impact the overall effect observed for this outcome (RR 0.63, 95% CI 0.36 to 1.10; four trials, 1266 women) ([Analysis 1.1](#)). When we excluded data from [Wolff](#)

[2008](#) from the meta-analysis for pregnancy-induced hypertension, only data from [Thornton 2009](#) remained, which showed no clear difference between groups (different to in the main analysis) (RR 0.30, 95% CI 0.08 to 1.06; one trial, 232 women) ([Analysis 1.2](#)). Similarly, when data from [Wolff 2008](#) were excluded from the meta-analysis for pre-eclampsia, only data from [Thornton 2009](#) remained, with no clear difference between groups (as in the main analysis) (RR 0.64, 95% CI 0.26 to 1.58; one trial, 232 women) ([Analysis 1.3](#)). Only [Laitinen 2009](#) reported on perinatal mortality (with no deaths observed in this trial) ([Analysis 1.4](#)), and thus no sensitivity analysis was performed for this outcome.

For Comparison 2: Low-GI dietary advice versus moderate- to high-GI dietary advice, we planned to conduct sensitivity analyses for our primary outcomes with reported data (GDM and large-for-gestational age). However, as all of the four trials contributing data to these meta-analyses had an 'unclear risk of bias' or 'high risk of bias' for at least one of the two domains (sequence generation or allocation concealment), we did not perform these analyses.

No sensitivity analyses were performed for Comparison 3: High-fibre dietary advice versus standard dietary advice, given that only one trial was included ([Fraser 1983](#)), and this trial did not report on the primary outcomes.

## ADDITIONAL SUMMARY OF FINDINGS *[Explanation]*

Dietary advice interventions versus standard care (child outcomes)						
<b>Population:</b> pregnant women <b>Setting:</b> 6 studies carried out in Australia, the USA, Brazil, Denmark, Ireland and Finland <b>Intervention:</b> dietary advice interventions <b>Comparison:</b> standard care						
Outcomes	Anticipated absolute effects* (95% CI)		Relative effect (95% CI)	No of participants (studies)	Quality of the evidence (GRADE)	Comments
	Risk with standard care	Risk with dietary advice interventions				
Perinatal mortality	Study population		Not estimable	159 (1 RCT)	⊕○○○ VERY LOW <sup>1,2</sup>	Effect not estimable. Outcome reported in a single study with no events
	0 per 1000	0 per 1000 (0 to 0)				
Large-for-gestational age			Not estimable	(0 studies)		No data reported for large-for-gestational age in any of the included studies
Mortality or morbidity composite outcome			Not estimable	(0 studies)		No data reported for mortality or morbidity composite in any of the included studies
Neonatal hypoglycaemia			Not estimable	(0 studies)		No data reported for neonatal hypoglycaemia in any of the included studies

Childhood/adulthood adiposity: skinfold thickness at 6 months (mm)	The mean skinfold thickness in the intervention group was 0.1 mm less (0.71 less to 0.51 more)	MD -0.10 (-0.71 to 0.51)	132 (1 RCT)	⊕⊕○○ LOW <sup>1,3</sup>	Estimate based on findings from a single study
Childhood/adulthood type 2 diabetes mellitus		Not estimable	(0 studies)		No data reported for childhood/adulthood type 2 diabetes in any of the included studies
Childhood/adulthood neurosensory disability		Not estimable	(0 studies)		No data reported for childhood/adulthood neurosensory disability in any of the included studies

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

CI: confidence interval; MD: mean difference; mm: millimetre; RCT: randomised controlled trial; RR: risk ratio; SD: standard deviation

#### GRADE Working Group grades of evidence

**High quality:** We are very confident that the true effect lies close to that of the estimate of the effect

**Moderate quality:** We are moderately confident in the effect estimate: The true effect is likely to be close to the estimate of the effect, but there is a possibility that it is substantially different

**Low quality:** Our confidence in the effect estimate is limited: The true effect may be substantially different from the estimate of the effect

**Very low quality:** We have very little confidence in the effect estimate: The true effect is likely to be substantially different from the estimate of effect

<sup>1</sup>Single study with design limitations contributing data

<sup>2</sup>Single study with small sample size and no events

<sup>3</sup>Estimate based on single study with small sample size and wide 95% CI crossing the line of no effect

Low-GI dietary advice versus moderate- to high-GI dietary advice (maternal outcomes)						
<b>Population:</b> pregnant women <b>Setting:</b> 4 studies carried out in Australia (3) and the USA (1) <b>Intervention:</b> low-GI dietary advice <b>Comparison:</b> moderate- to high-GI dietary advice						
Outcomes	Anticipated absolute effects* (95% CI)		Relative effect (95% CI)	No. of participants (studies)	Quality of the evidence (GRADE)	Comments
	Risk with moderate- to high-GI dietary advice	Risk with low- GI dietary advice				
Gestational diabetes	Study population		RR 0.91 (0.63 to 1.31)	912 (4 RCTs)	⊕⊕○○ LOW <sup>1,2</sup>	
	110 per 1000	100 per 1000 (70 to 145)				
Hypertensive disorders of pregnancy			Not estimable	(0 studies)		No data reported for hypertensive disorders of pregnancy in any of the included studies
Caesarean birth	Study population		RR 1.27 (0.79 to 2.04)	201 (2 RCTs)	⊕○○○ VERY LOW <sup>3,4</sup>	
	227 per 1000	288 per 1000 (179 to 463)				
Perineal trauma			Not estimable	(0 studies)		No data reported for perineal trauma in any of the included studies
Gestational weight gain (kg)	The mean gestational weight gain in the intervention group was 1.23 kg less than in the control group (4.08 kg less to 1.61 more)		MD -1.23 (-4.08 to 1.61)	787 (4 RCTs)	⊕○○○ VERY LOW <sup>2,3,5</sup>	

Postnatal depression		Not estimable	(0 studies)	No data reported for postnatal depression in any of the included studies
Type 2 diabetes mellitus		Not estimable	(0 studies)	No data reported for type 2 diabetes in any of the included studies

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

**CI:** confidence interval; **kg:** kilogram; **MD:** mean difference; **RCT:** randomised controlled trial; **RR:** risk ratio

**GRADE Working Group grades of evidence**

**High quality:** We are very confident that the true effect lies close to that of the estimate of the effect

**Moderate quality:** We are moderately confident in the effect estimate: The true effect is likely to be close to the estimate of the effect, but there is a possibility that it is substantially different

**Low quality:** Our confidence in the effect estimate is limited: The true effect may be substantially different from the estimate of the effect

**Very low quality:** We have very little confidence in the effect estimate: The true effect is likely to be substantially different from the estimate of effect

<sup>1</sup>Studies contributing data had design limitations

<sup>2</sup>Wide 95% CI crossing the line of no effect

<sup>3</sup>Studies contributing data had serious or very serious design limitations

<sup>4</sup>Estimate based on studies with small sample sizes, and wide 95% CI crossing the line of no effect

<sup>5</sup>Substantial heterogeneity ( $I^2 = 90\%$ )

Low-GI dietary advice versus moderate- to high-GI dietary advice (child outcomes)						
<b>Population:</b> pregnant women <b>Setting:</b> 4 studies carried out in Australia (3) and the USA (1) <b>Intervention:</b> low-GI dietary advice <b>Comparison:</b> moderate- to high-GI dietary advice						
Outcomes	Anticipated absolute effects* (95% CI)		Relative effect (95% CI)	No. of participants (studies)	Quality of the evidence (GRADE)	Comments
	Risk with moderate- to high-GI dietary advice	Risk with low-GI dietary advice				
Perinatal mortality			Not estimable	(0 studies)		No data reported for perinatal mortality in any of the included studies
Large-for-gestational age	Study population		RR 0.60 (0.19 to 1.86)	777 (3 RCTs)	⊕○○○ VERY LOW <sup>1,2,3</sup>	
	114 per 1000	68 per 1000 (22 to 212)				
Mortality or morbidity composite outcome			Not estimable	(0 studies)		No data reported for mortality or morbidity composite in any of the included studies
Neonatal hypoglycaemia			Not estimable	(0 studies)		No data reported for neonatal hypoglycaemia in any of the included studies
Childhood/adulthood adiposity			Not estimable	(0 studies)		No data reported for childhood/adulthood adiposity in any of the included studies

Childhood/adulthood type 2 diabetes mellitus		Not estimable	(0 studies)	No data reported for childhood/adulthood type 2 diabetes in any of the included studies
Childhood/adulthood neurosensory disability		Not estimable	(0 studies)	No data reported for childhood/adulthood neurosensory disability in any of the included studies

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

**CI:** confidence interval; **RCT:** randomised controlled trial; **RR:** risk ratio

**GRADE Working Group grades of evidence**

**High quality:** We are very confident that the true effect lies close to that of the estimate of the effect

**Moderate quality:** We are moderately confident in the effect estimate: The true effect is likely to be close to the estimate of the effect, but there is a possibility that it is substantially different

**Low quality:** Our confidence in the effect estimate is limited: The true effect may be substantially different from the estimate of the effect

**Very low quality:** We have very little confidence in the effect estimate: The true effect is likely to be substantially different from the estimate of effect

<sup>1</sup>Studies contributing data had serious or very serious design limitations

<sup>2</sup>Substantial heterogeneity ( $I^2 = 62\%$ )

<sup>3</sup>Wide 95% CI crossing the line of no effect



## DISCUSSION

### Summary of main results

Eleven trials (involving 2786 women and their babies) met the inclusion criteria for the review. Six trials compared dietary advice interventions with standard care (Laitinen 2009; Quinlivan 2011; Thornton 2009; Vitolo 2011; Walsh 2012; Wolff 2008); four compared low glycaemic index (GI) with moderate- to high-GI dietary advice interventions (Clapp 1998; Markovic 2016; Moses 2006; Moses 2014); and one compared specific (high-fibre focused) with standard dietary advice (Fraser 1983).

Meta-analysis of trials comparing dietary advice interventions with standard care demonstrated a trend towards a reduction in the risk of gestational diabetes mellitus (GDM) (8.60% versus 12.60%, in five trials) ( $P = 0.07$ ). The subgroup analysis based on body mass index (BMI) at trial entry, suggested a possible differential treatment effect for this outcome based on trial entry BMI, with a greater effect on GDM incidence observed in overweight and/or obese women receiving dietary advice. No clear differences were observed for the other reported primary outcomes pre-eclampsia and perinatal mortality; these findings remained unchanged in the sensitivity analyses involving trials with a low risk of bias in the domains of sequence generation and allocation concealment.

Similarly, we found no clear differences in these trials for the majority of reported secondary outcomes for the mother and the child. In these trials, reductions in pregnancy-induced hypertension (2.88% versus 9.79%, across two trials) and gestational weight gain (on average 4.70 kg less, across five trials) were observed, along with greater weight loss at three months postpartum (on average 1.43 kg more, in one trial) for women who received the dietary advice interventions. Well-being (assessed using the WHO well-being index) at 14 to 28 weeks' gestation, was found to be lower in the dietary intervention group (a percentage score 3.60 lower, in one trial). Diet-related behaviour changes were mixed at three months postpartum (one trial). Over-interpretation of these findings is cautioned in view of the small sample sizes and limited high-quality evidence.

In the trials comparing low-GI with moderate- to high-GI dietary advice interventions, no clear differences were seen for the reported primary outcomes: GDM and large-for-gestational age; or for the majority of reported secondary outcomes for the mother and the child. While no difference was shown for fasting glucose at 24 to 28 weeks (one trial), women receiving low-GI dietary advice had a marginally lower fasting blood glucose at 32 to 36 weeks (0.27 mmol/L lower, in two trials).

In the trial comparing high-fibre dietary advice with standard dietary advice, no clear differences were seen for the two reported secondary outcomes: blood glucose following an oral glucose tolerance test (OGTT) at 35 weeks; and birthweight centile.

### Overall completeness and applicability of evidence

The trials in this review were conducted with healthy women and those considered at high risk of developing GDM, including overweight/obese women. These trials recruited pregnant women from hospitals in Australia (Markovic 2016; Moses 2006; Moses 2014; Quinlivan 2011), the USA (Clapp 1998; Thornton 2009), Brazil (Vitolo 2011), Denmark (Wolff 2008), Finland (Laitinen 2009), Ireland (Walsh 2012), and the UK (Fraser 1983); and thus, the results may not be applicable to all settings or countries worldwide. Four of the included trials (Clapp 1998; Fraser 1983; Moses 2006; Wolff 2008) included 73 women or fewer, five trials included 315 women or fewer (Markovic 2016; Quinlivan 2011; Thornton 2009; Vitolo 2011). The two largest trials were in 691 and 800 women (Moses 2014; Walsh 2012). Thus, largely the included trials were far too small to show differences in important but rare outcomes, such as perinatal mortality, and even in more common outcomes, such as GDM.

Though there were a total of 2786 women and their babies across the included trials, for the majority of outcomes, only one or two trials reported data, further limiting the statistical power of the meta-analyses and the precision of the estimates of treatment effects. Lack of uniformity in reporting outcomes including diagnostic criteria limits the interpretation of data. Many important outcomes for women and their babies were not reported across the comparisons.

- The six trials assessing dietary advice interventions versus standard care (Laitinen 2009; Quinlivan 2011; Thornton 2009; Vitolo 2011; Walsh 2012; Wolff 2008), did not report on the primary outcomes included in this review: large-for-gestational age and neonatal mortality and morbidity composite; and the trials did not report on a number of secondary outcomes, particularly long-term outcomes for the mother and child, and health services outcomes.

- The four trials comparing low-GI dietary advice with moderate- to high-GI dietary advice (Clapp 1998; Markovic 2016; Moses 2006; Moses 2014), did not report on the primary outcomes of this review: hypertensive disorders of pregnancy, perinatal mortality, and neonatal mortality and morbidity composite; and similarly did not report the majority of secondary outcomes, including long-term outcomes for the mother and child, and health services outcomes.

- The one trial comparing high-fibre dietary advice with "standard advice" (Fraser 1983) did not report on any primary outcomes, and reported on only two secondary outcomes.

This may reflect multiple factors influencing outcome data collection and reporting, including evolving recognition of important outcome measures and changes in diagnostic parameters, particularly affecting older trials, the selective reporting of outcome data by trials where no differences were observed, or limitations at manuscript publication. The limited data regarding longer-term

health of women and their children reported to date from the included trials, could reflect a 'lag' time between recognition by trialists of the importance of such outcomes, and the subsequent collection and reporting of these outcome data (Bain 2016). While long-term follow-up of trials assessing perinatal interventions is widely regarded as important, it has been recognised that only a minority of trials are able to do so; often due to the time-consuming and expensive nature of follow-up, commonly falling outside of the funding period, or interest of the trialists (Teune 2013).

While the often 'negative' results from included trials to date may reflect lack of statistical power, the absence of observed clear differences could also be partially attributable to lack of intervention uptake (Halperin 2014). The efficacy of dietary intervention is likely to be influenced by many factors, including background dietary habits and barriers such as affordability, satisfaction with changes and convenience. It is noteworthy that in one small trial, low-GI diet was associated with a poorer sense of well-being, which is likely to influence intervention adherence. In the included trials, information regarding adherence, but particularly women's views, has to date been limited. As noted by Halperin 2014 in a recent narrative review of the role of lifestyle interventions for GDM prevention, in addition to being powered to detect reductions in GDM, future trials should be designed to monitor lifestyle changes closely, and may include a psychological component as part of the intervention, such as in Quinlivan 2011.

Earlier trials, such as Clapp 1998 and Moses 2006 compared low-GI with high or moderate- to high-GI dietary advice, including to test the hypothesis that a woman's dietary carbohydrate mix modifies glucose and insulin responses (Clapp 1998). In recognition that current best practice for GDM prevention, and indeed management, focuses on optimising glycaemic control, it is unlikely that further trials specifically assessing high-GI diets will be conducted. More recent trials, such as Markovic 2016 and Moses 2014, have instead focused on comparisons of low-GI specific dietary advice with 'standard' healthy eating advice (including for a moderate-GI diet), or on comparing more general 'healthy eating' advice (including in combination with a multi-disciplinary approach (Quinlivan 2011), or with an additional focus on low-GI foods (Walsh 2012)) with standard care.

### Quality of the evidence

Overall, the 11 included trials were judged to be at unclear to moderate risk of bias. Often there was insufficient information provided to enable a judgement of risk, particularly with regard to sequence generation and allocation concealment, blinding of outcome assessment, and selective reporting. Only four trials (Laitinen 2009; Quinlivan 2011; Thornton 2009; Walsh 2012) were considered to be at low risk of selection bias (with methods considered to be adequate for both sequence generation and allocation concealment); where possible, these trials were included in sensitivity analyses for primary outcomes in the first comparison.

We used GRADE profiler to assess the quality of the evidence for the two comparisons 'Dietary advice interventions versus standard care', [Summary of findings for the main comparison](#); [Summary of findings 2](#), and 'Low-GI dietary advice versus moderate- to high-GI dietary advice' [Summary of findings 3](#); [Summary of findings 4](#). Several maternal and child outcomes were assessed for their quality of evidence.

For the first comparison, the evidence was of low quality (assessed using the GRADE system) for the following outcomes: hypertensive disorders of pregnancy (pre-eclampsia; pregnancy-induced hypertension), caesarean section, gestational weight gain, and childhood adult adiposity (skin-fold thickness at six months). The evidence for the outcomes: GDM, perineal trauma and perinatal mortality was very low quality.

For the second comparison, the evidence for GDM was assessed of being low quality, while the evidence for the outcomes: caesarean section, gestational weight gain, and large-for-gestational age, was assessed as being very low quality.

Evidence downgrading was based on study limitations (risk of bias), imprecision (largely the presence of wide confidence intervals crossing the line of no effect), and inconsistency.

### Potential biases in the review process

To reduce the potential for publication bias, a detailed, systematic search process was conducted by the Information Specialist of the Cochrane Pregnancy and Childbirth Group, without language or publication status restrictions. It is possible that additional trials assessing dietary advice intervention in pregnancy have been published but not identified; and/or that further trials have been conducted but are not yet published. Should any such studies be identified, we will include them in future updates of this review. In order to minimise bias throughout the review process, two review authors independently assessed eligibility for inclusion, extracted data and assessed risk of bias.

### Agreements and disagreements with other studies or reviews

Two Cochrane systematic reviews have assessed exercise alone (Han 2012) and combined diet and exercise interventions (Bain 2015) for preventing GDM. In Han 2012, five randomised trials (involving 1115 women and their babies) assessing exercise interventions were included. Han 2012 found no clear difference between women who received exercise interventions during pregnancy and those who received standard care for the risk of GDM, nor for any of the other primary or secondary outcomes for women and their babies reported by the included trials (Han 2012). Bain 2015 included 13 randomised trials (involving 4983 women and their babies) assessing combined diet and exercise interventions. In this review no clear difference between groups was shown for the

risk of GDM, nor for the majority of other primary and secondary outcomes reported by the included trials (Bain 2015). The review did show a possible reduction in preterm birth for women receiving diet and exercise interventions (risk ratio (RR) 0.71, 95% confidence interval (CI) 0.55 to 0.93; five trials, 2713 infants) (Bain 2015) (a difference not observed in this review), however the review authors suggested caution in interpretation of these results due to clinical and statistical heterogeneity of the trials combined in the meta-analysis. In Bain 2015, similar to our review, a possible benefit in relation to less weight gain during pregnancy was observed for women receiving combined diet and exercise interventions. On average, women receiving diet and exercise interventions gained 0.76 kg less than women in the control group (mean difference (MD) -0.76 kg, 95% CI -1.55 to 0.03; eight trials, 2707 women) (Bain 2015). These reviews have largely been limited by quality of evidence, heterogeneity in trial methodologies and outcome reporting, similar to this review.

An additional Cochrane review (Muktabhant 2015) has assessed diet or exercise, or both, interventions for preventing excessive weight gain during pregnancy, and associated complications. This review included 65 randomised trials (49 involving 11,444 women with data in the meta-analyses) (Muktabhant 2015). Similar to in our review, a benefit in relation to less weight gain during pregnancy was observed - women receiving diet or exercise, or both interventions (including interventions involving low glycaemic load diets, supervised or unsupervised exercise only, or diet and exercise combined) were less likely to gain excessive gestational weight gain (RR 0.80, 95% CI 0.73 to 0.87; 24 trials, 7096 women) (Muktabhant 2015). Also similar to our review, while no clear difference was seen in the risk of pre-eclampsia, a possible reduction in maternal hypertension was observed for women receiving diet or exercise, or both interventions (RR 0.70, 95% CI 0.51 to 0.96; 11 trials, 5162 women) (Muktabhant 2015).

A number of additional non-Cochrane systematic reviews have recently assessed a range of interventions, such as dietary advice, exercise, metformin, self-monitoring of weight gain, and probiotics for preventing GDM (Oostdam 2011), behaviour-modification interventions for preventing GDM (Skouteris 2014), lifestyle interventions for overweight and obese pregnant women for improving pregnancy outcomes, including GDM (Oteng-Ntim 2012), and 'nutritional manipulation in pregnancy' for preventing GDM (Rogozinska 2015). The methods of these reviews, and particularly their inclusion/exclusion criteria, differed to those employed in our review, and thus they have revealed some similar, and some contrasting findings.

For example, the Rogozinska 2015 review (which included 20 randomised trials) assessed diet-based, mixed approach (diet and lifestyle) interventions, and nutritional supplements (myo-inositol and probiotics) and did not find a clear difference in GDM risk overall (RR 0.67, 95% CI 0.39 to 1.15; six trials, 1479 women). With the same three trials included in our overweight and/or obese subgroup, the review similarly showed a reduced risk of GDM

in overweight or obese women with diet-based interventions (RR 0.40, 95% CI 0.18 to 0.86; three trials, 455 women). The Oteng-Ntim 2012 review (which included 13 randomised and six non-randomised trials, all in overweight and/or obese women), found that dietary and lifestyle interventions in pregnancy could reduce gestational weight gain (MD -2.21, 95% CI -2.86 to 1.59; 10 trials, 1228 women); this review also reported a trend towards a reduction in GDM risk (OR 0.80, 95% CI 0.58 to 1.10, six trials, 1011 women) (Oteng-Ntim 2012) specifically for overweight and/or obese women, as was seen in our review.

Skouteris 2014 (which included nine trials), did not pool data from individual studies in meta-analyses. Skouteris 2014 did however similarly conclude that the majority of trials incorporating 'behaviour change techniques' designed to prevent GDM as a primary or secondary outcome have not demonstrated clear effectiveness. Skouteris 2014 highlighted the need for further research to inform the combination of information delivery and behaviour-modification techniques used to prevent GDM. Oostdam 2011 (including 19 studies assessing a variety of interventions for GDM prevention) reported that dietary counselling reduced GDM compared with standard care (risk difference (RD) -0.05, 95% CI -0.10 to -0.01, seven trials, 813 women). Of note, the relevant meta-analysis in the Oostdam 2011 review, included data from some trials which were included in the Bain 2015 Cochrane review (where exercise advice or sessions were provided in addition to dietary advice). Further, the Oostdam 2011 review did not include a number of trials which have been included in our review (reported after its publication), and included data from the two intervention arms of Laitinen 2009 (one of which assessed dietary advice and a probiotic; and thus this arm was excluded from our review).

## AUTHORS' CONCLUSIONS

### Implications for practice

There is a limited and incomplete body of evidence from randomised trials assessing the effects of dietary advice interventions for preventing gestational diabetes (GDM), which is insufficient to guide practice.

Very low-quality evidence from five trials of dietary advice interventions during pregnancy suggests a possible reduction in the risk of GDM for women receiving dietary advice versus standard care (five trials); current evidence suggests that this reduction may be greater for women who are overweight and/or obese. Low-quality evidence from four trials suggests no clear difference in GDM risk between low- versus moderate- to high-GI dietary advice (four trials). Where reported, no clear differences were seen for the review's other primary outcomes (pre-eclampsia; perinatal mortality; large-for-gestational age; neonatal mortality and morbidity composite),

except for a possible reduction in pregnancy-induced hypertension for women receiving dietary advice. There were very little outcome data and few differences observed for the majority of the review's secondary outcomes.

For outcomes assessed using GRADE, the evidence was considered to be low to very low quality, with downgrading based on study limitations (risk of bias), imprecision, and inconsistency.

## Implications for research

In light of the limitations associated with the current evidence, further randomised controlled trials are warranted to determine the effects of dietary advice interventions during pregnancy on prevention of GDM and other relevant adverse health consequences for women and their babies. Future trials must be sufficiently powered, and well-designed to assess women's adherence and views, and to allow important differences in relevant clinical outcomes for women and babies to be detected, including longer-term infant, child and/or adult outcomes and those related to the use and costs of health services. In view of the subgroup analyses observing greater treatment effect in overweight and obese women, who are at higher risk of GDM, exploring treatment effect in high-risk subgroups could be of value. Such trials should aim to collect and report on core outcomes for GDM research, such as those that are pre-specified in the review.

Five additional trials have been identified as being planned or underway, and four are awaiting classification (pending the availability of further information). These trials are assessing a variety of dietary advice interventions during pregnancy, for preventing adverse health outcomes for women and their babies, including GDM, and will be considered for inclusion in the next update of this review.

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As part of the pre-publication editorial process, this review has been commented on by two peers (an editor and referee who is external to the editorial team), members of the Pregnancy and Childbirth Group's international panel of consumers and the Group's Statistical Adviser.

As stated in the previous version of this review, we thank Robert Fraser for his support in providing additional information regarding [Fraser 1983](#) and [Fraser 1988](#) and Robert Moses and Megan Barker for providing additional data from [Moses 2006](#).

For this update, we used the Cochrane Pregnancy and Childbirth core outcome set for reviews of diabetes in pregnancy, developed by the Cochrane Pregnancy and Childbirth Australian and New Zealand satellite.

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- \* Indicates the major publication for the study

## CHARACTERISTICS OF STUDIES

### Characteristics of included studies [ordered by study ID]

#### Clapp 1998

Methods	Randomised controlled trial.
Participants	20 women were randomised. <b>Setting:</b> teaching hospital in the USA. <b>Inclusion criteria:</b> healthy women who eventually completed an uncomplicated pregnancy (all participants were enrolled before conception). From the 1997 paper: "Briefly, at the time of entry, the 6 women eventually randomized to each of the dietary regimens were similar in age ( $35 \pm 1$ versus $34 \pm 1$ years), pre-conceptional weight ( $62.0 \pm 2.1$ versus $62.5 \pm 3.1$ kg), % body fat ( $20.7 \pm 1.9$ versus $20.5 \pm 1.9$ , and parity (0-1 versus 0-3)". <b>Exclusion criteria:</b> none specified.
Interventions	All those recruited commenced a weight-maintaining diet of low-GI carbohydrates in addition to a standardised exercise regimen preconception. At 8 weeks' gestation, participants were randomly assigned to either a high-GI/cafeteria diet or a low-GI/Aboriginal diet. During pregnancy all women were allowed to increase caloric intake according to appetite with advancing gestation <b>Low-GI dietary advice (Aboriginal diet) (n = 10)</b> Diet with carbohydrates from dense whole grain, fruits, beans and vegetables, dairy products etc, with a composition of 17% to 19% protein, 20% to 25% fat and 55% to 60% carbohydrate. Total intake: 35 to 45 kcal/kg of lean body mass <b>High-GI dietary advice (cafeteria diet) (n = 10)</b> Diet based on carbohydrates from highly processed grains, root vegetables, simple sugars, etc with a composition of 17% to 19% protein, 20% to 25% fat and 55% to 60% carbohydrate. Total intake: 35 to 45 kcal/kg of lean body mass
Outcomes	Outcomes included in this review: GDM; large-for-gestational age; gestational weight gain; fasting glucose at 24 to 28 weeks and 32 to 36 weeks; birthweight; head circumference at birth; length at birth; ponderal index at birth; adiposity (% body fat)
Notes	

#### Risk of bias

#### Risk of bias

Bias	Authors' judgement	Support for judgement
Random sequence generation (selection bias)	Unclear risk	No detail reported: quote - "A prospective randomized design".
Allocation concealment (selection bias)	Unclear risk	As above.
Blinding of participants and personnel (performance bias) All outcomes	High risk	Blinding was not detailed, however considered unlikely particularly for women and personnel in view of the intervention

**Clapp 1998** (Continued)

Blinding of outcome assessment (detection bias) All outcomes	Unclear risk	As above; blinding of outcome assessors not reported.
Incomplete outcome data (attrition bias) All outcomes	Low risk	No losses to follow-up reported. Data were analysed according to participants' randomly allocated group; all participants were included in the analyses
Selective reporting (reporting bias)	Unclear risk	In this 'Plenary Lecture' the methods are not separated from the experimental results; thus it is not possible to assess whether outcomes were pre-specified, and whether data for all pre-specified outcomes were reported. Many results (such as for GDM and large-for-gestational age reported only narratively in text)
Other bias	Unclear risk	Methods are largely not described, thus it is unclear as to whether there are other potential sources of bias

**Fraser 1983**

Methods	Randomised controlled trial.
Participants	25 women were randomised. <b>Setting:</b> Department of Dietetics, Northern General Hospital, Sheffield, UK. <b>Inclusion criteria:</b> healthy, non-obese primigravid European women in their second half of pregnancy, with non-pregnant weights < 110% of ideal for height, who had no family history of diabetes and "were free of the stigmata of potential diabetes". <b>Exclusion criteria:</b> none specified.
Interventions	<b>Dietary advice focused on high fibre (n = 13)</b> Women in the intervention/high dietary fibre group were interviewed at 27 weeks' gestation by a dietitian and were advised to reduce intakes of sucrose and white flour and to make as many high-fibre substitutions as possible, whilst aiming for a calorie intake of 2400. They were given diet and recipe sheets and tokens for free wholemeal bread to encourage compliance <b>Standard dietary advice (n = 12)</b> Women were given "standard advice" at an interview with a dietitian at 27 weeks' gestation (no further details of the advice provided), with a suggested calorie intake of 2400 All women were seen by the dietitian at their antenatal attendances
Outcomes	Outcomes included in this review: OGTT at 35 weeks; birthweight (centile); narrative text reported relating to gestational weight gain and neonatal anthropometry
Notes	Participants were selected to be at low risk of GDM on the basis of weight and past history We received further information from Dr Fraser regarding Fraser 1983. This resulted in the inclusion of additional data on birthweight centile in the review. Variance in extra information were assumed to be standard deviations

Fraser 1983 (Continued)

<i>Risk of bias</i>		<i>Risk of bias</i>
Bias	Authors' judgement	Support for judgement
Random sequence generation (selection bias)	Unclear risk	Not detailed.
Allocation concealment (selection bias)	Unclear risk	Quote: <i>"the randomisation was performed by opening sealed envelopes after the patients had agreed to participate"</i> (Unclear whether envelopes were consecutively numbered and opaque)
Blinding of participants and personnel (performance bias) All outcomes	High risk	Blinding was not detailed, however considered unlikely particularly for women and personnel in view of the intervention
Blinding of outcome assessment (detection bias) All outcomes	Unclear risk	As above.
Incomplete outcome data (attrition bias) All outcomes	Unclear risk	Unclear if there were any lost participants, attrition or exclusions
Selective reporting (reporting bias)	High risk	In the manuscript, results reported for diurnal plasma glucose concentration and insulin concentration (though numbers not reported for groups) only, and for the other outcomes a general statement was made: quote: <i>"results of the antenatal monitoring (including maternal weight gain and serum ferritin) and fetal anthropometry showed no significant differences between the groups"</i> .
Other bias	Unclear risk	Methods are largely not described, thus it is unclear as to whether there are other potential sources of bias

Laitinen 2009

Methods	Randomised controlled trial.
Participants	256 women were randomised. <b>Setting:</b> from April 2002 to November 2005, women were recruited from maternal welfare clinics in Turku, and neighbouring areas in South-West Finland <b>Inclusion criteria:</b> women < 17 weeks' gestation (including singleton and twin pregnancies) <b>Exclusion criteria:</b> women with metabolic or chronic diseases (e.g. diabetes) (allergic diseases (atopic eczema, allergic rhinitis or asthma) were allowed)



Interventions	<p><b>Dietary counselling focused on fat and fibre and probiotics (n = 85 randomised, 73 completed 6 and 12 month follow up)</b>  Women received dietary counselling (see below) and probiotics - containing <i>Lactobacillus rhamnosus</i> and <i>Bifidobacterium lactis</i> 10<sup>10</sup> colony-forming units/d each.</p> <p><b>Dietary counselling focused on fat and fibre and placebo (n = 86,73 completed 6-month follow-up and 69 completed 12-month follow-up)</b>  Women received dietary counselling (see below) and placebo capsules - containing microcrystalline cellulose and dextrose anhydrate (identical in appearance, smell and taste to the probiotics)</p> <p><b>Standard care/placebo (n = 85, 70 completed 6-month follow-up and 66 completed 12-month follow-up)</b>  Women received no additional dietary counselling, and the placebo capsules (see above)  <i>“All women attended communal dietary counselling provided by welfare clinics according to a national program, which consists of information of dietary guidelines through conversations and written material mediated by educated nurses.”</i></p> <p><b>All women:</b> visits occurred 3 times during pregnancy (mean of -13.0, 23.8, 33.9 weeks) and at 1, 6, 12, months postpartum. Dosing with standard content capsules commenced at first study visit and lasted until the end of exclusive breastfeeding. All capsules stored were stored 5° Celsius (and viability of probiotic capsules confirmed by regular analysis - blind). Adherence in consumption was assessed by interview.</p> <p><b>For the 2 dietary counselling groups:</b> counselling was given by a dietitian at each study visit, and aimed to modify dietary intake to conform with recommendations (Nordic Nutrition Recommendations) - with a particular focus on quality of dietary fat (saturated fatty acids (SFA) providing 10% or less of energy intake, monounsaturated (MUFA) 10% to 15% and polyunsaturated fatty acids (PUFA) 5% to 10%). Women were provided with readily available food products of favourable fat composition (e.g. low erucic acid rapeseed oil based margarines, spreads and salad dressings, Raisio plc, Raisio, Finland) and fibre contents (e.g. fibre-enriched pasta, breakfast muesli, and porridge cereals) to take home</p> <p>Dietary intake was assessed at each trimester using 3 day food diaries (energy and nutrient intakes calculated with a computerised program)</p>
Outcomes	<p>Outcomes included in this review: GDM; perinatal mortality; caesarean section; breastfeeding at 6 months; gestational weight gain; postpartum BMI; stillbirth; neonatal mortality; preterm birth (&lt; 37 weeks' gestation); preterm birth (&lt; 32 weeks' gestation); gestational age at birth; birthweight; head circumference at birth; length at birth; weight at 6 months; length at 6 months; head circumference at 6 months; skin-fold thickness at 6 months; systolic, diastolic and mean blood pressure at 6 months; heart rate at 6 months</p>
Notes	<p>In this review, we have only included 2 arms: the dietary counselling focused on fat and fibre and placebo arm, and the standard care/placebo arm</p>

**Risk of bias****Risk of bias**

Bias	Authors' judgement	Support for judgement
Random sequence generation (selection bias)	Low risk	Women were assigned to 3 study groups according to <i>“computer-generated block randomization of six women”</i> .

		The randomisation list was generated by a statistician not involved in recruitment or study visits
Allocation concealment (selection bias)	Low risk	Sealed envelopes contained subject numbers corresponding to numbered probiotics and placebo containers, and information on whether the subjects would receive dietary counselling. The envelopes were opened by the research nurse and nutritionist in the presence of each study subject in their order of recruitment (unknown whether envelopes were opaque)
Blinding of participants and personnel (performance bias) All outcomes	High risk	No blinding of the dietary intervention, due to its nature; blinding likely achieved for the capsules: quote: "Randomisation to receive probiotics...or placebo...in the dietary counselling groups took place in a double-blind manner, while the control group received placebo in a single-blind manner". The capsules were numbered according to the randomisation list by a member of the research group not involved with the conduct or reporting of the study
Blinding of outcome assessment (detection bias) All outcomes	Unclear risk	See above. Quote: "All personnel who handled or analysed blood samples were blind to the intervention"; not detailed for other clinical outcomes however, and there was no blinding for the dietary component of the intervention
Incomplete outcome data (attrition bias) All outcomes	Low risk	From Aaltonen et al. 2008: of the 256 mothers participating in the study 216 completed 6-month follow-up From Laitinen et al. 2009: of the 256 mothers participating in the study, 208 completed the 12-month follow-up; reasons for discontinuation: Group 1 (12 discontinued): miscarriage (3); illness in mother (2); illness in child (4); unwilling (1); moved (1); unknown (3): n = 73 (86%) Group 2 (17 discontinued): miscarriage (2); illness in mother (3); illness in child (2); unwilling (6); moved (3); unknown (1): n = 69 (80%) Group 3 (19 discontinued): illness in mother (3); illness in child (4); unwilling (9); moved (0); unknown (3): n = 66 (78%) A further 23 women were again pregnant at the end of the follow-up and were excluded from the postpartum analysis From Luoto et al. 2010: "altogether, 256 pregnant women were recruited and 238 of them continued the study throughout pregnancy. Three twin pairs were excluded from the growth follow-up. Of the 241 children delivered, 191

Laitinen 2009 (Continued)

		<p>completed the 24 months' follow-up" (Group 1 n = 67, Group 2 n = 63; Group 3 n = 61).</p> <p>From Hoppu et al. 2013: "Altogether 256 mothers started the study at the first trimester of pregnancy, and 208 mothers and their infants were still participating at the one-year study visit and 185 and 127 at 2 and 4 years, respectively". The numbers of mother and infants and mothers with reported outcome data at various time-points differ between papers</p>
Selective reporting (reporting bias)	Unclear risk	<p>Whilst the majority of outcomes were pre-specified, a number of outcomes not pre-specified were reported, including breastfeeding, Apgar score at 5 minutes. The results for the GCT are hard to interpret, with no clear numbers for the women screened in each group. Plasma glucose values are presented in text, with means but no standard deviations. It is also difficult to interpret and use the infant data (birthweight; gestation; birth height; head circumference; Apgar at 5 minutes) reported with 'ranges' of infants, rather than single value (n)) (Laitinen et al. 2009)</p>
Other bias	Low risk	No other obvious sources of bias identified.

Markovic 2016

Methods	Randomised controlled trial: GI Baby 3.
Participants	<p>147 women were randomised.</p> <p><b>Setting:</b> Antenatal clinic at Royal Prince Alfred Hospital, Camperdown, New South Wales, Australia, between January 2011 and October 2012</p> <p><b>Inclusion criteria:</b> women &gt; 18 years of age, between 12 to 20 weeks of gestation, at high risk of GDM (at least 1 of the following risk factors: age &gt; 35 years, first degree relative with T2DM, pre-pregnancy BMI <math>\geq</math> 30, past history of GDM or glucose intolerance, history of a previous baby &gt; 4000 g, belonging to a high-risk ethnic group (Aboriginal or Torres Strait Islander, Polynesian, Middle Eastern, Indian, Asian) with an otherwise healthy singleton pregnancy</p> <p><b>Exclusion criteria:</b> women with pre-existing diabetes or special dietary requirements (including vegetarianism/veganism); women with a fasting BGL <math>\geq</math> 5.9 mmol/L or 2-hour reading <math>\geq</math> 11.1 mmol/L in an initial (&lt; 20 week) OGTT</p>
Interventions	<p><b>Low-GI dietary advice (n = 76 randomised, 72 analysed)</b> Target GI <math>\leq</math> 50.</p> <p><b>High-fibre, moderate-GI dietary advice (n = 71 randomised, 67 analysed)</b> Similar to Australian population average (target GI 60).</p> <p><b>All women</b> Women were asked to complete a 3-day food record at baseline and again at 34 to 36 weeks' gestation Women attended a total of 5 individual dietary consultations with a dietitian (14-20,</p>

	<p>18-24, 22-28, 26-32, 34-36 weeks' gestation)</p> <p>1) Visit 1: women randomised to either low-GI diet or high-fibre, moderate-GI diet; similar macronutrient composition recommended (protein 15% to 25% total energy intake, fat 25% to 39% total energy intake, carbohydrate 40% to 45% total energy intake), and both diets provided all the essential nutrients for pregnancy (other than iron and iodine, which were supplemented as appropriate). Baseline 3-day food record served as the basis of dietary counselling, where written information regarding suitable low-GI or high-fibre foods and pregnancy nutrition was provided</p> <p>2) Visits 2, 3, 4: 4-stage multiple-pass 24-hour recalls were performed to check compliance (low-GI group: <math>\leq 50</math>; <math>&gt; 50</math> for high-fibre group); for women who were non-compliant, suitable alternative foods were encouraged, and a selection of recipes provided</p> <p>Women were also provided with food samples containing key foods for the assigned diet at all 5 consultations (supplementary baskets)</p>
Outcomes	<p>Outcomes included in this review: GDM; large-for-gestational age; caesarean section; gestational weight gain; macrosomia; small-for-gestational age; gestational age at birth; birthweight and z score; ponderal index at birth; adiposity at birth (% body fat); growth at follow-up; neonatal intensive care unit admission</p>
Notes	

<i>Risk of bias</i>		<i>Risk of bias</i>
<b>Bias</b>	<b>Authors' judgement</b>	<b>Support for judgement</b>
Random sequence generation (selection bias)	Low risk	Quote: "computer-generated random numbers".
Allocation concealment (selection bias)	Unclear risk	Quote: "which were unpredictable and concealed from the recruiter".
Blinding of participants and personnel (performance bias) All outcomes	High risk	Women were aware of group allocation; quote: "Apart from the study dietitian... who provided dietary education, all study personnel were blinded to dietary assignment".
Blinding of outcome assessment (detection bias) All outcomes	Low risk	As above; and quote: "A biostatistician blinded to the dietary allocation performed the statistical analysis".
Incomplete outcome data (attrition bias) All outcomes	Low risk	Of the 76 women in the intervention group 4 were excluded from the analyses (1 premature birth, 1 termination, 2 moved overseas); of the 71 women in the control group 4 were excluded from the primary intention-to-treat analysis (1 overt diabetes, 1 twin pregnancy, 1 termination, 2 moved interstate). A further 14 (7 in each group) withdrew after commencing (4: too busy, 2: lost

Markovic 2016 (Continued)

		to follow-up; 4: did not wish to follow the diet; 4: no reason) leaving 125 in a “completers analysis”.
Selective reporting (reporting bias)	Low risk	No clear evidence of selective reporting, though the results do not clearly report data for maternal complications “nor was there any difference in mode of delivery or maternal complications (data not presented)”.
Other bias	Unclear risk	Baseline characteristics balanced between groups, except for family history of T2DM, which was more common in the low-GI group

Moses 2006

Methods	Quasi-randomised controlled trial.
Participants	<p>70 women were randomised.</p> <p><b>Setting:</b> Antenatal clinic at Wollongong Hospital, Wollongong, NSW, Australia.</p> <p><b>Inclusion criteria:</b> healthy, pregnant women from antenatal clinic of 2 obstetricians at Wollongong Hospital; 21-40 years old, with a singleton pregnancy at 12-16 weeks’ gestation; non-smokers, with no more than 1 alcoholic drink/day. (All women were “white”.)</p> <p><b>Exclusion criteria:</b> individually assessed: any problem associated with glucose metabolism/insulin resistance/interference with ability to follow dietary instructions (e. g. PCOS, Crohn’s disease)</p>
Interventions	<p><b>Low-GI dietary advice (n = 35 randomised, 32 analysed)</b></p> <p>Women were seen by dietitian 5 times during pregnancy; at visit 1, a 3 day food record and diet history were taken and women were measured/weighed; at visit 2 (1 week later) , women received dietary education for low-GI diet with 33% fat, 55% carbohydrate, based on verified low-GI foods including pasta, brand-name breads and breakfast cereals with high-fibre content; at visits 3 (22 weeks) and 4 (30 weeks), 24 hour diet recall was taken; at visit 5 (36 weeks) a second 3 day food record and diet history were taken</p> <p><b>Moderate- to high-GI dietary advice (n = 35, 32 analysed)</b></p> <p>Women were also seen by dietitian 5 times during their pregnancy; at visit 1, a 3 day food record and diet history were taken and women were measured/weighed; at visit 2 (1 week later), women received dietary education for moderate to high-GI diet (high fibre, low sugar) with 33% fat, 55% carbohydrate; at visits 3 (22 weeks) and 4 (30 weeks), 24 hour diet recall was taken; at visit 5 (36 weeks) a second 3 day food record and diet history were taken</p> <p>For all women: No specific or individual recommendations were made about the intake of total energy, fibre, and fat. During visits, the dietitian referred to the diets as the “high-fiber, low-sugar” diet or the “low-GI” diet. Women were provided with a booklet that outlined the carbohydrate choices and the food amounts that constituted 1 serving. To encourage compliance with both diets, key foods were provided in a monthly hamper.</p>

Moses 2006 (Continued)

	The dietitian also provided information on the whole diet to ensure energy and overall nutrient balance and was available for telephone queries outside of scheduled visits
Outcomes	Outcomes included in this review: GDM; large-for-gestational age; caesarean section; operative vaginal birth; gestational weight gain; adherence to the intervention; fasting glucose at 36 weeks; views of the interventions; Apgar score < 7 at 5 minutes; small-for-gestational age; gestational age at birth; birthweight; head circumference at birth; length at birth; ponderal index at birth
Notes	Dr Robert Moses and Megan Barker provided further information regarding <a href="#">Moses 2006</a> .

<i>Risk of bias</i>		<i>Risk of bias</i>
<b>Bias</b>	<b>Authors' judgement</b>	<b>Support for judgement</b>
Random sequence generation (selection bias)	High risk	Quasi-randomised; women were assigned alternately to the 2 intervention and control groups
Allocation concealment (selection bias)	High risk	As above.
Blinding of participants and personnel (performance bias) All outcomes	High risk	No blinding of subjects or investigators due to the nature of the interventions. Quotes: whilst "study personnel were not blinded to dietary assignment", they were "aware of the need for impartiality and equivalent treatment;" and "the obstetric health care providers were not specifically informed of the diet assignment".
Blinding of outcome assessment (detection bias) All outcomes	Unclear risk	Blinding not specifically mentioned for outcome assessors (only "study personnel" and "obstetric health care providers").
Incomplete outcome data (attrition bias) All outcomes	Unclear risk	Whilst there were 70 women randomised, in the low-GI group, 3 women were excluded from analyses: 2 women withdrew as they were unwilling to follow the diet, and 1 gave birth before the final visit (< 36 weeks). In the high-GI group 5 women were excluded: 1 woman was unwilling to follow the diet, 1 lost to follow up, and 3 women had miscarriages. Data were provided for the 62/70 women who completed the study 19/62 women (30%) did not wish to participate in follow up (those women had a similar BMI and age, but a higher parity than the 43 women who agreed to participate)

Moses 2006 (Continued)

Selective reporting (reporting bias)	Unclear risk	No clear evidence of selective reporting; however, with no access to a trial protocol, it is not possible to confidently assess reporting bias and some results were reported incompletely: quote: “ <i>The analysis of the diet histories produced similar findings (data not shown)</i> ”.
Other bias	Unclear risk	Quote: “ <i>Women who were assigned to the HGI diet group had a slightly higher BMI (P = 0.04) and higher HOMA2- cell function (P = 0.07) than did women in the LGI diet group (Table 1)</i> ”. No other obvious sources of bias.

Moses 2014

Methods	Randomised controlled trial: PREGGIO.
Participants	691 women were randomised. <b>Setting:</b> South Eastern Sydney Illawarra Area Health Service (women with intention to deliver at Wollongong Hospital or Illawarra Private Hospital under the care of 2 specific private obstetricians). New South Wales, Australia, from February 2010 to March 2013 <b>Inclusion criteria:</b> women attending for their initial obstetric/midwife appointment at < 20 weeks’ gestation, with a singleton pregnancy, aged ≥ 18 years old, able to read and understand a consent form in English, and able to comply with visit schedules <b>Exclusion criteria:</b> women with known diabetes or previous GDM, special dietary needs or any medical conditions that may affect metabolic status (e.g. thyroid disorders) or use of medication likely to affect body weight
Interventions	<b>Low-GI dietary advice (n = 354 randomised, 296 analysed)</b> Women received a set of booklets that included information of choices for and serving sizes of carbohydrate-rich foods, with specific information on LGI alternatives for relevant food groups, and specific dietary goals were provided to each participant focusing on differentiating between carbohydrate rich foods <b>Healthy eating dietary advice (n = 337 randomised, 280 analysed)</b> Women received a set of booklets, and were counselled to follow a conventional healthy diet with recommended foods and serving sizes as noted in the Australian Guide to Healthy Eating and were not given any guidance on the GI Both groups: women received a detailed dietary education tailored for the assigned diet and their individual requirements for pregnancy; there was no intended difference in macronutrient distribution in diets. Women in both groups were counselled to adopt diets that were consistent with nationally recommended nutritional intake for pregnant women and recommendations of the Australian Guide to Healthy Eating For all women there were 4 contact points in pregnancy. 1) First visit: at the first visit, a 3-day food record was reviewed, and details clarified. Diet education was given, specific to assigned group by the research dietitian 2) Phone call: a phone call was made at 4 weeks after the initial visit to ensure adherence to the prescribed diet and goals set, identify barriers and other dietary issues/concerns 3) Midway assessment: dietitian reviewed women face-to-face at 28 weeks before their

	<p>obstetric appointment to monitor progress and address any new issues</p> <p>4) Final visit: as late as possible (minimum 34 weeks) the dietitian collected and reviewed the final 3-day food record, and measured final weight</p> <p>The research dietitian was available for telephone queries outside of scheduled visits. An email was sent monthly (5 in total) to all women who provided an email address, with content dependent on group (nutrition tips and recipes)</p>
Outcomes	Outcomes included in this review: GDM; large-for-gestational age; gestational weight gain; adherence to the intervention; views of the intervention; macrosomia; small-for-gestational age; gestational age at birth; birthweight; length at birth; ponderal index at birth
Notes	

**Risk of bias****Risk of bias**

Bias	Authors' judgement	Support for judgement
Random sequence generation (selection bias)	Low risk	Quote: "Women were randomly assigned by using computer-generated random numbers".
Allocation concealment (selection bias)	Unclear risk	Quote: "The allocation sequence was unpredictable and concealed from the research dietitian".
Blinding of participants and personnel (performance bias) All outcomes	High risk	Quote: "Study personnel were not blinded to the dietary assignment but were aware of the need for impartiality and equivalent treatment".
Blinding of outcome assessment (detection bias) All outcomes	Unclear risk	Quote: "Obstetric care providers were not specifically blinded to the study allocation but were also not informed"; unclear if/how lack of blinding would have impacted on outcome assessment
Incomplete outcome data (attrition bias) All outcomes	Unclear risk	Of 354 women in intervention group, 58 were excluded after randomisation (27 because they developed GDM; quote: "Women with diagnosed GD were withdrawn from the study and treated conventionally"). Of 337 in the control group, 57 women were excluded after randomisation (28 because they developed GDM). Loss was approximately 17%, though was reasonably balanced between groups, with similar reasons between groups. Note: these data for GDM diagnosis have been included in the relevant meta-analysis (Analysis 2.1).



Selective reporting (reporting bias)	Unclear risk	Trial was retrospectively registered (no published protocol available). Table 1 presents “selected fetal outcomes”; some additional outcomes mentioned in Discussion (e.g. GTT glucose concentrations “data not shown”) and mode of birth mentioned in published abstract but not reported; development of GDM was listed as a primary outcome in the trial registration, but not reported as such in the manuscript (childhood obesity at 2 years also listed as an outcome; may be the subject of future paper/s)
Other bias	Low risk	No baseline imbalance apparent. No other obvious sources of bias

Quinlivan 2011

Methods	Randomised controlled trial.
Participants	<p>132 women were randomised.</p> <p><b>Setting:</b> the maternity service of a public general hospital serving a socio-economically disadvantaged area in Melbourne, Australia</p> <p><b>Inclusion criteria:</b> pregnant women, with a fetus with no known anomalies, who spoke English, did not intend to relinquish their infant, did not have a multiple gestation, who were able to attend hospital for antenatal care, and were overweight (BMI 25 to 29.9) or obese (BMI &gt; 29.9)</p> <p><b>Exclusion criteria:</b> no further exclusions detailed.</p> <p>Women who experienced a later fetal loss were withdrawn.</p>
Interventions	<p><b>Dietary advice focused on healthy eating (n = 67 randomised, 63 analysed)</b></p> <p>A ‘four-step multidisciplinary approach’ was used (guided by key elements of the Australian National Health and Medical Research Council’s recommendations for the management of overweight/obesity in adults) at a study-specific antenatal clinic (“obesity clinic”).</p> <ol style="list-style-type: none"> <li>1. Continuity of care by a single maternity care provider.</li> <li>2. Assessment of weight gain at each antenatal visit.</li> <li>3. A brief intervention (5 minutes) by a food technologist before each visit</li> <li>4. An assessment by a clinical psychologist (an individualised solution-focused plan was implemented if difficulties identified)</li> </ol> <p>Women diagnosed with GDM remained in this clinic for care, but were treated with identical clinical care guidelines as women in the control group</p> <p><b>Control (n = 65 randomised, 61 analysed)</b></p> <p>Women received standard obstetric antenatal care (routine public care), with access to high-risk clinics if indicated on medical grounds. Women diagnosed with GDM were referred to a public obstetric diabetes clinic for ongoing care</p> <p>All other clinic protocols across the 2 clinics were identical</p>

Outcomes	Outcomes included in this review: GDM; gestational weight gain; birthweight	
Notes		
<b>Risk of bias</b>		<b>Risk of bias</b>
Bias	Authors' judgement	Support for judgement
Random sequence generation (selection bias)	Low risk	A computer-generated number sequence was used (with stratification by category - overweight or obese)
Allocation concealment (selection bias)	Low risk	Computer-generated, numbered, sealed, opaque envelopes were used. The envelopes were only opened by the midwife after the enrolment was completed
Blinding of participants and personnel (performance bias) All outcomes	High risk	Personnel and participants were not blinded due to the nature of the intervention
Blinding of outcome assessment (detection bias) All outcomes	Low risk	Outcome data for the mother and infant were 'audited' by a nurse, independent of clinical care pathways, and blind to group allocation. It was not clear if weight measurements, OGTTs, etc. were performed/recorded by personnel blinded to group allocation
Incomplete outcome data (attrition bias) All outcomes	Low risk	4 women from each group were excluded from the analysis following randomisation as they "withdrew from study". No other losses, attritions or exclusions detailed.
Selective reporting (reporting bias)	Unclear risk	Data for all pre-specified outcomes (in the trial manuscript) were provided, however very few outcomes (3) were pre-specified. Additional outcomes (changes in diet and serious adverse events) were reported but not pre-specified in methods. The outcomes reported do not correspond to all outcomes specified in the trial registration (e.g. breastfeeding is not reported; nor are 'pooled adverse events in the fetus or newborn'), and there is no published trial protocol
Other bias	Low risk	No other obvious sources of bias identified.

Thornton 2009

Methods	Randomised controlled trial.
Participants	257 women were randomised. <b>Setting:</b> urban, public obstetric clinics in 3 tertiary care medical centres in the USA (Morristown Memorial Hospital, St Luke's-Roosevelt Hospital Centre, Jamaica Hospital Medical Centre) from June 1998 to May 2005 <b>Inclusion criteria:</b> pregnant women with single fetus, 12 to 28 weeks' gestation, BMI $\geq 30$ <b>Exclusion criteria:</b> women with pre-existing diabetes, hypertension, or chronic renal disease
Interventions	<b>Dietary advice focused on healthy eating (n = 124 randomised, 116 analysed)</b> Nutritional monitoring. Women were prescribed a balanced nutritional diet based on their weight at study entry and were asked to record all food and drink consumed each day in a food diary; these records were reviewed at each prenatal visit by the physician. Women were counselled at least once by registered dietitian regarding conventional nutrition guidelines, with more detailed dietary intake advice compared with the control group women; the nutrition regimen was similar to that used for GDM at the time: 18 to 24 kcal/kg consisting of 40% carbohydrates, 30% protein and 30% fat; at least 2000 calories <b>Standard care (n = 133, 116 analysed)</b> Usual care (unmonitored). Women were advised to eat to appetite following general prenatal dietary guidelines. Women were counselled at least once by registered dietitian regarding conventional nutrition guidelines Women in both groups were weighed at each visit, and were encouraged to walk for 30 minutes each day
Outcomes	Outcomes included in this review: GDM; pre-eclampsia; pregnancy-induced hypertension; caesarean section; induction of labour; postpartum haemorrhage; postpartum infection; gestational weight gain; postpartum weight loss at 6 weeks; preterm birth (< 37 week's gestation); Apgar score < 7 at 5 minutes; macrosomia; gestational age at birth; birthweight
Notes	

*Risk of bias*

*Risk of bias*

Bias	Authors' judgement	Support for judgement
Random sequence generation (selection bias)	Low risk	Random number table.
Allocation concealment (selection bias)	Low risk	Sealed sequentially numbered envelopes (no comment re: opaque/not opaque)
Blinding of participants and personnel (performance bias) All outcomes	High risk	Blinding not detailed, and considered unlikely particularly for women and personnel in view of the intervention

Thornton 2009 (Continued)

Blinding of outcome assessment (detection bias) All outcomes	Unclear risk	As above.
Incomplete outcome data (attrition bias) All outcomes	Unclear risk	25/257 lost to follow-up (8/124 in the intervention group and 17/133 in the control group; some suggestion of greater loss in control group)
Selective reporting (reporting bias)	Unclear risk	Whilst the majority of pre-specified outcomes discussed in the methods were subsequently reported, no access to a trial protocol to confidently assess selective reporting. Some outcomes, e.g. shoulder dystocia, were mentioned and then not reported
Other bias	Low risk	Groups appeared balanced at baseline for demographic data although women in the control group were heavier and had higher BMI at baseline (P = 0.06). No other obvious sources of bias

Vitolo 2011

Methods	Randomised controlled trial.
Participants	315 women were randomised from January 2007 to May 2008. <b>Setting:</b> a health unit in the metropolitan region of Porto Alegre, in Rio Grande do Sul, Brazil (the unit mainly takes care of a population of low socio-economic status) <b>Inclusion criteria:</b> pregnant women with a gestational age between 10 to 29 weeks' gestation, belonging to the prenatal care unit of the health unit <b>Exclusion criteria:</b> women who had a positive HIV test, who had previously been diagnosed with diabetes, who had hypertension, anaemia or other conditions requiring a special diet, or who were > 35 years
Interventions	<b>Dietary advice focused on healthy eating (n = 159 randomised, 152 analysed*)</b> Women received dietary counselling, according to their nutritional status (regarded as low weight, eutrophic, or overweight). Women received dietetic counselling, with the aim of adjusting the speed of weight gain and improving the quality of food consumed. Women received 1 interview per month after the first session for reinforcement <b>Standard care (n = 162 randomised, 155 analysed*)</b> Women were instructed to follow the routine of the health service facility, and received no dietetic counselling (however women in low weight or overweight categories were advised to seek assistance)
Outcomes	Outcomes included in this review: gestational weight gain; clinical complications (GDM, hypertension, pre-eclampsia, prematurity and low newborn weight)
Notes	*The numbers in the flow diagram appear to be incorrect (i.e. with 159 and 162 totaling more than the 315 women that were reported to be randomised) Emailed Dr Vitolo on 16/06/2016 to request data on the components of the composite

		outcome 'clinical complications'
<i>Risk of bias</i>		<i>Risk of bias</i>
Bias	Authors' judgement	Support for judgement
Random sequence generation (selection bias)	Unclear risk	Quote from translation - women "were randomized by means of a dark pouch with two equal sized cubes containing the term intervention in one and control in the other".
Allocation concealment (selection bias)	Unclear risk	As above.
Blinding of participants and personnel (performance bias) All outcomes	High risk	Quote from translation - "After randomization, the procedure that was pertinent to the group was realized". No further details regarding blinding were provided; blinding considered unlikely in view of the intervention
Blinding of outcome assessment (detection bias) All outcomes	Unclear risk	As above.
Incomplete outcome data (attrition bias) All outcomes	Unclear risk	315 women were randomised; 7 were excluded from the intervention group (3 'missing', 1 change of health unit, and 3 premature births) and 9 from the control group (3 'missing', 1 change of health unit, and 5 premature births). Women who had their children prematurely, not completing the last interview, were considered 'losses' in the study and were excluded  The values in the flow diagram do not appear to be accurate, with 315 women being randomised, 159 to the intervention and 162 to the control (a total of 321, not 315)
Selective reporting (reporting bias)	Unclear risk	The methods specify data that were obtained, including: newborn weight, length, gestational age, cephalic perimeter, Apgar scores at 1 and 5 minutes, mode of birth, gestational complications, diabetes and gestational hypertension. From the translation, data for these outcomes did not however appear to be clearly reported
Other bias	Unclear risk	The values in the flow diagram do not appear to be accurate. Somewhat difficult to determine other potential biases from the translation. Data for maternal weight gain also difficult to interpret as reported by 'nutritional state' groups only (low weight, eutrophic, excess weight)

Walsh 2012

Methods	Randomised controlled trial: ROLO.
Participants	<p>800 women were randomised.</p> <p><b>Setting:</b> National Maternity Hospital, Dublin, Ireland from January 2007 to January 2011</p> <p><b>Inclusion criteria:</b> women who were secunda gravida, if their first infant weighted &gt; 4000 g, were recruited at their first antenatal visit</p> <p><b>Exclusion criteria:</b> women with any underlying medical disorders, including women with a previous history of GDM, women on any drugs, and women unable to give full informed consent. Women &lt; 18 years old, at a gestational age &gt; 18 weeks and who had multiple pregnancies were excluded</p>
Interventions	<p><b>Dietary advice focused on healthy eating/low-GI diet (n = 394 randomised, 372 analysed)</b></p> <p>Women attended 1 dietary session (mean gestational age of 15.7 weeks) in groups of 2 to 6 that lasted for 2 hours with a research dietitian. Women received advice on general health eating guidelines for pregnancy following the food pyramid, and the remainder of the education session focused on GI (definition, concept and rationale) . Women were encouraged to choose as many low-GI foods as possible and exchange high-GI carbohydrates for low-GI alternatives. Women were advised not to reduce their total caloric intake. The research dietitian met with the women at 28 and 34 weeks for reinforcement of the low-GI diet and to answer any dietary queries the women had</p> <p><b>Standard care (n = 406 randomised, 387 analysed)</b></p> <p>Women received routine antenatal care which did not involve any formal dietary advice or specific advice about gestational weight gain</p> <p>All women completed 3 food diaries, of 3 days each; 1 before dietary intervention and 1 in the second and third trimesters of pregnancy</p>
Outcomes	<p>Outcomes included in this review: GDM; caesarean section; induction of labour; perineal trauma; postpartum haemorrhage; breastfeeding at 3 months; gestational weight gain; behaviour changes associated with the intervention; fasting glucose at 28 weeks, and <math>\geq 5.1</math> mmol/L; GCT at 28 weeks and <math>&gt; 7.8</math> mmol/L; sense of well-being; postnatal weight retention; return to pre-pregnancy weight; stillbirth; preterm birth (&lt; 37 weeks' gestation) ; preterm birth (&lt; 32 weeks' gestation); macrosomia; shoulder dystocia; gestational age at birth; birthweight; head circumference at birth; length at birth; ponderal index at birth; adiposity at birth: skin-folds; weight at 3 months</p>
Notes	

<i>Risk of bias</i>		<i>Risk of bias</i>
Bias	Authors' judgement	Support for judgement
Random sequence generation (selection bias)	Low risk	A computer-generated random sequence was used.
Allocation concealment (selection bias)	Low risk	Sealed, opaque envelopes were used.

Walsh 2012 (Continued)

Blinding of participants and personnel (performance bias) All outcomes	High risk	Blinding not detailed, and considered unlikely particularly for women and personnel in view of the intervention
Blinding of outcome assessment (detection bias) All outcomes	Unclear risk	As above for most outcomes, though blinded sonographers made ultrasound measurements
Incomplete outcome data (attrition bias) All outcomes	Low risk	Reasons for losses to follow up were documented and similar between groups (10 women in each group 'opted out'; 1 in each group discontinued the intervention; 2 women in each group were found to have twins; 9 women in the intervention group and 6 in the control group had early pregnancy losses). Therefore 759 (372 in the intervention group and 387 in the control group) of the 800 (95%) women randomised were included in the final analyses
Selective reporting (reporting bias)	Low risk	Outcomes reported as per published trial registration and/or protocol (across a number of separate manuscripts). Data for mode of birth were not reported in Walsh et al. 2012, and for caesarean rate no data were presented: "We found no significant difference in the rate of caesarean delivery between the two groups"; however data were reported in Walsh et al. 2015.
Other bias	Low risk	No other obvious sources of bias identified. Characteristics of the groups were similar at baseline

Wolff 2008

Methods	Randomised controlled trial.
Participants	<p>73 women were recruited (group numbers below include 7 fewer women who became "ineligible").</p> <p><b>Setting:</b> Department of Clinical Nutrition, Hvidovre Hospital and the Department of Obstetrics and Gynecology, Herlev Hospital (Frederiksberg and Copenhagen), Denmark</p> <p><b>Inclusion criteria:</b> non-diabetic, non-smoking Caucasian, obese pregnant women were recruited, with a BMI <math>\geq</math> least 30. Women were recruited in early pregnancy (15 <math>\pm</math> 3 weeks of gestation) from the register of newly diagnosed pregnancies</p> <p><b>Exclusion criteria:</b> women who smoked, who were aged &lt; 18 years, or &gt; 45 years, who had a multiple pregnancy, or had any medical complications known to affect fetal growth adversely or to indicate limitation of weight gain</p>

Interventions	<p><b>Dietary advice focused on healthy eating (n = 28 included, 23 analysed)</b>  Women in the intervention group received 10 consultations of 1 hour each with a trained dietitian during pregnancy. Women were instructed to eat a healthy diet according to the official Danish dietary recommendations (fat intake: max 30 energy percent (E%), protein intake: 15 to 20 E%, carbohydrate intake: 50 to 55 E%); the energy intake was restricted based on individually estimated energy requirements, and estimated energetic cost of fetal growth</p> <p><b>Standard care (n = 38 included, 27 analysed)</b>  Women received no consultations with the dietitian and no restrictions on energy intake or gestational weight gain</p> <p>For all women: 7 day weighed food records were obtained at inclusion, 27 weeks and 36 weeks. For the intervention group, these records served as a tool to identify unhealthy eating patterns and give individualised suggestions for improvement. All women received dietary supplements to ensure sufficient intake of vitamins and trace elements (an emphasis on iron and folic acid intake). All women followed the routine clinical schedule</p>
Outcomes	Outcomes included in this review: GDM; pregnancy-induced hypertension; pre-eclampsia; caesarean birth; gestational weight gain; gestational age at birth; birthweight; head circumference at birth; length at birth
Notes	

<i>Risk of bias</i>		<i>Risk of bias</i>
Bias	Authors' judgement	Support for judgement
Random sequence generation (selection bias)	Low risk	Quote: "The computerized randomization".
Allocation concealment (selection bias)	Unclear risk	No details provided.
Blinding of participants and personnel (performance bias) All outcomes	High risk	The women and dietitians were not blinded.
Blinding of outcome assessment (detection bias) All outcomes	Unclear risk	Quote: "The physicians and midwives were blinded in regard to the treatment assignment, and the women were asked not to reveal the allocation by the randomization"; unclear whether this was successfully achieved.
Incomplete outcome data (attrition bias) All outcomes	High risk	73 women were recruited to the study; 7 developed "conditions that made them ineligible to continue participation" (spontaneous abortion, twin pregnancy, smoker, bedridden, diagnosis of GDM at inclusion). It was somewhat unclear whether these exclusions were pre or post randomisation; the unbalanced groups (n = 28 and n = 38 suggested this was following randomisation). A further 13 women dropped out of the study due to lack



		of time, or disappointment due to being in the control group. 3 additional women developed GDM in the control group were excluded from the analyses (apart from GDM incidence). 50 women were followed to delivery (23 in the intervention group; 27 in the control group). There were missing data for blood samples (3/50), and weight measurements (15/50) postpartum; <i>“The analyses were subsequently controlled for impact of missing values by replacing these with average of the entire group to ensure that the statistical test did not differ, significantly”</i> .
Selective reporting (reporting bias)	Unclear risk	Whilst the majority of pre-specified outcomes discussed in the methods were subsequently reported, data for Apgar scores were not, nor ‘methods of delivery’ (data for caesarean delivery were only reported). No access to a trial protocol to confidently assess selective reporting
Other bias	Low risk	No other obvious sources of bias identified.

BGL: blood glucose level

BMI: body mass index

GCT: glucose challenge test

GDM: gestational diabetes mellitus

GI: glycaemic index

HGI: high glycaemic index

LGI: low glycaemic index

OGTT: oral glucose tolerance test

T2DM: type 2 diabetes mellitus

### Characteristics of excluded studies [ordered by study ID]

Study	Reason for exclusion
<a href="#">Althuisen 2013</a>	This randomised controlled trial assessed a combined diet and exercise intervention (which is the focus of another Cochrane review ( <a href="#">Bain 2015</a> )).
<a href="#">Asbee 2009</a>	This randomised controlled trial assessed a combined diet and exercise intervention (which is the focus of another Cochrane review ( <a href="#">Bain 2015</a> )).
<a href="#">Asemi 2013</a>	This randomised controlled trial assessed a probiotic intervention (which is the focus of another Cochrane review ( <a href="#">Barrett 2014</a> )).
<a href="#">Brand-Miller 2007</a>	Correspondence with Dr Brand-Miller 02/02/2016 indicated this trial was not undertaken due to lack of funding

(Continued)

<a href="#">Dodd 2014</a>	This randomised controlled trial assessed a combined diet and exercise intervention (which is the focus of another Cochrane review ( <a href="#">Bain 2015</a> )).
<a href="#">Facchinetti 2013</a>	This randomised controlled trial assessed myo-inositol (which is the focus of another Cochrane review <a href="#">Brown 2015</a> )).
<a href="#">Fraser 1988</a>	This was a cross-over trial.
<a href="#">Hellenes 2015</a>	This randomised controlled trial assessed an exercise intervention (which is the focus of another Cochrane review ( <a href="#">Han 2012</a> )).
<a href="#">Hui 2006</a>	This randomised controlled trial assessed a combined diet and exercise intervention (which is the focus of another Cochrane review ( <a href="#">Bain 2015</a> )).
<a href="#">King 2013</a>	This was a cross-over trial.
<a href="#">Korpi-Hyovalti 2012</a>	This randomised controlled trial assessed a combined diet and exercise intervention (which is the focus of another Cochrane review ( <a href="#">Bain 2015</a> )).
<a href="#">Krummel 2009</a>	This randomised controlled trial is assessing docosahexaenoic acid (which is the focus of another Cochrane review ( <a href="#">Makrides 2006</a> )).
<a href="#">Laitinen 2015</a>	This randomised controlled trial is assessing fish oil and probiotics (which are the focus of 2 other Cochrane reviews ( <a href="#">Brown 2015</a> ; <a href="#">Makrides 2006</a> )).
<a href="#">Lesser 2015</a>	This was a cross-over trial.
<a href="#">Lindsay 2014</a>	This randomised controlled trial assessed myo-inositol (which is the focus of another Cochrane review <a href="#">Brown 2015</a> )).
<a href="#">Liu 2013</a>	This randomised controlled trial is assessing magnesium supplementation (which is the focus of another Cochrane review ( <a href="#">Makrides 2014</a> )).
<a href="#">Luoto 2011</a>	This randomised controlled trial assessed a combined diet and exercise intervention (which is the focus of another Cochrane review ( <a href="#">Bain 2015</a> )).
<a href="#">Maitland 2014</a>	This was a cross-over trial.
<a href="#">Matarrelli 2013</a>	This randomised controlled trial assessed myo-inositol (which is the focus of another Cochrane review <a href="#">Brown 2015</a> )).
<a href="#">Mike O'Callaghan Federal Hospital 2011</a>	This randomised controlled trial assessed folic acid for prevention of GDM [this trial was withdrawn prior to enrolment]
<a href="#">Min 2014</a>	The women in this randomised controlled trial had type 2 diabetes; docosahexaenoic acid was assessed (which is the focus of another Cochrane review ( <a href="#">Makrides 2006</a> )).
<a href="#">Moses 2009</a>	Women with GDM were the participants in this randomised controlled trial

(Continued)

<a href="#">Phelan 2011</a>	This randomised controlled trial is assessing a combined diet and exercise intervention (which is the focus of another Cochrane review ( <a href="#">Bain 2015</a> )).
<a href="#">Phelan 2016</a>	This randomised controlled trial assessed a combined diet and exercise intervention (which is the focus of another Cochrane review ( <a href="#">Bain 2015</a> )).
<a href="#">Poston 2015</a>	This randomised controlled trial assessed a combined diet and exercise intervention (which is the focus of another Cochrane review ( <a href="#">Bain 2015</a> )).
<a href="#">Reyes-Munoz 2014</a>	This randomised controlled trial assessed metformin for the prevention of GDM
<a href="#">Rhodes 2010</a>	This randomised controlled trial assessed diet interventions in overweight and obese women and did not report on GDM; it is included in the <a href="#">Muktabhant 2015</a> review.
<a href="#">Taghizadeh 2014</a>	This randomised controlled trial assessed a probiotic intervention (which is the focus of another Cochrane review ( <a href="#">Barrett 2014</a> )).
<a href="#">Vesco 2014</a>	This randomised controlled trial assessed a combined diet and exercise intervention (which is the focus of 2 other Cochrane reviews ( <a href="#">Bain 2015</a> ; <a href="#">Muktabhant 2015</a> )).
<a href="#">Yap 2014</a>	This randomised controlled trial assessed vitamin D for the prevention of GDM
<a href="#">Zhou 2011</a>	This is part of a larger randomised controlled trial assessing docosahexaenoic acid (which is the focus of another Cochrane review ( <a href="#">Makrides 2006</a> )).

GDM: gestational diabetes mellitus

## Characteristics of studies awaiting assessment *[ordered by study ID]*

### [Angel 2011](#)

Methods	Randomised controlled trial.
Participants	64 overweight and obese pregnant women aged 18-42 years were recruited from prenatal clinics from Francisco General Hospital, USA hospital between 2006 and 2009
Interventions	Low glycaemic-load diet versus low-fat diet from 20 weeks' gestation to term
Outcomes	Dietary intake (assessed from food diaries); maternal body composition
Notes	Published abstract with limited information regarding methods, intervention and outcomes. Results are not reported by randomisation group Awaiting assessment pending publication of the full study report. Emailed re: full study report 03/02/2016; awaiting reply

### Parat 2015

Methods	Randomised control trial.
Participants	268 pregnant women who were overweight or obese from 4 French centres
Interventions	Intervention: 2 individual and 4 collective dietary counselling sessions at 18, 26, 33 weeks' gestation and 2 months after birth aimed at educating the future mother "for infant and maternal nutritional aspects, without weight objectives;" versus control: routine care including at least 1 dietary visit
Outcomes	Events during pregnancy (GDM; gestational weight gain; birthweight); postnatal catch-up growth; overweight 2 years after birth for mothers and children
Notes	Published abstract with limited information regarding methods, intervention and outcomes. Results for GDM are not reported by randomisation group Awaiting assessment pending publication of the full study report. Emailed re: full study report 03/02/2016; awaiting reply

### Simmons 2015

Methods	Randomised controlled trial.
Participants	146 pregnant women at risk of GDM (BMI $\geq$ 29) from 9 European centres
Interventions	Healthy eating versus physical activity versus healthy eating and physical activity Women received 5 face-to-face, and 4 telephone coaching sessions up to 35 weeks' gestation, based on the principles of motivational interviewing. Each intervention included discussion about the risks of GDM and 7 healthy eating messages and/or 5 physical activity messages. A gestational weight gain target of < 5 kg was emphasised
Outcomes	GDM; fasting glucose; gestational weight gain < 5 kg; "obstetric outcomes".
Notes	Published abstract with limited information regarding methods, intervention and outcomes. Results are not reported by randomisation group Awaiting assessment pending publication of the full study report. Emailed re: full study report 03/02/2016; awaiting reply ISRCTN70595832

### Zhang 2015

Methods	Randomised controlled trial.
Participants	261 "parturient women, who enrolled in regular pregnancy testing".
Interventions	Medical nutrition guidance.
Outcomes	Maternal health and pregnancy outcomes (hypertensive disorders in pregnancy; GDM; caesarean section birth; vaginal birth); newborn health (macrosomia)

Zhang 2015 (Continued)

Notes	Published abstract with limited information regarding methods, intervention and outcomes. Results for GDM are not reported by randomisation group Awaiting assessment pending availability of the full study report. No contact details available
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BMI: body mass index

GDM: gestational diabetes mellitus

**Characteristics of ongoing studies** [ordered by study ID]

**NCT01056406**

Trial name or title	Nutrition intervention for the promotion of healthy weight gain during pregnancy: the reverse pregnancy weight management study
Methods	Randomised controlled trial.
Participants	<b>Setting:</b> Massachusetts General Hospital, USA. <b>Inclusion criteria:</b> pregnant women < 16 weeks' gestation, ages 18-49 with a BMI > 25 and < 40 at the first prenatal visit, both women who have and have not received previous nutritional counselling or who have and have not dieted in the past, women with a documented medical history or who report a history of disordered eating including over eating, night eating, or binge eating without a diagnosis listed in the "exclusion criteria" will not be excluded <b>Exclusion criteria:</b> pregnant women over age 49, women with a BMI < 25 or > 40 at their first prenatal visit, > 16 weeks' gestation, multiple pregnancy, diabetes prior to pregnancy, women with a documented medical history of an eating disorder including anorexia nervosa, bulimia nervosa, anorexia athletica, and orthorexia Recruitment target: 300 women.
Interventions	Twice-monthly interaction with a registered dietitian from 6-16 weeks' gestation through 6 months postpartum for women who are overweight or obese compared with no dietitian intervention
Outcomes	<b>Primary outcomes:</b> percentage of total weight gained during pregnancy compared to current Institute of Medicine Guidelines (weight at first prenatal care appointment compared with weight at last appointment prior to delivery) <b>Secondary outcomes:</b> improvement of the intake of nutritious foods as quantified by a written nutrition and exercise questionnaire (assessed at enrolment, 6 weeks postpartum, 6 months postpartum); prevalence of complications (hypertension and eclampsia, GDM, caesarean delivery, macrosomia, admission to neonatal intensive care unit; initiation and maintenance of breastfeeding (assessed at 6 weeks and 6 months postpartum) , postpartum weight reduction in comparison to pre-pregnancy baseline weight (BMI) (assessed at 6 months postpartum), maintenance of improvements in overall intake of nutritious foods as quantified by the nutrition and exercise questionnaire (assessed at 6 weeks postpartum, and 6 months postpartum), maintenance of improvements in physical activity as quantified by the nutrition and exercise questionnaire (assessed at 6 weeks postpartum, 6 months postpartum)
Starting date	December 2009.

**NCT01056406** (Continued)

Contact information	Alessandra Peccei, MD, Massachusetts General Hospital.
Notes	Estimated study completion date: January 2015. Emailed re: study completion 01/02/2016; awaiting reply.

**NCT01105455**

Trial name or title	The effect of a low-GI diet on blood sugar control in pregnant women at risk for gestational diabetes
Methods	Randomised controlled trial.
Participants	<b>Setting:</b> Toronto, Canada. <b>Inclusion criteria:</b> attending study hospital, with BMI $\geq$ 25 OR age $\geq$ 35 years OR high risk ethnicity (Asian, South Asian, Hispanic, African, Aboriginal) <b>Exclusion criteria:</b> pre-existing diabetes (type 1 or type 2) before becoming pregnant, acute or chronic illness which may affect carbohydrate metabolism, language/literacy barriers which cannot be overcome via available resources, > 16 weeks' gestation Recruitment target: 103 enrolled.
Interventions	Group nutrition classes supplemented by handouts and provision of key study foods compared with advice leaflet re high-fibre diet
Outcomes	<b>Primary outcome:</b> serum glucose concentration 1hr after OGCT (assessed at 26 weeks' gestation) <b>Secondary outcomes:</b> GDM, maternal weight (assessed at 12, 16, 20, 24, 28, 32, 36 and 40 weeks' gestation), mode of delivery, birth trauma, infant birthweight, macrosomia, large-for-gestational-age baby, small-for-gestational-age baby, maternal food frequency questionnaire (at 12 weeks and 26 weeks' gestation) and acceptability of study foods (assessed at 26 weeks' gestation)
Starting date	March 2010.
Contact information	Professor Thomas Wolever, University of Toronto, <a href="mailto:thomas.wolever@utoronto.ca">thomas.wolever@utoronto.ca</a>
Notes	Study completion date: December 2011. Emailed re: study completion 01/02/2016. Reply received 02/02/2016; trial has been completed; aiming for publication later in 2016

**NCT01628835**

Trial name or title	Low-GI diet management for pregnant women with overweight.
Methods	Randomised controlled trial.
Participants	<b>Setting:</b> Shanghai, China. <b>Inclusion criteria:</b> women with first prenatal examination BMI $\geq$ 24, primiparous, singleton pregnancy, aged 18 years to 45 years, first prenatal examination equal to or < 12 weeks, willing and able to give informed consent <b>Exclusion criteria:</b> assisted conception, history of hypertension, diabetes, coronary heart disease or mental

**NCT01628835** (Continued)

	disorder, special diet habit (e.g. vegetarianism/veganism) Target recruitment: 400 women.
Interventions	4 diet consultations at baseline (first prenatal examination), the end of the 1st trimester, the 2nd trimester and the 3rd trimester respectively, including diet assessment and diet consultation specifically recommending a low-GI diet versus routine diet advice for pregnant women
Outcomes	<b>Primary outcomes:</b> maternal insulin measured at first prenatal visit and last visit before delivery, cord blood C-peptide at delivery, GDM, macrosomia <b>Secondary outcomes:</b> gestational hypertension (defined as systolic and diastolic blood pressure $\geq$ 140/90 mmHg), birthweight, caesarean delivery, gestational age at birth, preterm birth (gestational age < 37 weeks at birth), maternal gestational weight gain (from baseline measurement to delivery)
Starting date	June 2012.
Contact information	Professor Weili Yan, Director, Children's Hospital of Fudan University: <a href="mailto:yanwl@fudan.edu.cn">yanwl@fudan.edu.cn</a>
Notes	Estimated study completion date: February 2015. Emailed re: study completion 01/02/2016

**NCT01894139**

Trial name or title	An optimised programming of healthy children (APPROACH).
Methods	Randomised controlled trial.
Participants	<b>Setting:</b> The Nutrition Research Unit at Copenhagen University Hospital, Denmark <b>Inclusion criteria:</b> obese pregnant women (BMI $\geq$ 30) age 18-42 years, with singleton pregnancy recruited during late first or early second trimester <b>Exclusion criteria:</b> multiple pregnancy, history of spontaneous abortions, GDM, pre-eclampsia or spontaneous preterm birth, dairy product intolerant or allergic, > 5 kg weight loss during the past year, abuse of alcohol or drugs (> 14 units of alcohol per week), critical or chronic disease: diabetes, kidney disease, medically treated heart diseases or arthritis, sarcoidosis, tuberculosis, cancer, liver disease, inflammatory gastrointestinal or lung disease, known active metabolic disease Recruitment target: 390 pregnant women.
Interventions	A dietary and advice intervention involving a high-protein diet, especially marine and dairy protein and low-GI diet versus a diet according to the Nordic Nutritional Recommendations. The intervention aims to increase knowledge of the effect of a high-protein to carbohydrate-ratio diet and weight retention during pregnancy
Outcomes	<b>Primary outcomes:</b> gestational weight gain (weeks 14, 15, 17, 21, 25, 28, 36, 39) Changes in bodyweight, body composition and measurements of body fat by means of skinfold thickness and mid-upper arm circumference <b>Other outcomes:</b> growth and development of fetus and child (weeks 11 + 2, 14 + 0, 28, 32, 36 and month 0, 6, 18, 36 and year 5, 9); fetus: Nuchal Translucency Scan (11 + 3 - 14 + 0) and ultrasound scan (28, 32, 36); child: height, weight, body composition (Bioimpedance (month 6,18) and Dual-energy X-ray absorptiometry, DXA (month 0, 36, year 5, 9)), skin fold thickness and mid-upper arm circumference, IGF-1; fetal programming of obesity and metabolic disorders (month 0, 6,18, 36 and year 5, 9); fasting blood samples (month 0 from umbilical cord); dietary intake, physical activity, growth and development

**NCT01894139** (Continued)

Starting date	November 2013.
Contact information	Nina RW Geiker, Post Doc, <a href="mailto:nina.rica.wium.geiker@regionh.dk">nina.rica.wium.geiker@regionh.dk</a> Annette Vedelspang, Dietician RD <a href="mailto:annette.vedelspang@regionh.dk">annette.vedelspang@regionh.dk</a> Principal investigator: Professor Arne V Astrup, Copenhagen University Hospital, Denmark
Notes	Estimated study completion date: December 2025.

**NCT02218931**

Trial name or title	ESTEEM - Effect of simple, targeted diet in pregnant women with metabolic risk factors on pre-eclampsia
Methods	Randomised controlled trial.
Participants	<b>Setting:</b> Queen Mary University of London, UK. <b>Inclusion criteria:</b> pregnant women < 18 weeks of gestation with at least 1 of the following: BMI $\geq$ 30; raised serum triglycerides $\geq$ 1.7 mmol/L; raised blood pressure of systole $\geq$ 140 mmHg or diastole $\geq$ 90 mmHg <b>Exclusion criteria:</b> BMI < 18.5 or $\geq$ 40, women on lipid altering drugs, history of diabetes, chronic renal disease, auto-immune disease, multiple pregnancy, poor understanding of written and spoken English, not able to follow Mediterranean diet for religious or other reasons, < 16 years of age, not able to consume nuts or extra virgin olive oil Recruitment target: 3640 women.
Interventions	Targeted advice intervention based on Mediterranean dietary pattern (high intake of vegetables, nuts, non-refined grains, legumes and fruits, moderate to high consumption of fish, small to moderate intake of poultry and dairy products such as yoghurt and cheese, low consumption of red meat and processed meat and avoidance of sugary drinks, fast food and high fat food, high fibre; high intake of nuts including walnuts and almonds that are rich sources of monounsaturated and polyunsaturated fatty acids (30 g/day), olive oil to cook and dress salads as the main source of fat (0.5 L/week)). The intervention will include structured meal plans and grocery lists, recipes for healthy diet and appropriate choices at restaurants compared with usual antenatal dietary advice
Outcomes	<b>Primary outcomes:</b> diagnosis of pre-eclampsia (defined as: new onset hypertension after 20 weeks' gestation defined as systolic BP $\geq$ 140 mmHg or diastolic BP $\geq$ 90 mmHg, in at least 2 readings AND new onset proteinuria defined as spot urine protein/creatinine ratio test > 30 mg/mmol or > 24 hour urine 300 mg/24 hours or 2+ or more on standard urinary dipstick tests after 20 weeks' gestation, superimposed pre-eclampsia in women with chronic hypertension or chronic proteinuria or women with eclamptic seizures with no hypertension or proteinuria) <b>Secondary outcomes:</b> adverse maternal outcomes, adverse fetal outcomes, dietary outcomes (assessed at baseline and 36 weeks or delivery depending on which is sooner), nutrient and food intakes derived from Food Frequency Questionnaire and ESTEEM questionnaires, measure of laboratory outcomes (assessed at 36 weeks of gestation or at delivery whichever is the earliest) (concentrations of triglycerides, high density lipoproteins, ratio of triglycerides and non-high density lipoprotein cholesterol)
Starting date	July 2014.



**NCT02218931** (Continued)

Contact information	Shakila Thangaratinam, Queen Mary University of London, <a href="mailto:s.thangaratinam@qmul.ac.uk">s.thangaratinam@qmul.ac.uk</a> Julie Dodds, <a href="mailto:j.dodds@qmul.ac.uk">j.dodds@qmul.ac.uk</a>
Notes	Estimated study completion date: December 2016.

BMI: body mass index

GDM: gestational diabetes mellitus

GI: glycaemic index

OGCT: oral glucose challenge test

## DATA AND ANALYSES

### Comparison 1. Dietary advice interventions versus standard care

Outcome or subgroup title	No. of studies	No. of participants	Statistical method	Effect size
1 Gestational diabetes	5	1279	Risk Ratio (M-H, Random, 95% CI)	0.60 [0.35, 1.04]
1.1 All women	2	870	Risk Ratio (M-H, Random, 95% CI)	0.93 [0.64, 1.36]
1.2 Overweight or obese women (BMI > 25)	3	409	Risk Ratio (M-H, Random, 95% CI)	0.39 [0.19, 0.79]
2 Hypertensive disorders of pregnancy (pregnancy-induced hypertension)	2	282	Risk Ratio (M-H, Fixed, 95% CI)	0.30 [0.10, 0.88]
3 Hypertensive disorders of pregnancy (pre-eclampsia)	2	282	Risk Ratio (M-H, Fixed, 95% CI)	0.61 [0.25, 1.46]
4 Perinatal mortality	1	159	Risk Ratio (M-H, Fixed, 95% CI)	0.0 [0.0, 0.0]
5 Caesarean section	4	1194	Risk Ratio (M-H, Random, 95% CI)	0.98 [0.78, 1.24]
6 Induction of labour	2	991	Risk Ratio (M-H, Random, 95% CI)	1.10 [0.48, 2.51]
7 Perineal trauma (anal sphincter injury)	1	759	Risk Ratio (M-H, Fixed, 95% CI)	0.83 [0.23, 3.08]
8 Postpartum haemorrhage	2	991	Risk Ratio (M-H, Fixed, 95% CI)	0.71 [0.28, 1.86]
9 Postpartum infection	1	232	Risk Ratio (M-H, Fixed, 95% CI)	0.83 [0.26, 2.65]
10 Breastfeeding (at 3 months)	1	452	Risk Ratio (M-H, Fixed, 95% CI)	1.02 [0.89, 1.17]
11 Breastfeeding (at 6 months)	1	146	Risk Ratio (M-H, Fixed, 95% CI)	0.99 [0.82, 1.19]
12 Gestational weight gain (kg)	5	1336	Mean Difference (IV, Random, 95% CI)	-4.70 [-8.07, -1.34]
13 Gestational weight gain (g/week)			Other data	No numeric data
14 Behaviour changes associated with the intervention: health behaviours at 3 months postpartum	1		Risk Ratio (M-H, Fixed, 95% CI)	Subtotals only
14.1 Weight reducing diet at 3 months postpartum	1	458	Risk Ratio (M-H, Fixed, 95% CI)	0.95 [0.66, 1.38]
14.2 Supplements at 3 months postpartum	1	459	Risk Ratio (M-H, Fixed, 95% CI)	1.12 [0.98, 1.28]
14.3 Made dietary changes since ROLO study	1	420	Risk Ratio (M-H, Fixed, 95% CI)	1.12 [0.98, 1.29]
14.4 Low GI diet at 3 months postpartum	1	197	Risk Ratio (M-H, Fixed, 95% CI)	5.37 [1.93, 14.89]
14.5 Reading food labels at 3 months postpartum	1	453	Risk Ratio (M-H, Fixed, 95% CI)	1.11 [1.01, 1.23]
14.6 Reading ingredients at 3 months postpartum	1	453	Risk Ratio (M-H, Fixed, 95% CI)	1.12 [0.91, 1.37]
14.7 Reading nutrients at 3 months postpartum	1	453	Risk Ratio (M-H, Fixed, 95% CI)	1.21 [1.03, 1.41]
14.8 Reading calories at 3 months postpartum	1	453	Risk Ratio (M-H, Fixed, 95% CI)	1.08 [0.89, 1.31]

14.9 Reading food weight at 3 months postpartum	1	453	Risk Ratio (M-H, Fixed, 95% CI)	1.51 [0.90, 2.54]
14.10 Reading additives at 3 months postpartum	1	453	Risk Ratio (M-H, Fixed, 95% CI)	1.33 [0.99, 1.79]
14.11 Reading serving size at 3 months postpartum	1	453	Risk Ratio (M-H, Fixed, 95% CI)	1.39 [0.83, 2.34]
14.12 Attending gym at 3 months postpartum	1	440	Risk Ratio (M-H, Fixed, 95% CI)	0.98 [0.65, 1.49]
15 Fasting glucose at 28 weeks (mmol/L)	1	759	Mean Difference (IV, Fixed, 95% CI)	-0.06 [-0.13, 0.01]
16 Fasting glucose at 28 weeks $\geq$ 5.1 mmol/L	1	673	Risk Ratio (M-H, Fixed, 95% CI)	0.64 [0.40, 1.04]
17 OGCT at 28 weeks (mmol/L)	1	759	Mean Difference (IV, Fixed, 95% CI)	-0.20 [-0.42, 0.02]
18 OGCT at 28 weeks > 7.8 mmol/L	1	721	Risk Ratio (M-H, Fixed, 95% CI)	0.72 [0.53, 0.99]
19 Fasting glucose at 28 weeks $\geq$ 5.1 mmol/L or OGCT at 28 weeks $\geq$ 7.8 mmol/L	1	672	Risk Ratio (M-H, Fixed, 95% CI)	0.74 [0.56, 0.97]
20 Sense of well-being: score (% score between 14 to 28 weeks)	1	618	Mean Difference (IV, Fixed, 95% CI)	-3.60 [-5.98, -1.22]
21 Postpartum weight loss at 6 weeks (kg)	1	232	Mean Difference (IV, Fixed, 95% CI)	-0.90 [-3.67, 1.87]
22 Postnatal weight retention: change in weight from late pregnancy to 3 months postpartum (kg)	1	165	Mean Difference (IV, Fixed, 95% CI)	-0.35 [-1.84, 1.14]
23 Return to pre-pregnancy weight: change in weight from baseline to 3 months postpartum (kg)	1	414	Mean Difference (IV, Fixed, 95% CI)	-1.43 [-2.66, -0.20]
24 Postpartum BMI			Other data	No numeric data
25 Stillbirth	2	959	Risk Ratio (M-H, Fixed, 95% CI)	3.09 [0.13, 75.65]
26 Neonatal mortality	1	159	Risk Ratio (M-H, Fixed, 95% CI)	0.0 [0.0, 0.0]
27 Preterm birth (less than 37 weeks' gestation)	3	1149	Risk Ratio (M-H, Fixed, 95% CI)	0.51 [0.21, 1.25]
28 Preterm birth (less than 32 weeks' gestation)	2	917	Risk Ratio (M-H, Fixed, 95% CI)	1.70 [0.23, 12.88]
29 Apgar score less than 7 at 5 minutes	1	232	Risk Ratio (M-H, Fixed, 95% CI)	3.0 [0.12, 72.89]
30 Macrosomia (> 4000 g)	1	759	Risk Ratio (M-H, Fixed, 95% CI)	0.99 [0.86, 1.14]
31 Macrosomia (> 4500 g)	1	232	Risk Ratio (M-H, Fixed, 95% CI)	2.25 [0.71, 7.10]
32 Shoulder dystocia	1	759	Risk Ratio (M-H, Fixed, 95% CI)	0.52 [0.10, 2.82]
33 Gestational age at birth (weeks)	4	1195	Mean Difference (IV, Random, 95% CI)	0.05 [-0.31, 0.40]
34 Birthweight (g)	5	1324	Mean Difference (IV, Fixed, 95% CI)	5.94 [-51.11, 62.99]
35 Head circumference at birth (cm)	3	968	Mean Difference (IV, Random, 95% CI)	-0.21 [-0.67, 0.25]
36 Length at birth (cm)	3	968	Mean Difference (IV, Random, 95% CI)	0.16 [-0.28, 0.60]
37 Ponderal index at birth	1	759	Mean Difference (IV, Fixed, 95% CI)	0.01 [-0.38, 0.40]
38 Adiposity at birth: skin-fold thickness (mm)	1		Mean Difference (IV, Fixed, 95% CI)	Subtotals only

38.1 Subscapular skin-fold (mm)	1	219	Mean Difference (IV, Fixed, 95% CI)	-0.04 [-0.45, 0.37]
38.2 Triceps skin-fold (mm)	1	219	Mean Difference (IV, Fixed, 95% CI)	-0.18 [-0.58, 0.22]
38.3 Biceps skin-fold (mm)	1	219	Mean Difference (IV, Fixed, 95% CI)	-0.10 [-0.50, 0.30]
38.4 Leg skin-fold (mm)	1	219	Mean Difference (IV, Fixed, 95% CI)	0.06 [-0.42, 0.54]
38.5 Sum of skin-folds (mm)	1	219	Mean Difference (IV, Fixed, 95% CI)	-1.60 [-4.77, 1.57]
39 Weight at 3 months (kg)	1	422	Mean Difference (IV, Fixed, 95% CI)	0.23 [-0.37, 0.83]
40 Weight at 6 months (kg)	1	143	Mean Difference (IV, Fixed, 95% CI)	-0.03 [-0.35, 0.29]
41 Length at 6 months (cm)	1	143	Mean Difference (IV, Fixed, 95% CI)	0.0 [-1.06, 1.06]
42 Head circumference at 6 months (cm)	1	132	Mean Difference (IV, Fixed, 95% CI)	-0.20 [-0.61, 0.21]
43 Skinfold thickness at 6 months (mm)	1	132	Mean Difference (IV, Fixed, 95% CI)	-0.10 [-0.71, 0.51]
44 Systolic blood pressure at 6 months (mmHg)	1	113	Mean Difference (IV, Fixed, 95% CI)	-1.0 [-4.53, 2.53]
45 Diastolic blood pressure at 6 months (mmHg)	1	113	Mean Difference (IV, Fixed, 95% CI)	-1.0 [-3.77, 1.77]
46 Mean blood pressure at 6 months (mmHg)	1	113	Mean Difference (IV, Fixed, 95% CI)	-1.0 [-3.77, 1.77]
47 Heart rate at 6 months (bpm)	1	113	Mean Difference (IV, Fixed, 95% CI)	2.0 [-2.89, 6.89]
48 Clinical complications (gestational diabetes, pre-eclampsia, low birthweight, prematurity)	1	305	Risk Ratio (M-H, Fixed, 95% CI)	0.37 [0.21, 0.66]

## Comparison 2. Low-GI dietary advice versus moderate- to high-GI dietary advice

Outcome or subgroup title	No. of studies	No. of participants	Statistical method	Effect size
1 Gestational diabetes	4	912	Risk Ratio (M-H, Fixed, 95% CI)	0.91 [0.63, 1.31]
2 Large-for-gestational age	3	777	Risk Ratio (M-H, Random, 95% CI)	0.60 [0.19, 1.86]
3 Caesarean birth	2	201	Risk Ratio (M-H, Fixed, 95% CI)	1.27 [0.79, 2.04]
4 Operative vaginal birth	1	62	Risk Ratio (M-H, Fixed, 95% CI)	1.25 [0.49, 3.18]
5 Gestational weight gain (kg)	4	787	Mean Difference (IV, Random, 95% CI)	-1.23 [-4.08, 1.61]
6 Adherence to the intervention	2	636	Mean Difference (IV, Fixed, 95% CI)	0.03 [-0.07, 0.13]
6.1 I adhered well to the dietary instructions	2	636	Mean Difference (IV, Fixed, 95% CI)	0.03 [-0.07, 0.13]
7 Fasting glucose at 24-28 weeks (mmol/L)	1	20	Mean Difference (IV, Fixed, 95% CI)	-0.17 [-0.57, 0.23]
8 Fasting glucose at 32-36 weeks (mmol/L)	2	82	Mean Difference (IV, Fixed, 95% CI)	-0.27 [-0.52, -0.03]
9 Views of the intervention	2		Mean Difference (IV, Random, 95% CI)	Subtotals only
9.1 It was easy to follow the diet recommended during this study	2	636	Mean Difference (IV, Random, 95% CI)	-0.09 [-0.45, 0.27]
9.2 I enjoyed the dietary changes that I made	2	636	Mean Difference (IV, Random, 95% CI)	-0.09 [-0.22, 0.03]

9.3 The changes recommended were affordable	2	636	Mean Difference (IV, Random, 95% CI)	0.04 [-0.08, 0.16]
9.4 My family was accepting of the changes made to my eating habits	2	636	Mean Difference (IV, Random, 95% CI)	-0.02 [-0.15, 0.10]
9.5 The study diet helped me meet the physical challenges of pregnancy	2	636	Mean Difference (IV, Random, 95% CI)	0.10 [-0.03, 0.22]
9.6 I enjoyed a wide variety of foods in my eating plan	2	636	Mean Difference (IV, Random, 95% CI)	-0.05 [-0.15, 0.05]
10 Apgar score less than 7 at 5 minutes	1	62	Risk Ratio (M-H, Fixed, 95% CI)	2.82 [0.12, 66.62]
11 Macrosomia (> 4000 g)	2	715	Risk Ratio (M-H, Fixed, 95% CI)	0.73 [0.49, 1.09]
12 Macrosomia (> 4500 g)	1	576	Risk Ratio (M-H, Fixed, 95% CI)	0.32 [0.06, 1.55]
13 Small-for-gestational age	3	777	Risk Ratio (M-H, Fixed, 95% CI)	0.88 [0.53, 1.45]
14 Gestational age at birth (weeks)	3	777	Mean Difference (IV, Fixed, 95% CI)	0.11 [-0.11, 0.33]
15 Birthweight (g)	4	797	Mean Difference (IV, Random, 95% CI)	-217.97 [-483.96, 48.02]
16 Birthweight (z score)	1	139	Mean Difference (IV, Fixed, 95% CI)	0.07 [-0.26, 0.40]
17 Head circumference at birth (cm)	2	82	Mean Difference (IV, Random, 95% CI)	-1.20 [-2.75, 0.36]
18 Length at birth (cm)	3	658	Mean Difference (IV, Random, 95% CI)	-0.77 [-1.98, 0.45]
19 Ponderal index at birth	4	797	Mean Difference (IV, Random, 95% CI)	-0.06 [-0.16, 0.04]
20 Adiposity at birth: % body fat	2	108	Mean Difference (IV, Fixed, 95% CI)	0.02 [-1.43, 1.47]
21 Neonatal intensive care unit admission	1	138	Risk Ratio (M-H, Fixed, 95% CI)	0.37 [0.12, 1.11]

### Comparison 3. High-fibre dietary advice versus 'standard' dietary advice

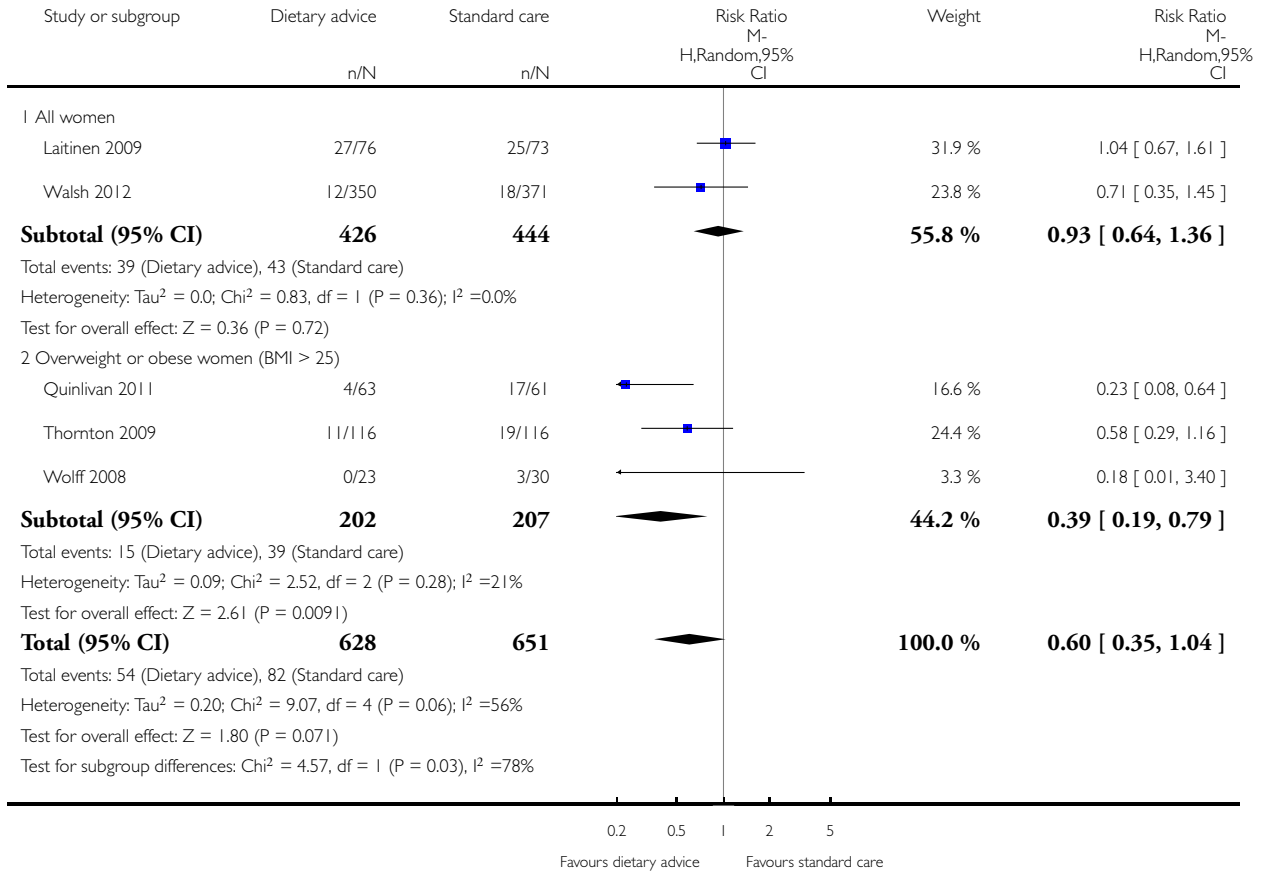
Outcome or subgroup title	No. of studies	No. of participants	Statistical method	Effect size
1 OGTT at 35 weeks (mmol/L)	1	25	Mean Difference (IV, Fixed, 95% CI)	-0.36 [-0.90, 0.18]
2 Birthweight centile	1	25	Mean Difference (IV, Fixed, 95% CI)	-0.30 [-5.40, 4.80]

### Analysis 1.1. Comparison 1 Dietary advice interventions versus standard care, Outcome 1 Gestational diabetes.

Review: Dietary advice interventions in pregnancy for preventing gestational diabetes mellitus

Comparison: 1 Dietary advice interventions versus standard care

Outcome: 1 Gestational diabetes

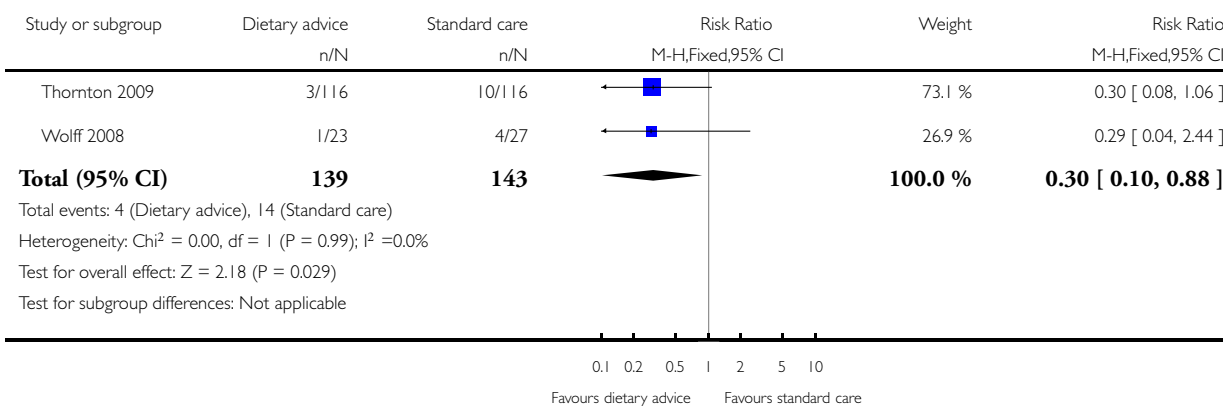


### Analysis 1.2. Comparison 1 Dietary advice interventions versus standard care, Outcome 2 Hypertensive disorders of pregnancy (pregnancy-induced hypertension).

Review: Dietary advice interventions in pregnancy for preventing gestational diabetes mellitus

Comparison: 1 Dietary advice interventions versus standard care

Outcome: 2 Hypertensive disorders of pregnancy (pregnancy-induced hypertension)

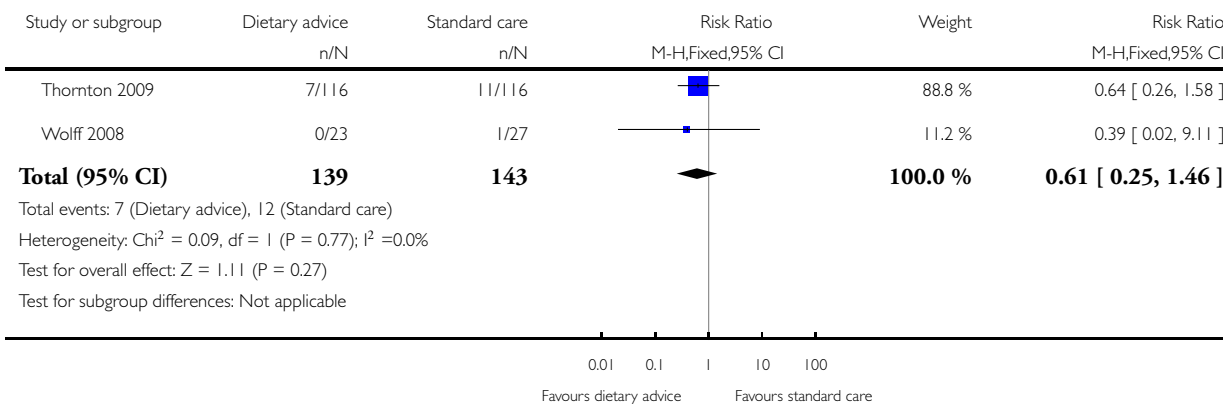


### Analysis 1.3. Comparison 1 Dietary advice interventions versus standard care, Outcome 3 Hypertensive disorders of pregnancy (pre-eclampsia).

Review: Dietary advice interventions in pregnancy for preventing gestational diabetes mellitus

Comparison: 1 Dietary advice interventions versus standard care

Outcome: 3 Hypertensive disorders of pregnancy (pre-eclampsia)

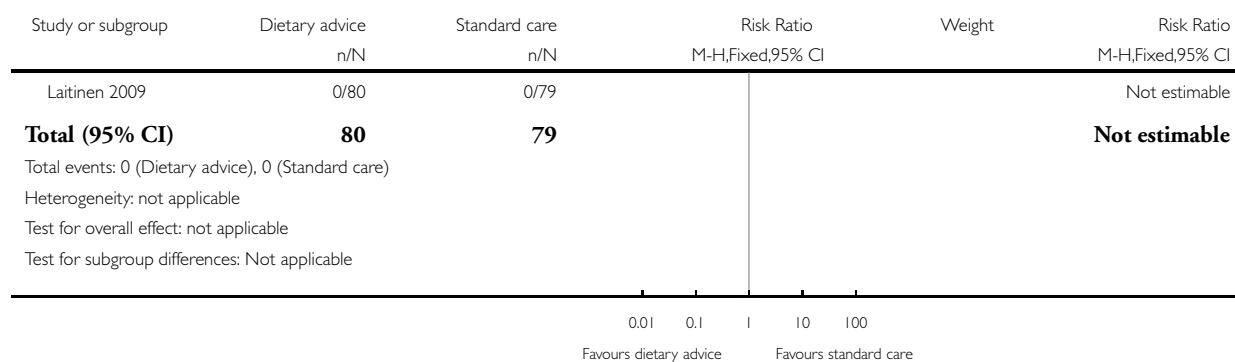


### Analysis 1.4. Comparison 1 Dietary advice interventions versus standard care, Outcome 4 Perinatal mortality.

Review: Dietary advice interventions in pregnancy for preventing gestational diabetes mellitus

Comparison: 1 Dietary advice interventions versus standard care

Outcome: 4 Perinatal mortality



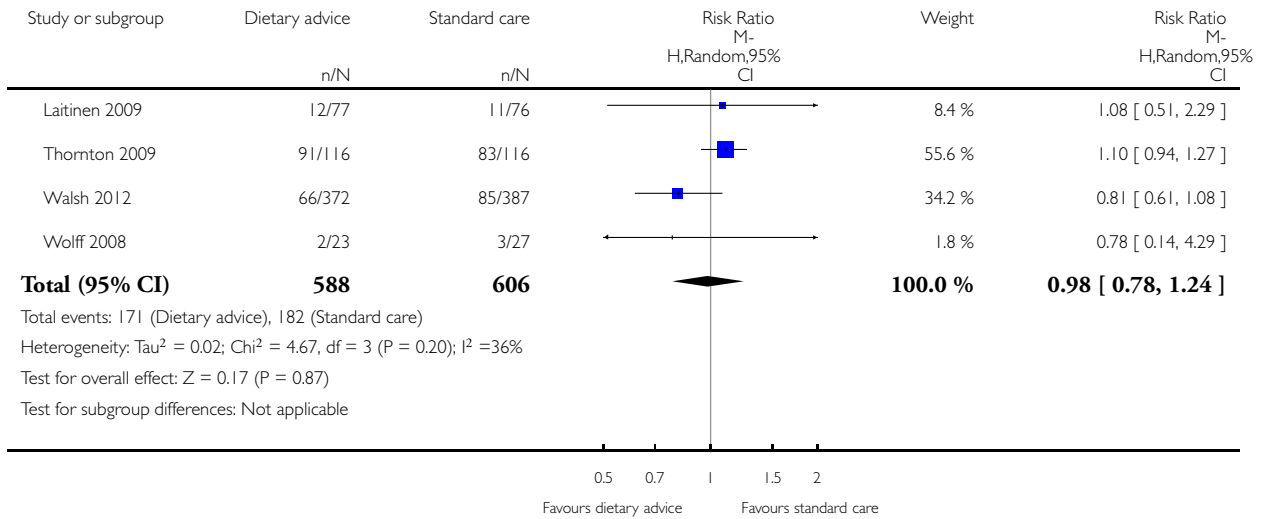


### Analysis 1.5. Comparison 1 Dietary advice interventions versus standard care, Outcome 5 Caesarean section.

Review: Dietary advice interventions in pregnancy for preventing gestational diabetes mellitus

Comparison: 1 Dietary advice interventions versus standard care

Outcome: 5 Caesarean section

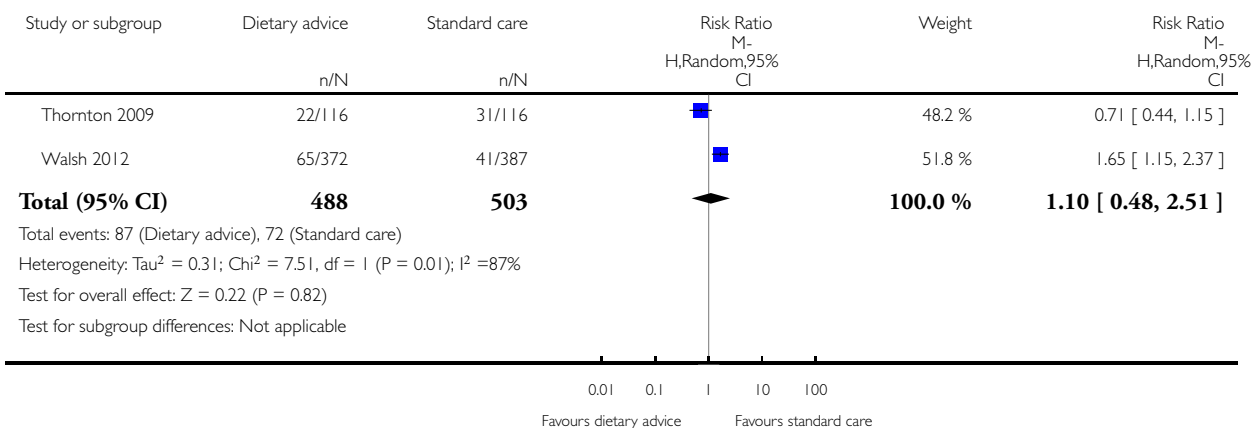


### Analysis 1.6. Comparison 1 Dietary advice interventions versus standard care, Outcome 6 Induction of labour.

Review: Dietary advice interventions in pregnancy for preventing gestational diabetes mellitus

Comparison: 1 Dietary advice interventions versus standard care

Outcome: 6 Induction of labour

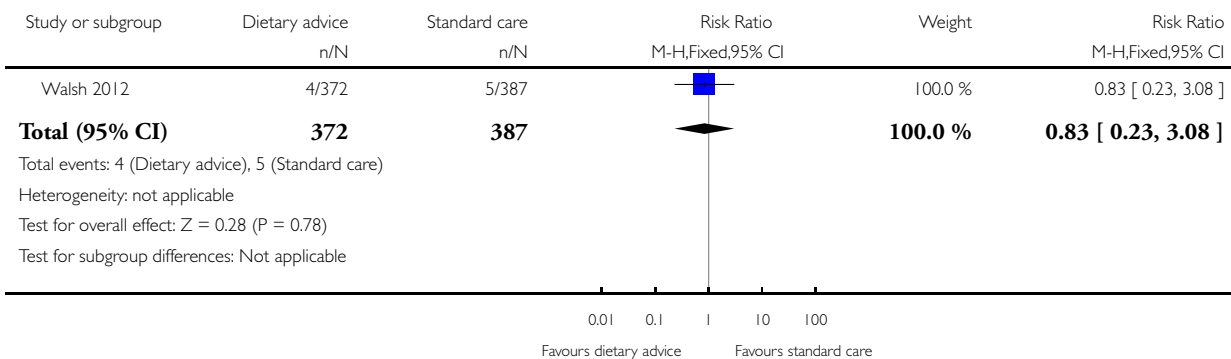


### Analysis 1.7. Comparison 1 Dietary advice interventions versus standard care, Outcome 7 Perineal trauma (anal sphincter injury).

Review: Dietary advice interventions in pregnancy for preventing gestational diabetes mellitus

Comparison: 1 Dietary advice interventions versus standard care

Outcome: 7 Perineal trauma (anal sphincter injury)

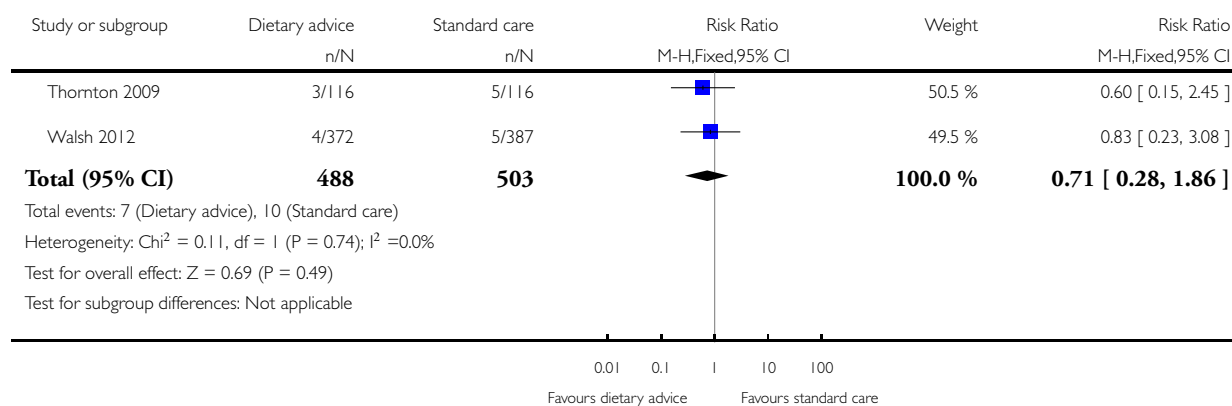


### Analysis 1.8. Comparison 1 Dietary advice interventions versus standard care, Outcome 8 Postpartum haemorrhage.

Review: Dietary advice interventions in pregnancy for preventing gestational diabetes mellitus

Comparison: 1 Dietary advice interventions versus standard care

Outcome: 8 Postpartum haemorrhage

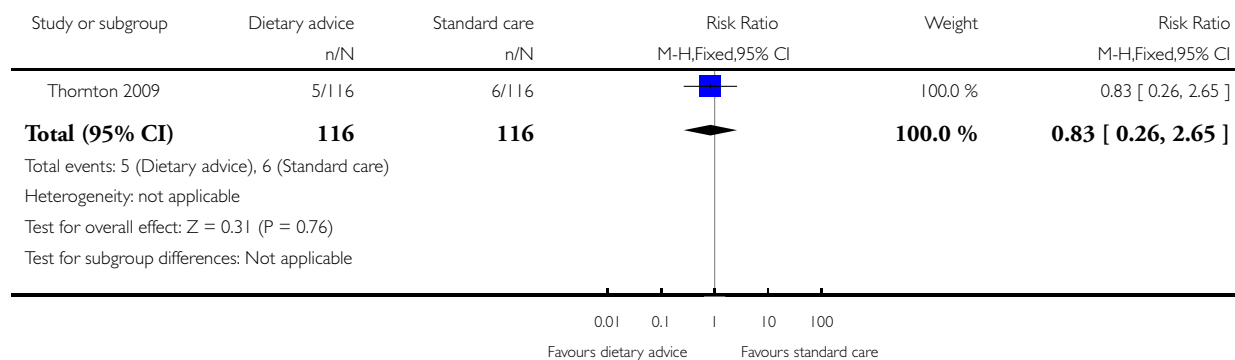


### Analysis 1.9. Comparison 1 Dietary advice interventions versus standard care, Outcome 9 Postpartum infection.

Review: Dietary advice interventions in pregnancy for preventing gestational diabetes mellitus

Comparison: 1 Dietary advice interventions versus standard care

Outcome: 9 Postpartum infection

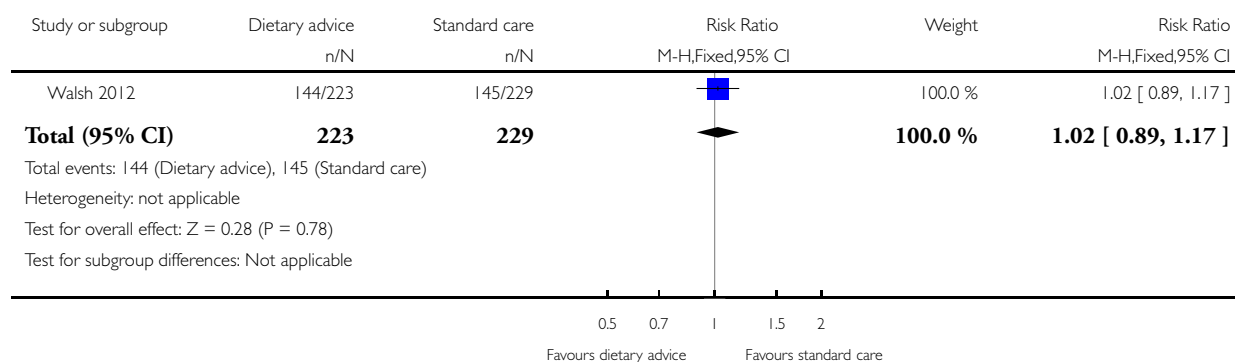


### Analysis 1.10. Comparison 1 Dietary advice interventions versus standard care, Outcome 10 Breastfeeding (at 3 months).

Review: Dietary advice interventions in pregnancy for preventing gestational diabetes mellitus

Comparison: 1 Dietary advice interventions versus standard care

Outcome: 10 Breastfeeding (at 3 months)

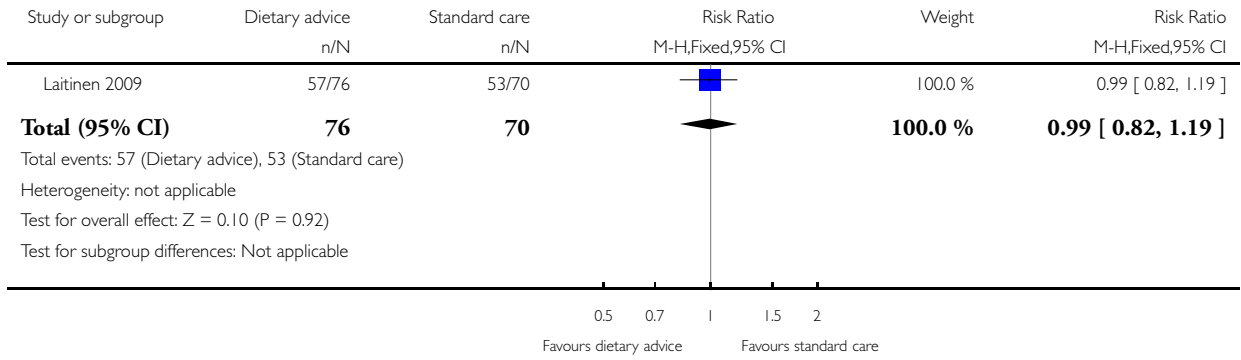


**Analysis 1.11. Comparison 1 Dietary advice interventions versus standard care, Outcome 11 Breastfeeding (at 6 months).**

Review: Dietary advice interventions in pregnancy for preventing gestational diabetes mellitus

Comparison: 1 Dietary advice interventions versus standard care

Outcome: 11 Breastfeeding (at 6 months)

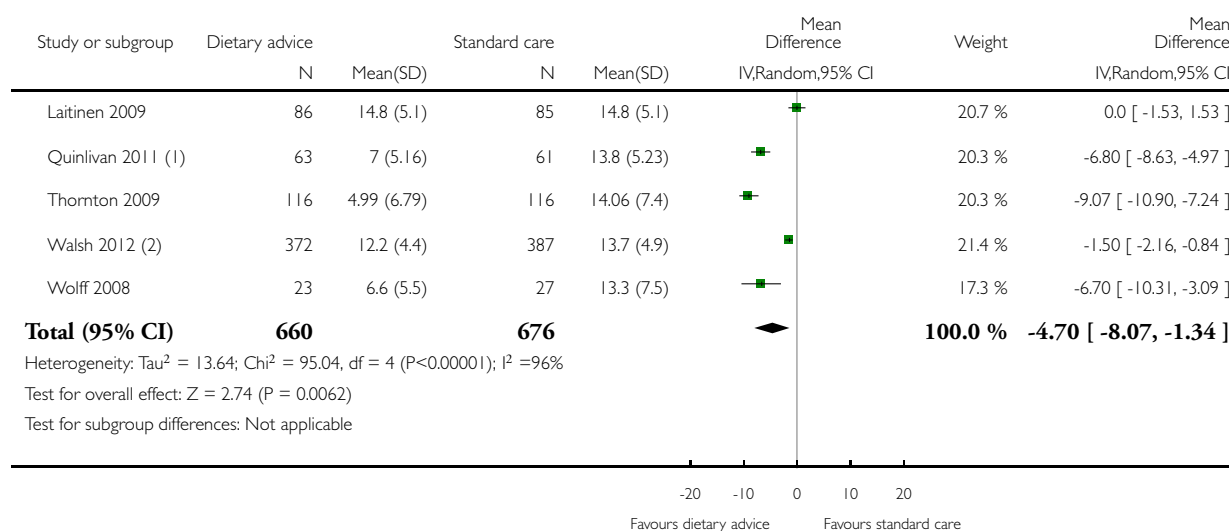


### Analysis 1.12. Comparison 1 Dietary advice interventions versus standard care, Outcome 12 Gestational weight gain (kg).

Review: Dietary advice interventions in pregnancy for preventing gestational diabetes mellitus

Comparison: 1 Dietary advice interventions versus standard care

Outcome: 12 Gestational weight gain (kg)



(1) SDs calculated based on SEs provided

(2) At 40 weeks' gestation

### Analysis 1.13. Comparison 1 Dietary advice interventions versus standard care, Outcome 13 Gestational weight gain (g/week).

Gestational weight gain (g/week)

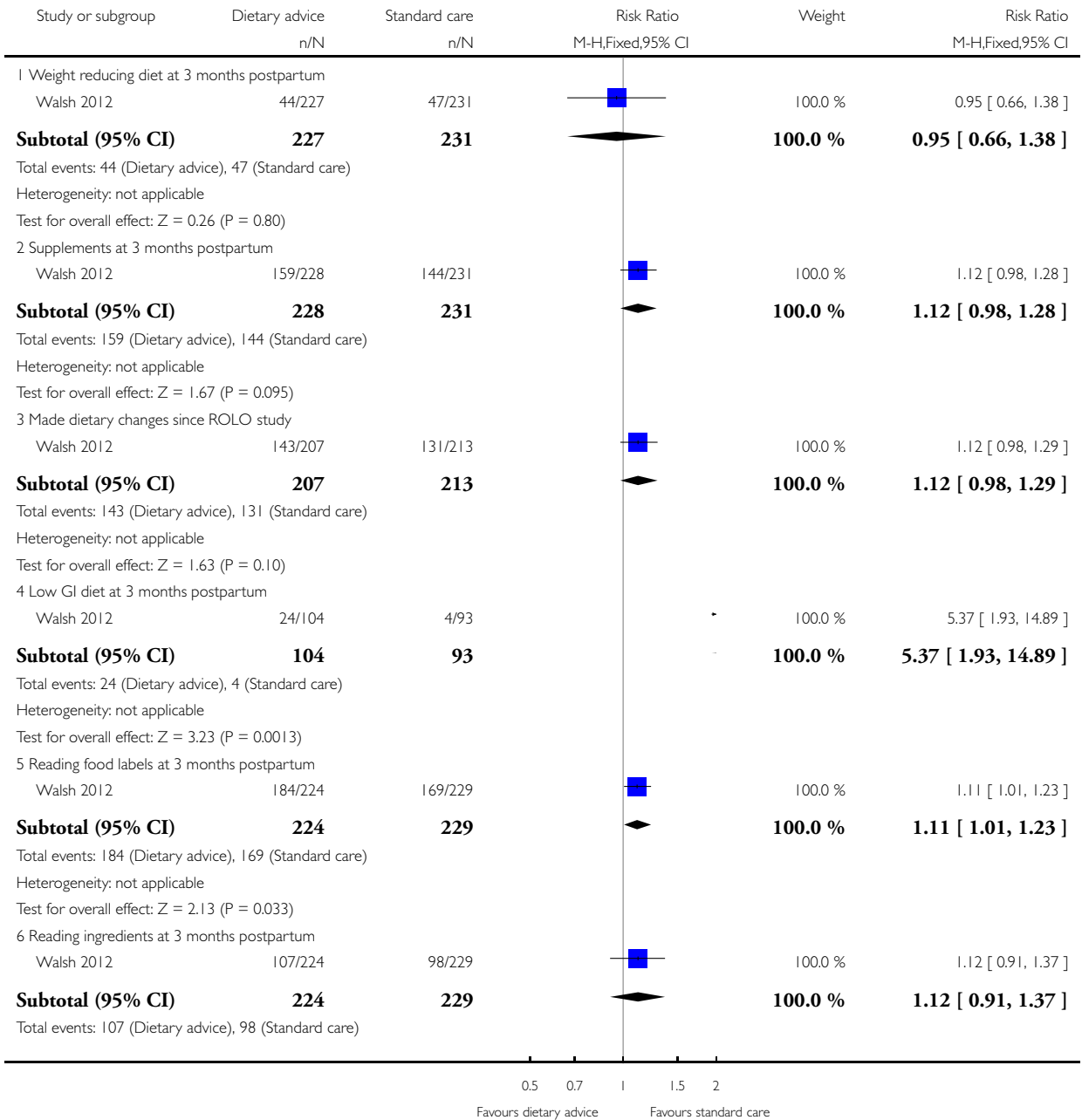
Study	Dietary advice intervention	Standard care	P value
Vitolo 2011	Weekly average weight gain Low weight: Mean: 507.8, standard deviation: 496.1; N=unclear Eutrophic: Mean: 460.1, standard deviation: 135.2; N=unclear Excess weight: Mean: 342.2, standard deviation: 143.6; N=unclear	Weekly average weight gain Low weight: Mean: 496.1, standard deviation: 177.0; N=unclear Eutrophic: Mean: 492.2, standard deviation: 222.1; N=unclear Excess weight: Mean: 143.6, standard deviation: 185.4; N=unclear	Low weight: 0.8 Eutrophic: 0.2 Excess weight: 0.01

**Analysis 1.14. Comparison 1 Dietary advice interventions versus standard care, Outcome 14 Behaviour changes associated with the intervention: health behaviours at 3 months postpartum.**

Review: Dietary advice interventions in pregnancy for preventing gestational diabetes mellitus

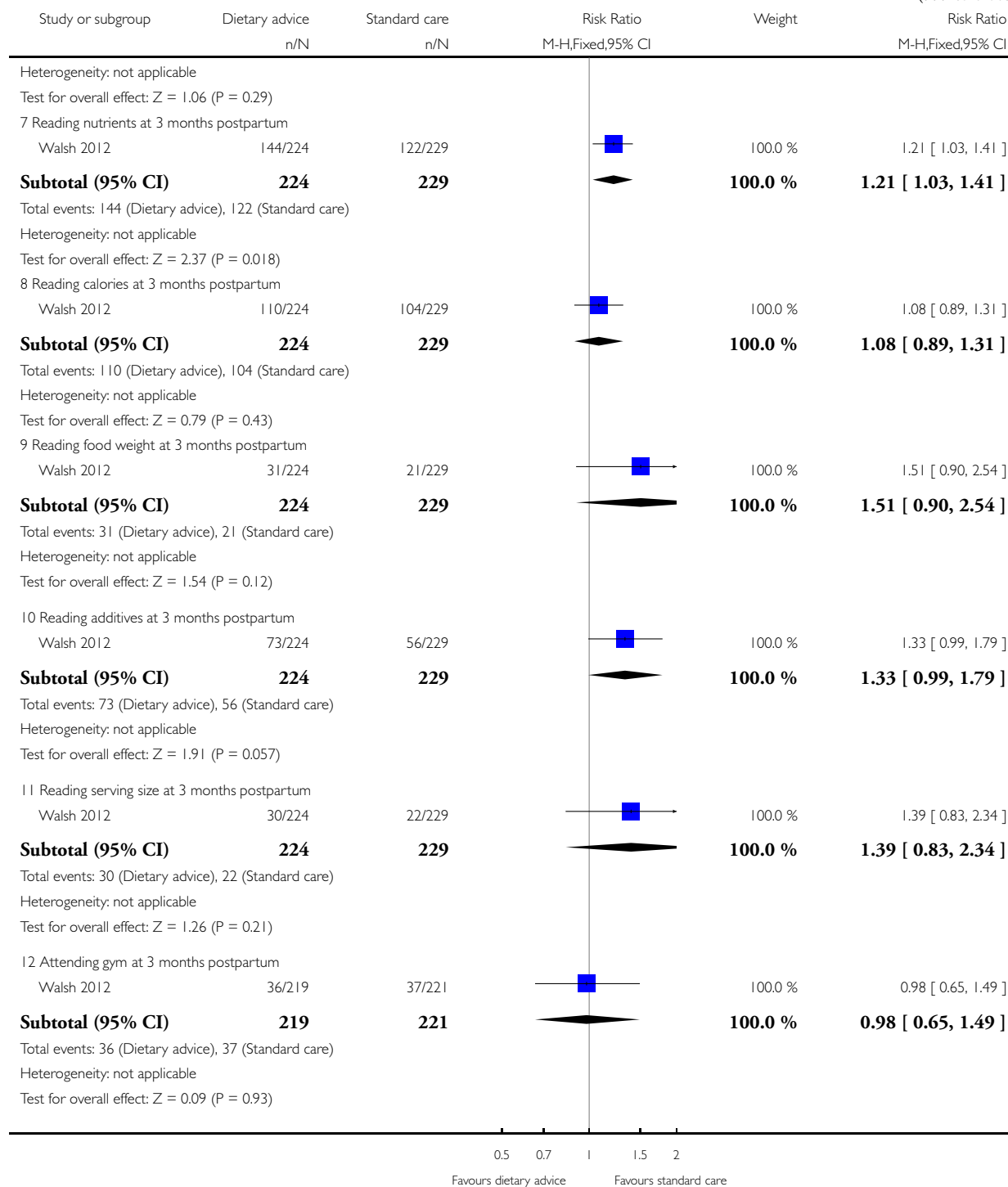
Comparison: 1 Dietary advice interventions versus standard care

Outcome: 14 Behaviour changes associated with the intervention: health behaviours at 3 months postpartum



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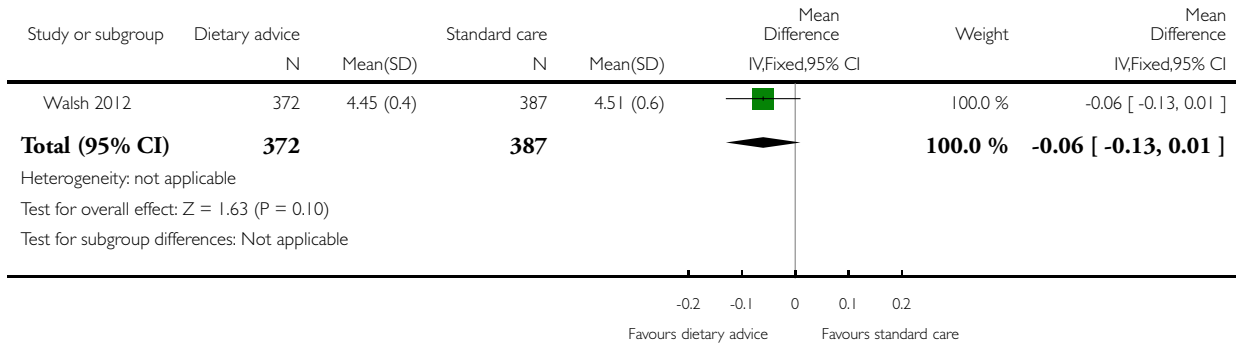


**Analysis I.15. Comparison I Dietary advice interventions versus standard care, Outcome 15 Fasting glucose at 28 weeks (mmol/L).**

Review: Dietary advice interventions in pregnancy for preventing gestational diabetes mellitus

Comparison: I Dietary advice interventions versus standard care

Outcome: 15 Fasting glucose at 28 weeks (mmol/L)

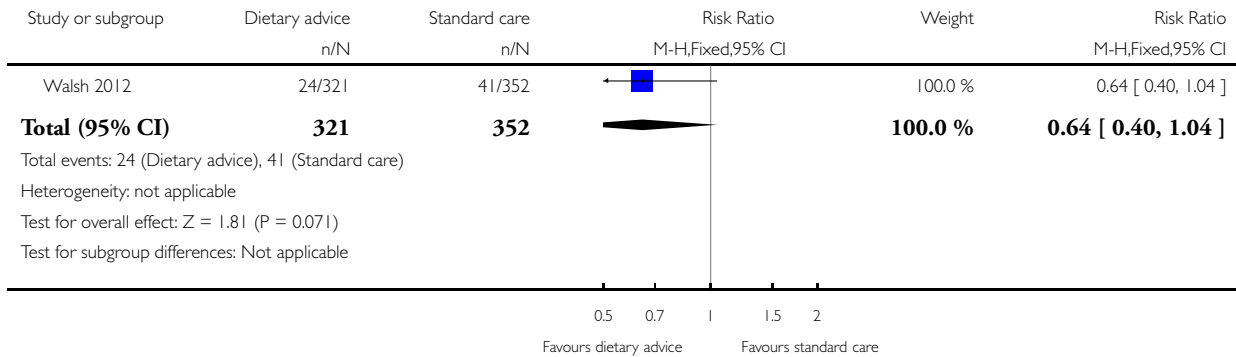


**Analysis I.16. Comparison I Dietary advice interventions versus standard care, Outcome 16 Fasting glucose at 28 weeks  $\geq$  5.1 mmol/L.**

Review: Dietary advice interventions in pregnancy for preventing gestational diabetes mellitus

Comparison: I Dietary advice interventions versus standard care

Outcome: 16 Fasting glucose at 28 weeks  $\geq$  5.1 mmol/L

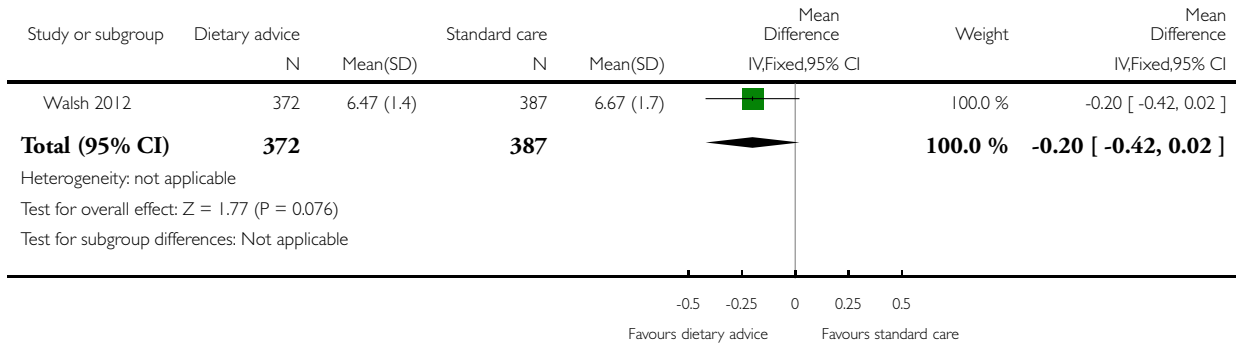


**Analysis I.17. Comparison I Dietary advice interventions versus standard care, Outcome 17 OGCT at 28 weeks (mmol/L).**

Review: Dietary advice interventions in pregnancy for preventing gestational diabetes mellitus

Comparison: I Dietary advice interventions versus standard care

Outcome: 17 OGCT at 28 weeks (mmol/L)

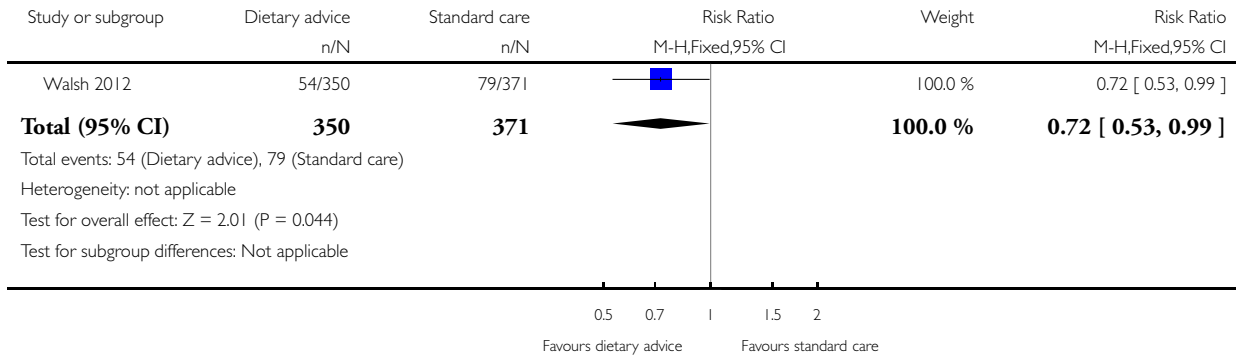


**Analysis I.18. Comparison I Dietary advice interventions versus standard care, Outcome 18 OGCT at 28 weeks > 7.8 mmol/L.**

Review: Dietary advice interventions in pregnancy for preventing gestational diabetes mellitus

Comparison: I Dietary advice interventions versus standard care

Outcome: 18 OGCT at 28 weeks > 7.8 mmol/L

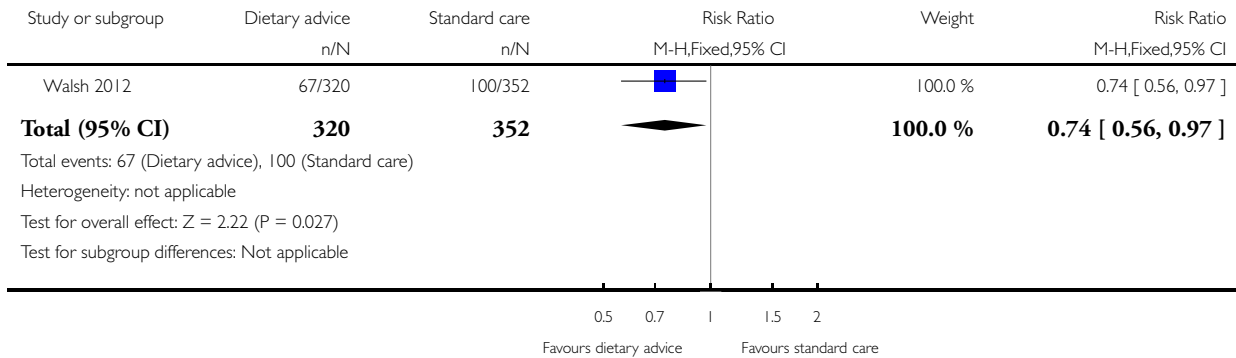


**Analysis 1.19. Comparison 1 Dietary advice interventions versus standard care, Outcome 19 Fasting glucose at 28 weeks  $\geq$  5.1 mmol/L or OGCT at 28 week  $s >$  7.8 mmol/L.**

Review: Dietary advice interventions in pregnancy for preventing gestational diabetes mellitus

Comparison: 1 Dietary advice interventions versus standard care

Outcome: 19 Fasting glucose at 28 weeks  $\geq$  5.1 mmol/L or OGCT at 28 week  $s >$  7.8 mmol/L

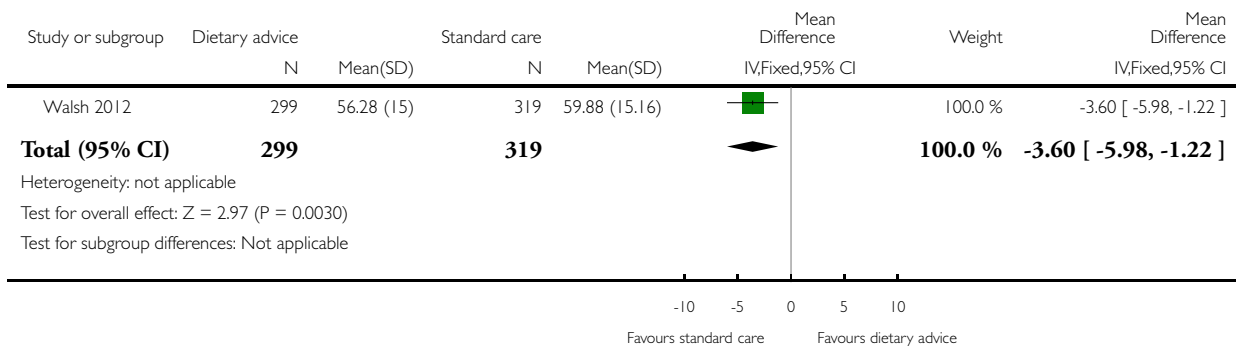


**Analysis 1.20. Comparison 1 Dietary advice interventions versus standard care, Outcome 20 Sense of well-being: score (% score between 14 to 28 weeks).**

Review: Dietary advice interventions in pregnancy for preventing gestational diabetes mellitus

Comparison: 1 Dietary advice interventions versus standard care

Outcome: 20 Sense of well-being: score (% score between 14 to 28 weeks)

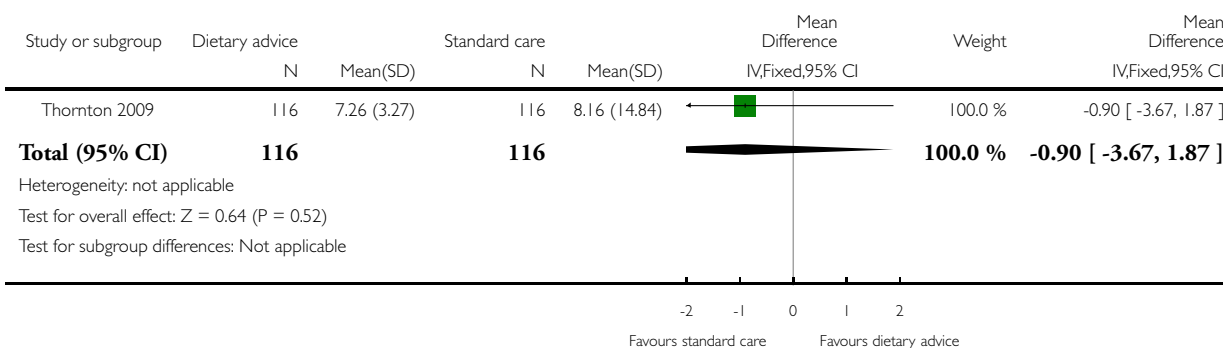


### Analysis 1.21. Comparison 1 Dietary advice interventions versus standard care, Outcome 21 Postpartum weight loss at 6 weeks (kg).

Review: Dietary advice interventions in pregnancy for preventing gestational diabetes mellitus

Comparison: 1 Dietary advice interventions versus standard care

Outcome: 21 Postpartum weight loss at 6 weeks (kg)

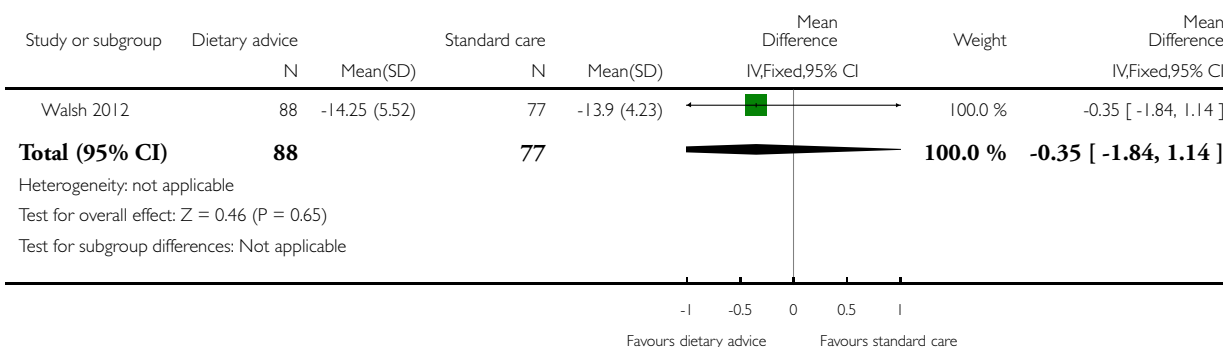


### Analysis 1.22. Comparison 1 Dietary advice interventions versus standard care, Outcome 22 Postnatal weight retention: change in weight from late pregnancy to 3 months postpartum (kg).

Review: Dietary advice interventions in pregnancy for preventing gestational diabetes mellitus

Comparison: 1 Dietary advice interventions versus standard care

Outcome: 22 Postnatal weight retention: change in weight from late pregnancy to 3 months postpartum (kg)

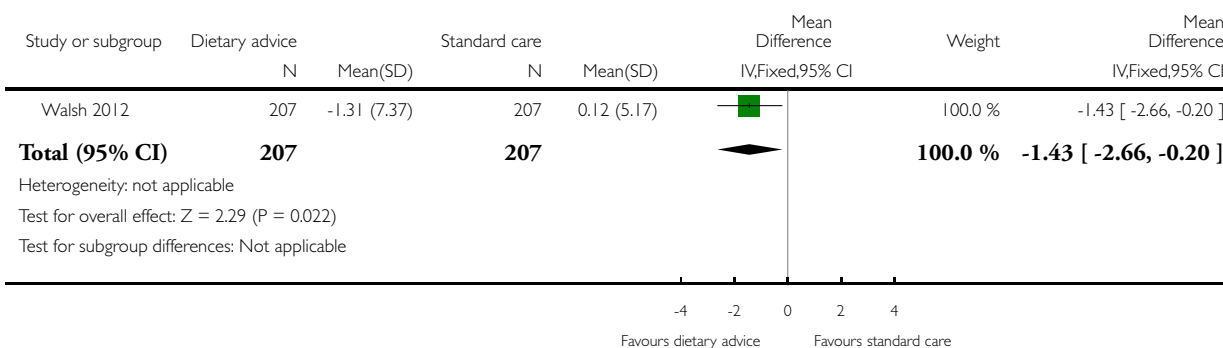


**Analysis 1.23. Comparison 1 Dietary advice interventions versus standard care, Outcome 23 Return to pre-pregnancy weight: change in weight from baseline to 3 months postpartum (kg).**

Review: Dietary advice interventions in pregnancy for preventing gestational diabetes mellitus

Comparison: 1 Dietary advice interventions versus standard care

Outcome: 23 Return to pre-pregnancy weight: change in weight from baseline to 3 months postpartum (kg)



**Analysis 1.24. Comparison 1 Dietary advice interventions versus standard care, Outcome 24 Postpartum BMI.**

**Postpartum BMI**

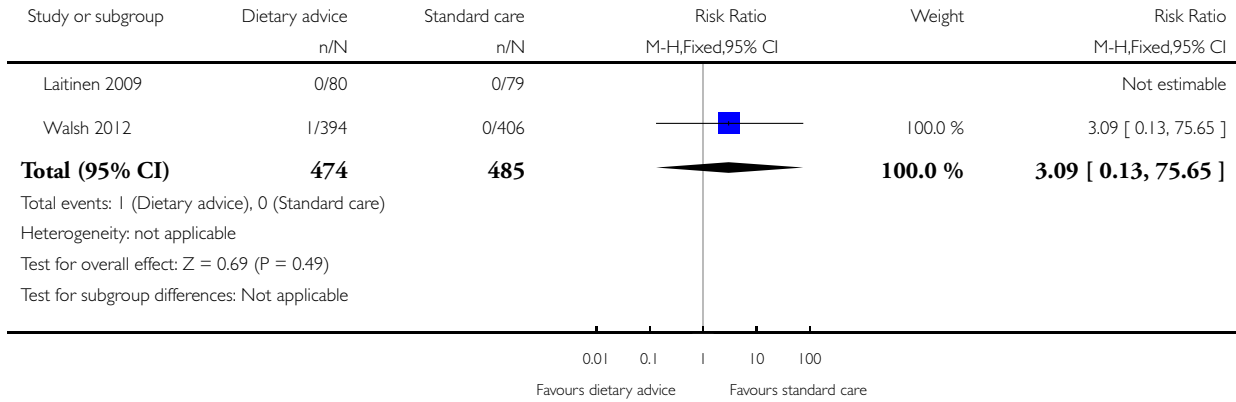
Study	Dietary advice intervention	Standard care
Laitinen 2009	Mean: 25.9; range: 19.5-35.8; N=85 (Vahamiko 2013)	Mean: 25.4; range: 18.6-35.9; N=84 (Vahamiko 2013)

### Analysis 1.25. Comparison 1 Dietary advice interventions versus standard care, Outcome 25 Stillbirth.

Review: Dietary advice interventions in pregnancy for preventing gestational diabetes mellitus

Comparison: 1 Dietary advice interventions versus standard care

Outcome: 25 Stillbirth

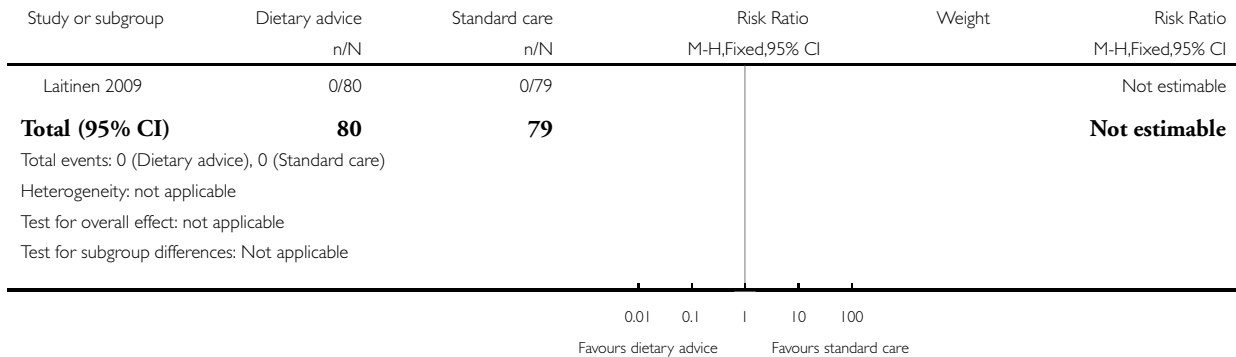


### Analysis 1.26. Comparison 1 Dietary advice interventions versus standard care, Outcome 26 Neonatal mortality.

Review: Dietary advice interventions in pregnancy for preventing gestational diabetes mellitus

Comparison: 1 Dietary advice interventions versus standard care

Outcome: 26 Neonatal mortality

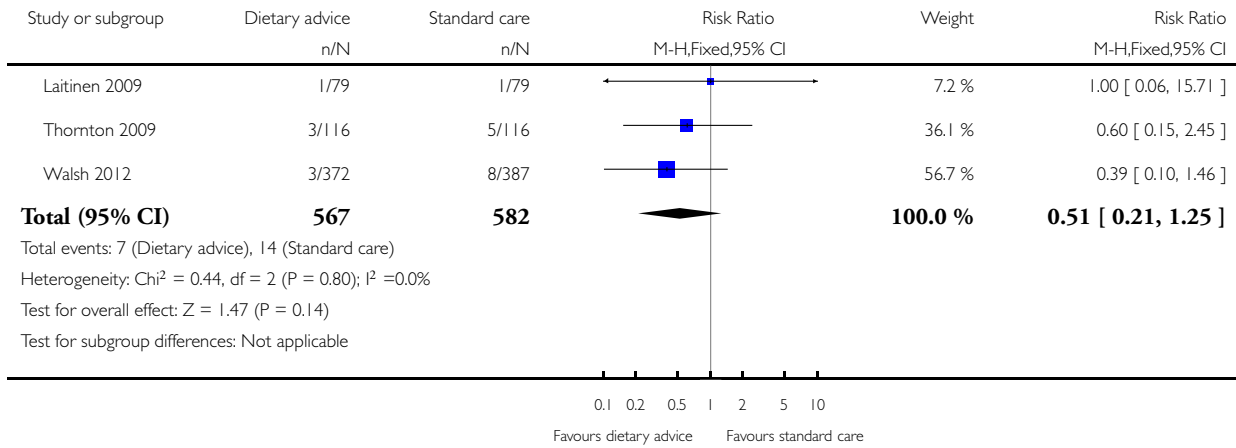


**Analysis 1.27. Comparison I Dietary advice interventions versus standard care, Outcome 27 Preterm birth (less than 37 weeks' gestation).**

Review: Dietary advice interventions in pregnancy for preventing gestational diabetes mellitus

Comparison: I Dietary advice interventions versus standard care

Outcome: 27 Preterm birth (less than 37 weeks' gestation)

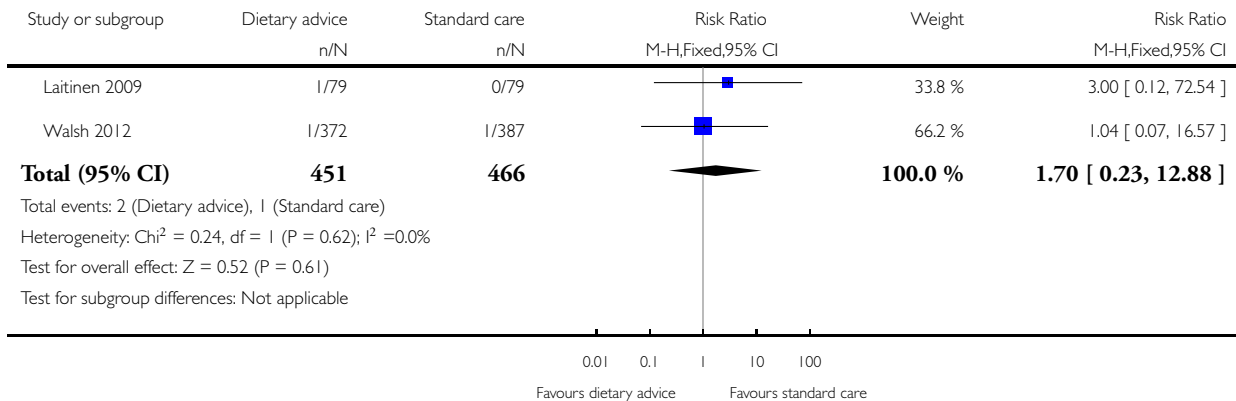


**Analysis 1.28. Comparison I Dietary advice interventions versus standard care, Outcome 28 Preterm birth (less than 32 weeks' gestation).**

Review: Dietary advice interventions in pregnancy for preventing gestational diabetes mellitus

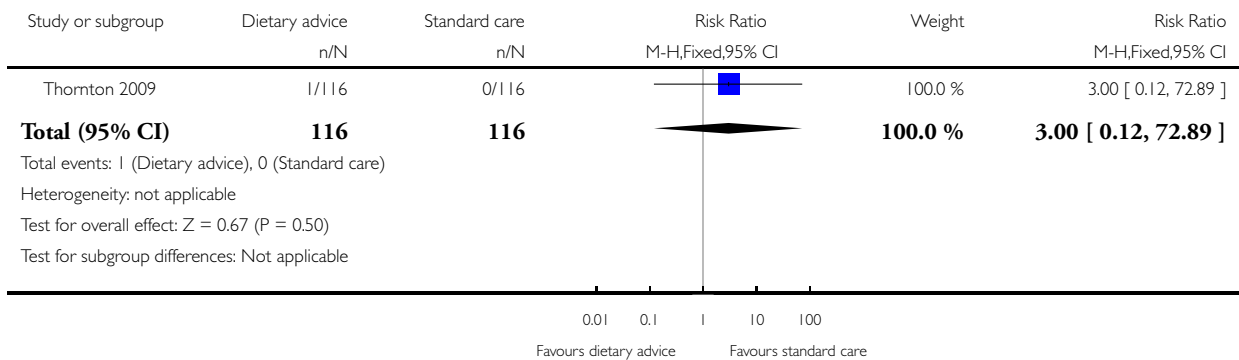
Comparison: I Dietary advice interventions versus standard care

Outcome: 28 Preterm birth (less than 32 weeks' gestation)



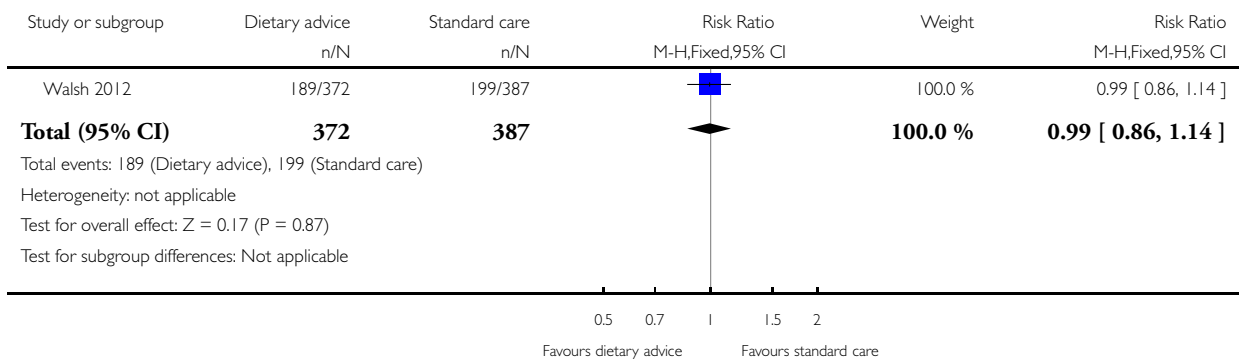
**Analysis 1.29. Comparison 1 Dietary advice interventions versus standard care, Outcome 29 Apgar score less than 7 at 5 minutes.**

Review: Dietary advice interventions in pregnancy for preventing gestational diabetes mellitus  
 Comparison: 1 Dietary advice interventions versus standard care  
 Outcome: 29 Apgar score less than 7 at 5 minutes



**Analysis 1.30. Comparison 1 Dietary advice interventions versus standard care, Outcome 30 Macrosomia (> 4000 g).**

Review: Dietary advice interventions in pregnancy for preventing gestational diabetes mellitus  
 Comparison: 1 Dietary advice interventions versus standard care  
 Outcome: 30 Macrosomia (> 4000 g)



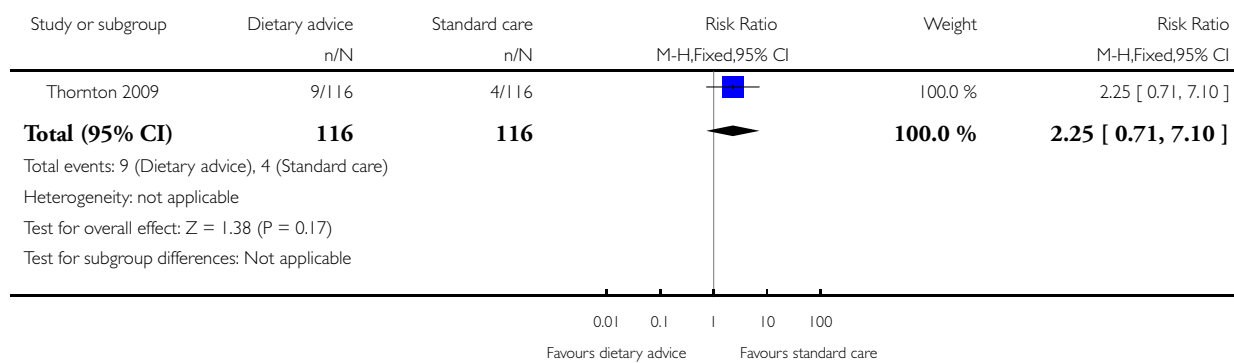


**Analysis 1.31. Comparison 1 Dietary advice interventions versus standard care, Outcome 31 Macrosomia (> 4500 g).**

Review: Dietary advice interventions in pregnancy for preventing gestational diabetes mellitus

Comparison: 1 Dietary advice interventions versus standard care

Outcome: 31 Macrosomia (> 4500 g)

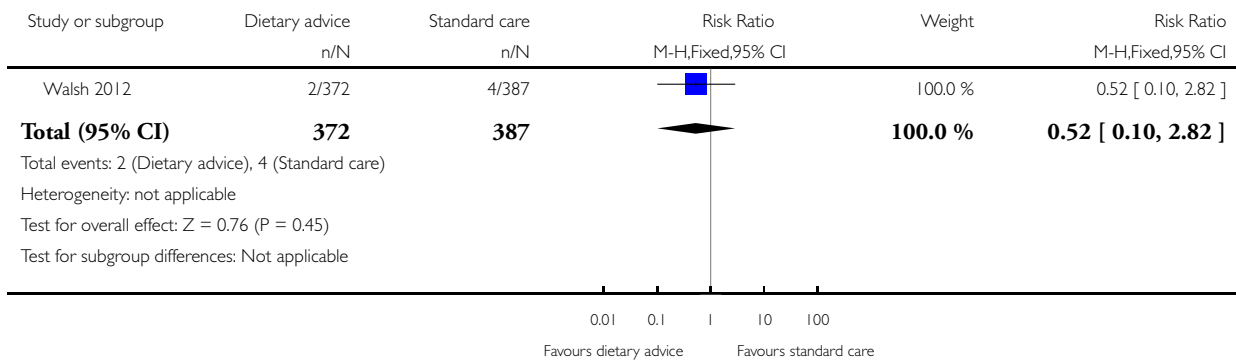


### Analysis 1.32. Comparison 1 Dietary advice interventions versus standard care, Outcome 32 Shoulder dystocia.

Review: Dietary advice interventions in pregnancy for preventing gestational diabetes mellitus

Comparison: 1 Dietary advice interventions versus standard care

Outcome: 32 Shoulder dystocia

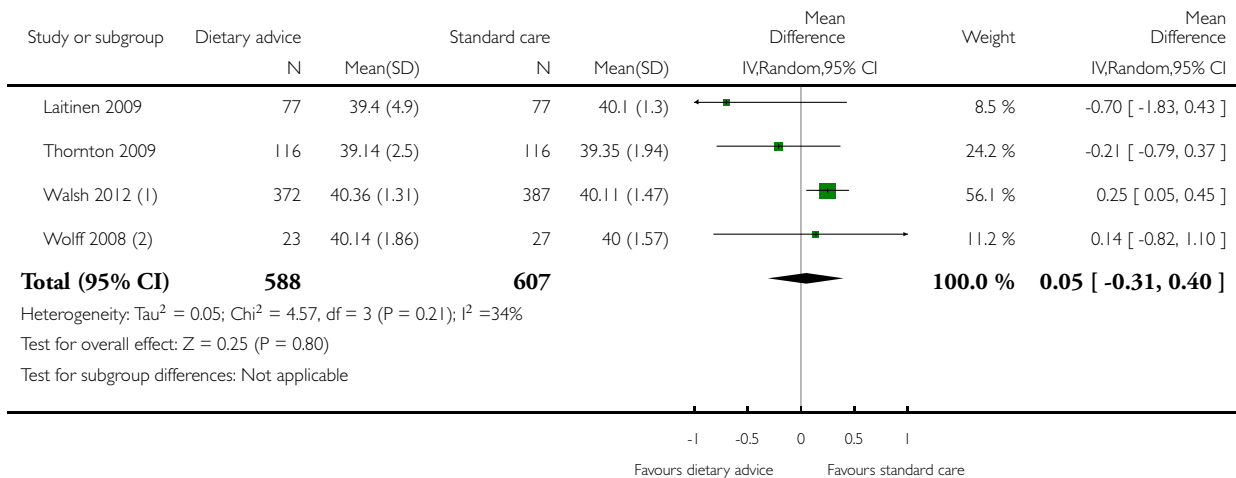


### Analysis 1.33. Comparison 1 Dietary advice interventions versus standard care, Outcome 33 Gestational age at birth (weeks).

Review: Dietary advice interventions in pregnancy for preventing gestational diabetes mellitus

Comparison: 1 Dietary advice interventions versus standard care

Outcome: 33 Gestational age at birth (weeks)



(1) Days were converted to weeks

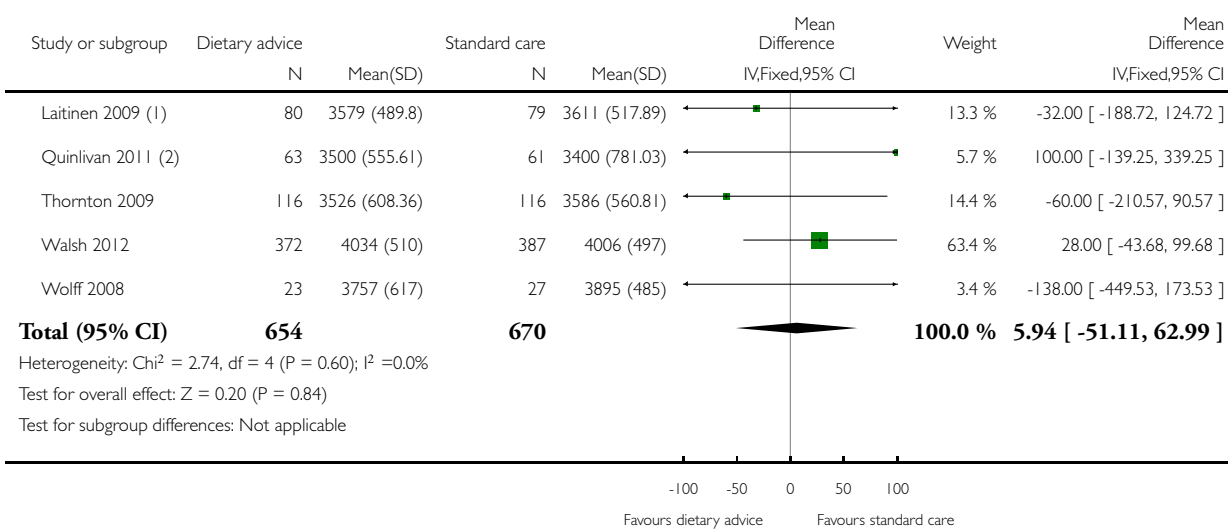
(2) Days were converted to weeks

### Analysis 1.34. Comparison 1 Dietary advice interventions versus standard care, Outcome 34 Birthweight (g).

Review: Dietary advice interventions in pregnancy for preventing gestational diabetes mellitus

Comparison: 1 Dietary advice interventions versus standard care

Outcome: 34 Birthweight (g)



(1) SDs calculated based on 95% CIs provided

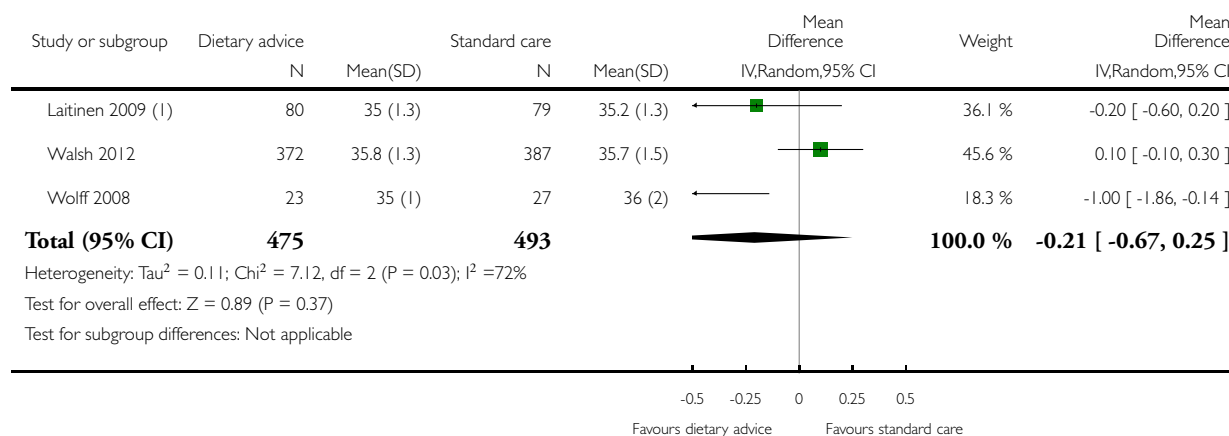
(2) SDs calculated based on SEs provided

### Analysis 1.35. Comparison 1 Dietary advice interventions versus standard care, Outcome 35 Head circumference at birth (cm).

Review: Dietary advice interventions in pregnancy for preventing gestational diabetes mellitus

Comparison: 1 Dietary advice interventions versus standard care

Outcome: 35 Head circumference at birth (cm)



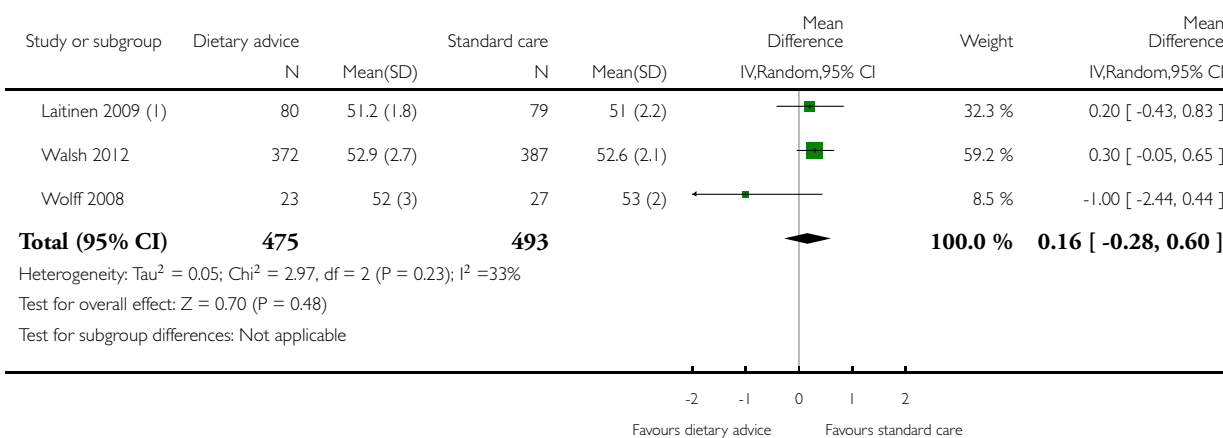
(1) SDs calculated based on 95% CIs provided

### Analysis 1.36. Comparison 1 Dietary advice interventions versus standard care, Outcome 36 Length at birth (cm).

Review: Dietary advice interventions in pregnancy for preventing gestational diabetes mellitus

Comparison: 1 Dietary advice interventions versus standard care

Outcome: 36 Length at birth (cm)



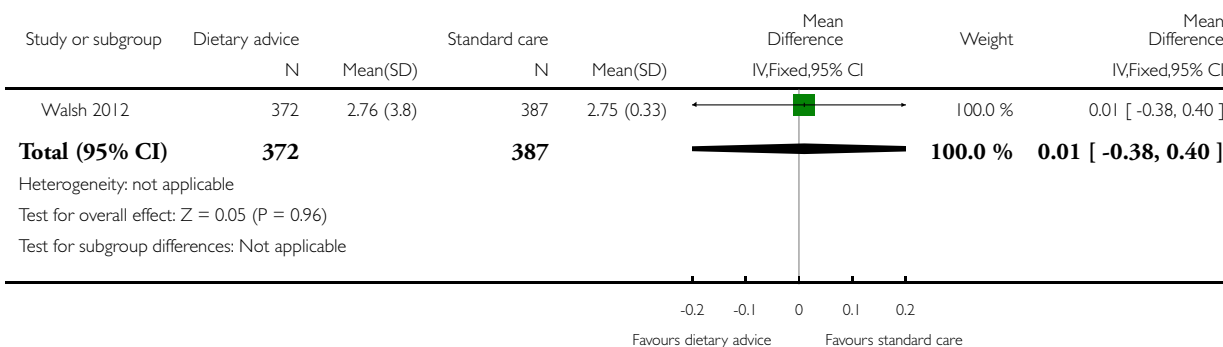
(1) SDs calculated based on 95% CIs provided

### Analysis 1.37. Comparison 1 Dietary advice interventions versus standard care, Outcome 37 Ponderal index at birth.

Review: Dietary advice interventions in pregnancy for preventing gestational diabetes mellitus

Comparison: 1 Dietary advice interventions versus standard care

Outcome: 37 Ponderal index at birth

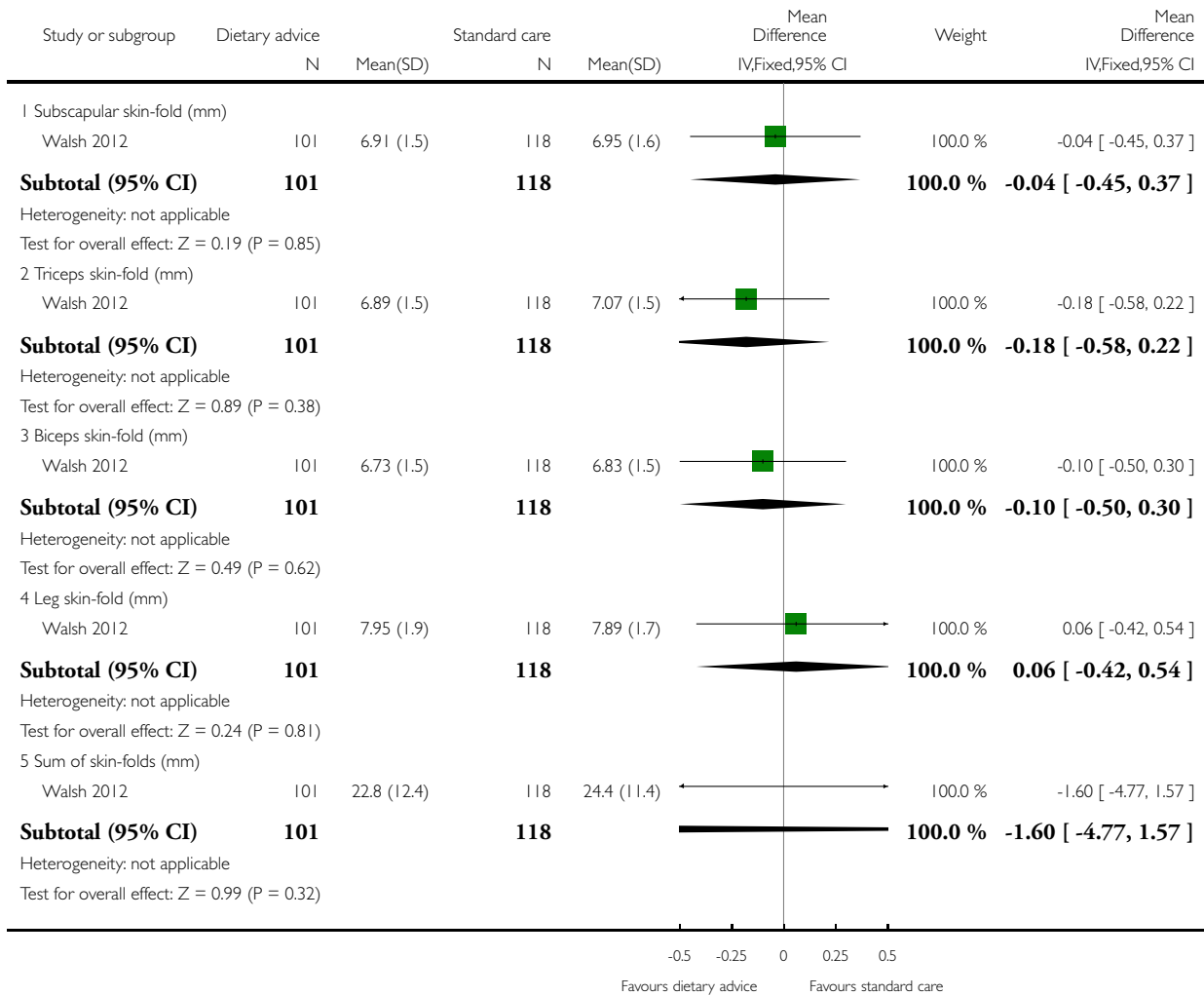


**Analysis I.38. Comparison I Dietary advice interventions versus standard care, Outcome 38 Adiposity at birth: skin-fold thickness (mm).**

Review: Dietary advice interventions in pregnancy for preventing gestational diabetes mellitus

Comparison: I Dietary advice interventions versus standard care

Outcome: 38 Adiposity at birth: skin-fold thickness (mm)

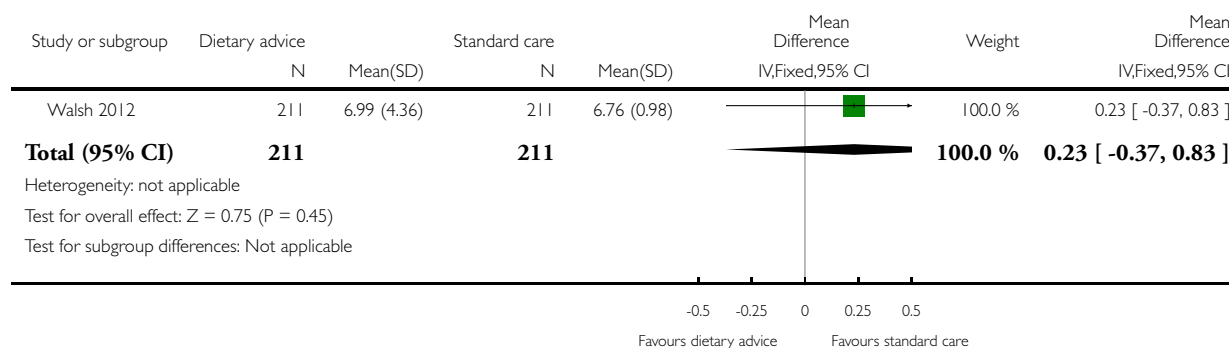


### Analysis 1.39. Comparison I Dietary advice interventions versus standard care, Outcome 39 Weight at 3 months (kg).

Review: Dietary advice interventions in pregnancy for preventing gestational diabetes mellitus

Comparison: I Dietary advice interventions versus standard care

Outcome: 39 Weight at 3 months (kg)

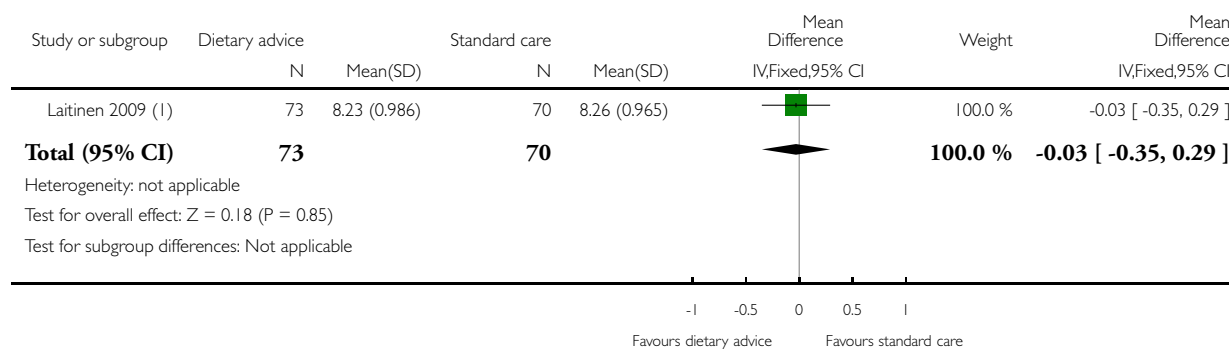


### Analysis 1.40. Comparison I Dietary advice interventions versus standard care, Outcome 40 Weight at 6 months (kg).

Review: Dietary advice interventions in pregnancy for preventing gestational diabetes mellitus

Comparison: I Dietary advice interventions versus standard care

Outcome: 40 Weight at 6 months (kg)



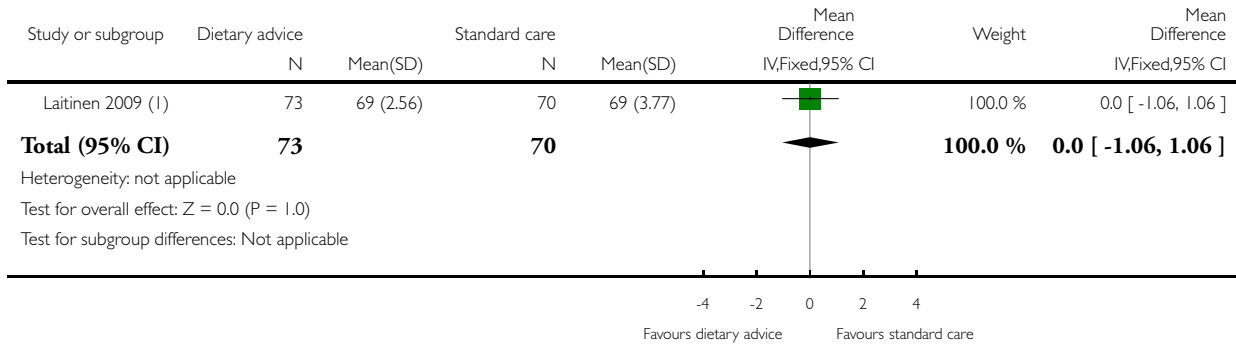
(1) SDs calculated based on 95% CIs provided

### Analysis 1.41. Comparison 1 Dietary advice interventions versus standard care, Outcome 41 Length at 6 months (cm).

Review: Dietary advice interventions in pregnancy for preventing gestational diabetes mellitus

Comparison: 1 Dietary advice interventions versus standard care

Outcome: 41 Length at 6 months (cm)



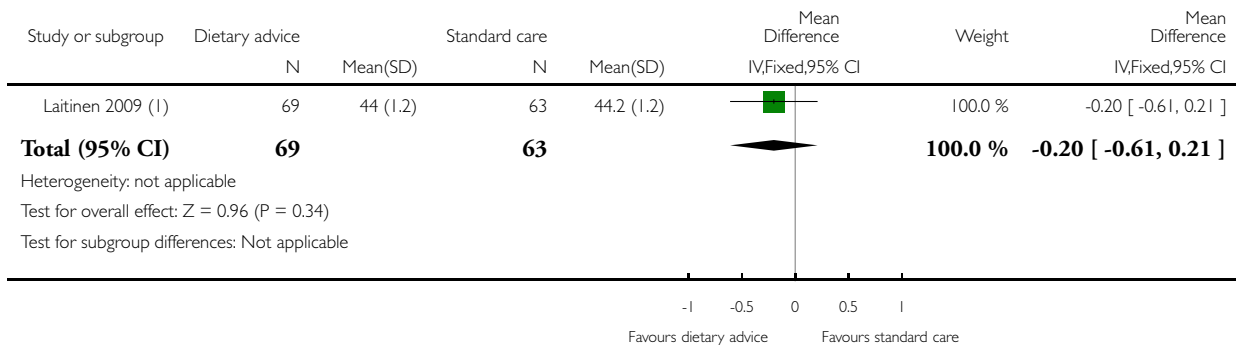
(1) SDs calculated based on 95% CIs provided

### Analysis 1.42. Comparison 1 Dietary advice interventions versus standard care, Outcome 42 Head circumference at 6 months (cm).

Review: Dietary advice interventions in pregnancy for preventing gestational diabetes mellitus

Comparison: 1 Dietary advice interventions versus standard care

Outcome: 42 Head circumference at 6 months (cm)



(1) SDs calculated based on 95% CIs provided

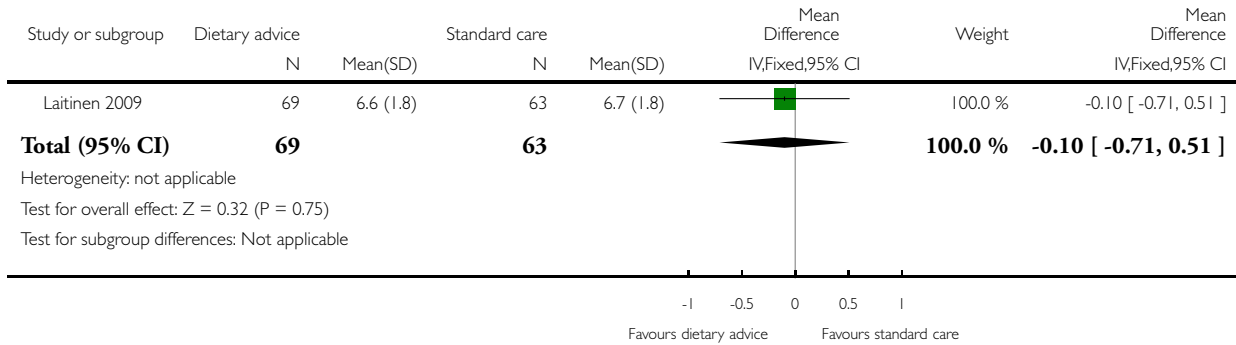


**Analysis 1.43. Comparison 1 Dietary advice interventions versus standard care, Outcome 43 Skinfold thickness at 6 months (mm).**

Review: Dietary advice interventions in pregnancy for preventing gestational diabetes mellitus

Comparison: 1 Dietary advice interventions versus standard care

Outcome: 43 Skinfold thickness at 6 months (mm)

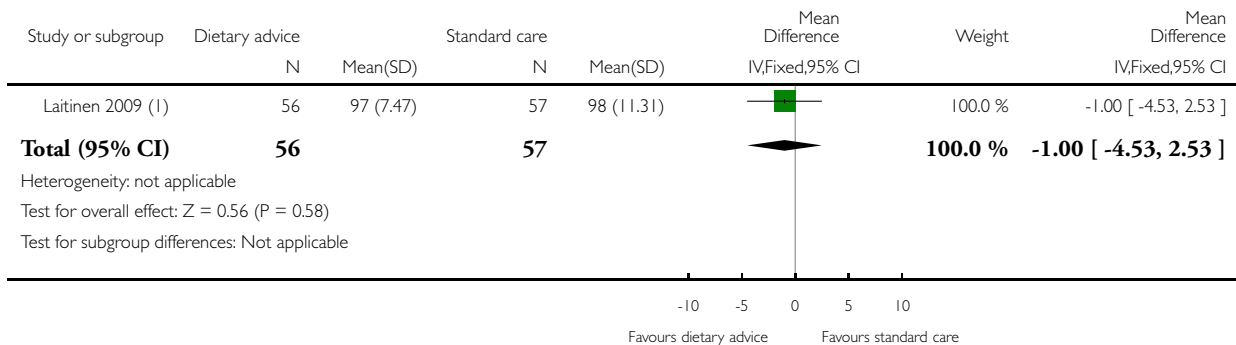


**Analysis 1.44. Comparison 1 Dietary advice interventions versus standard care, Outcome 44 Systolic blood pressure at 6 months (mmHg).**

Review: Dietary advice interventions in pregnancy for preventing gestational diabetes mellitus

Comparison: 1 Dietary advice interventions versus standard care

Outcome: 44 Systolic blood pressure at 6 months (mmHg)



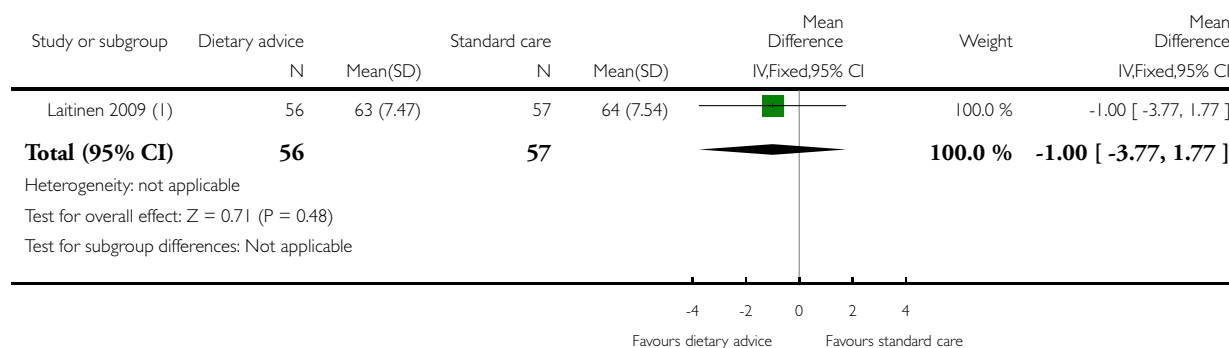
(1) SDs calculated based on 95% CIs provided

### Analysis 1.45. Comparison 1 Dietary advice interventions versus standard care, Outcome 45 Diastolic blood pressure at 6 months (mmHg).

Review: Dietary advice interventions in pregnancy for preventing gestational diabetes mellitus

Comparison: 1 Dietary advice interventions versus standard care

Outcome: 45 Diastolic blood pressure at 6 months (mmHg)



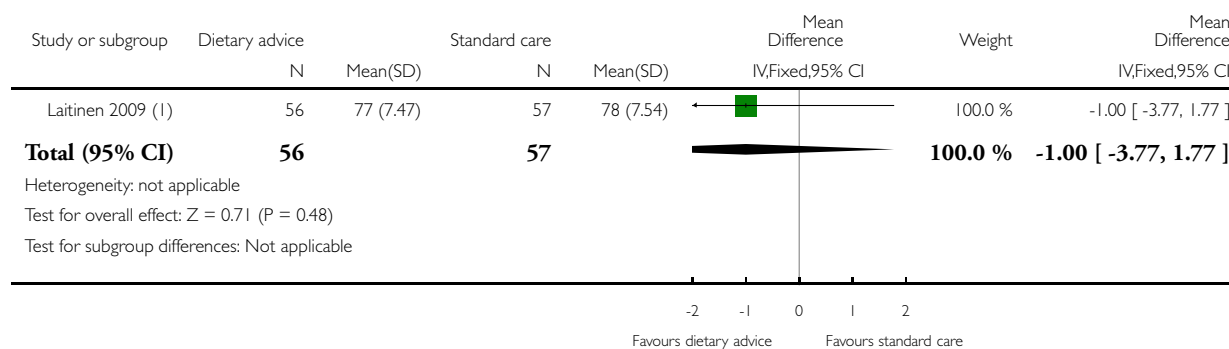
(1) SDs calculated based on 95% CIs provided

### Analysis 1.46. Comparison 1 Dietary advice interventions versus standard care, Outcome 46 Mean blood pressure at 6 months (mmHg).

Review: Dietary advice interventions in pregnancy for preventing gestational diabetes mellitus

Comparison: 1 Dietary advice interventions versus standard care

Outcome: 46 Mean blood pressure at 6 months (mmHg)



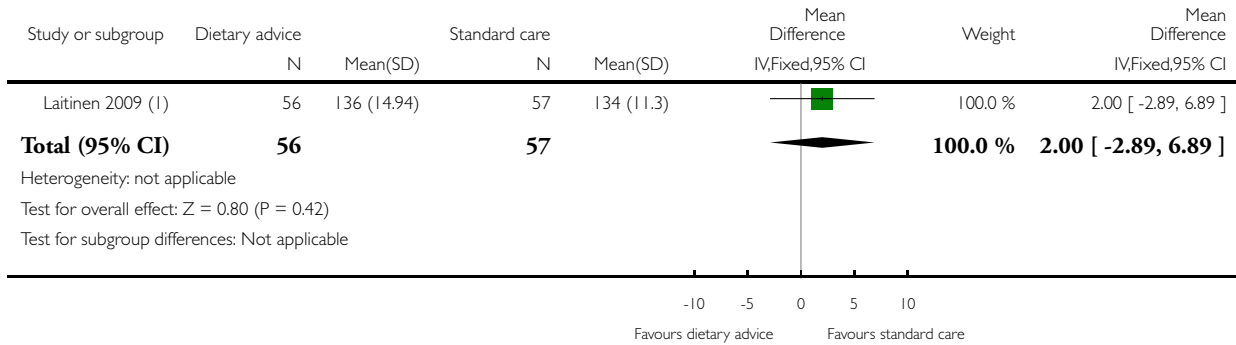
(1) SDs calculated based on 95% CIs provided

**Analysis 1.47. Comparison 1 Dietary advice interventions versus standard care, Outcome 47 Heart rate at 6 months (bpm).**

Review: Dietary advice interventions in pregnancy for preventing gestational diabetes mellitus

Comparison: 1 Dietary advice interventions versus standard care

Outcome: 47 Heart rate at 6 months (bpm)



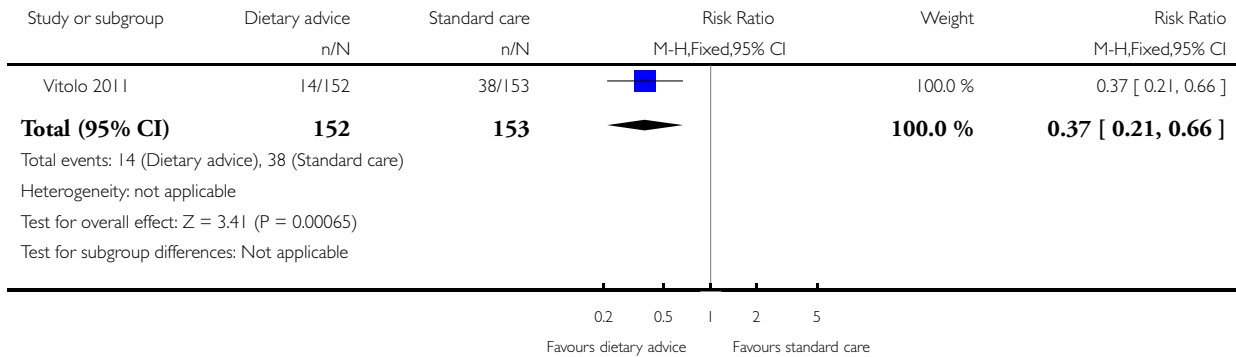
(1) SDs calculated based on 95% CIs provided

**Analysis 1.48. Comparison 1 Dietary advice interventions versus standard care, Outcome 48 Clinical complications (gestational diabetes, pre-eclampsia, low birthweight, prematurity).**

Review: Dietary advice interventions in pregnancy for preventing gestational diabetes mellitus

Comparison: 1 Dietary advice interventions versus standard care

Outcome: 48 Clinical complications (gestational diabetes, pre-eclampsia, low birthweight, prematurity)

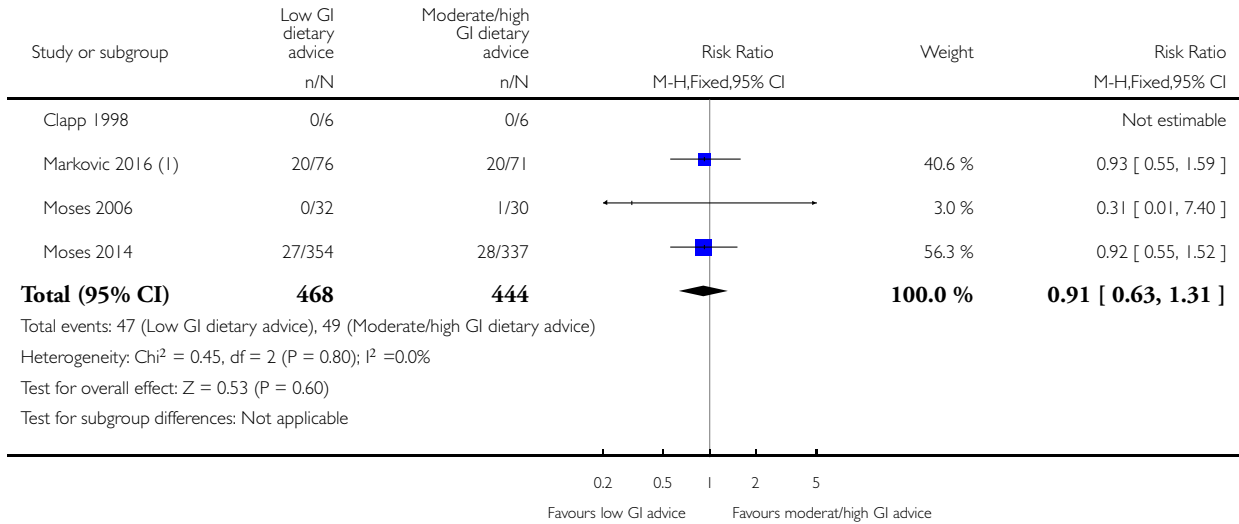


## Analysis 2.1. Comparison 2 Low-GI dietary advice versus moderate- to high-GI dietary advice, Outcome 1 Gestational diabetes.

Review: Dietary advice interventions in pregnancy for preventing gestational diabetes mellitus

Comparison: 2 Low-GI dietary advice versus moderate- to high-GI dietary advice

Outcome: 1 Gestational diabetes



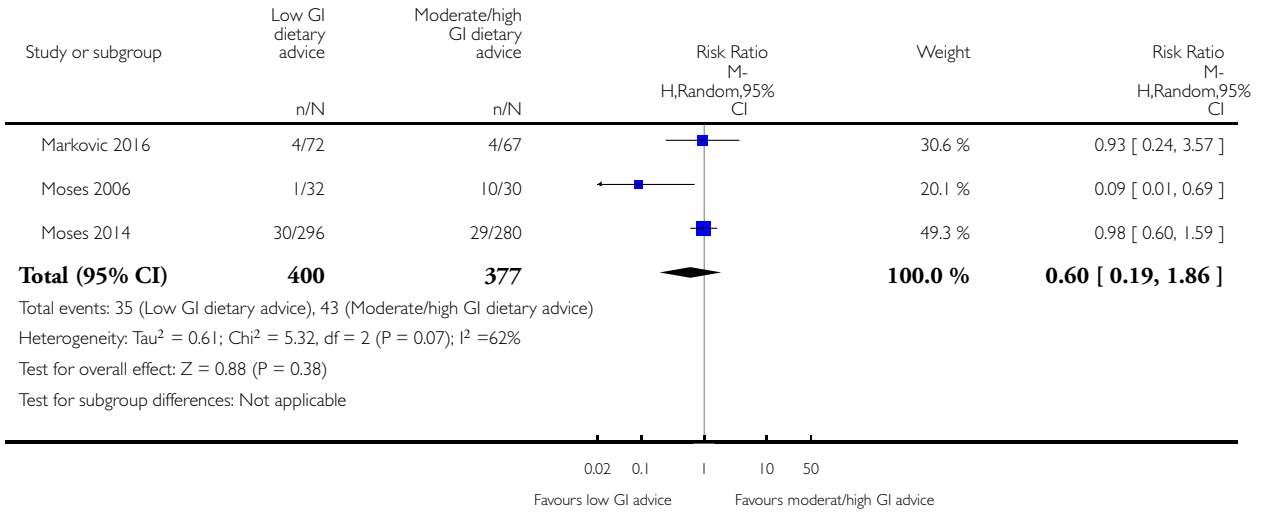
(1) 10 women developed GDM before 20 weeks in the low GI group and 11 in the moderate GI group

**Analysis 2.2. Comparison 2 Low-GI dietary advice versus moderate- to high-GI dietary advice, Outcome 2 Large-for-gestational age.**

Review: Dietary advice interventions in pregnancy for preventing gestational diabetes mellitus

Comparison: 2 Low-GI dietary advice versus moderate- to high-GI dietary advice

Outcome: 2 Large-for-gestational age

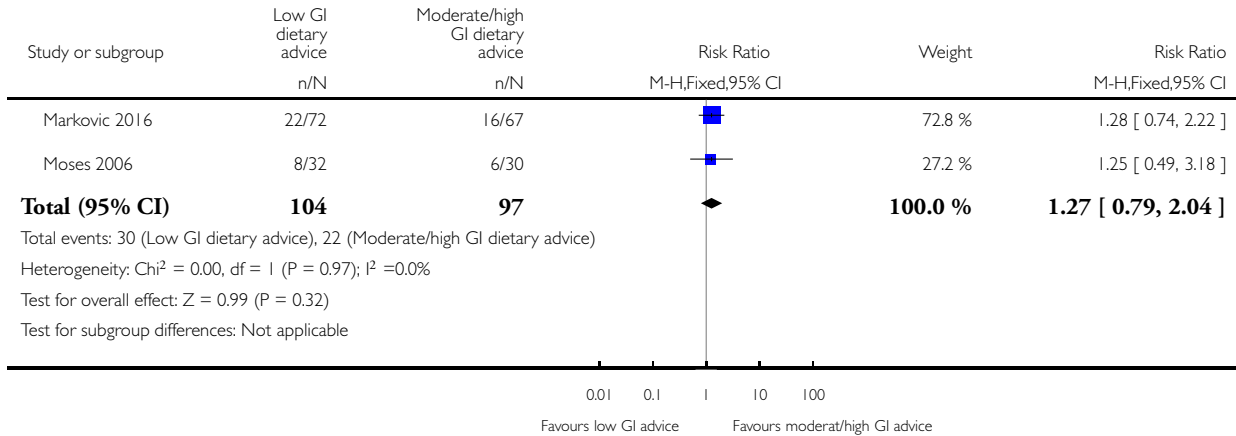


### Analysis 2.3. Comparison 2 Low-GI dietary advice versus moderate- to high-GI dietary advice, Outcome 3 Caesarean birth.

Review: Dietary advice interventions in pregnancy for preventing gestational diabetes mellitus

Comparison: 2 Low-GI dietary advice versus moderate- to high-GI dietary advice

Outcome: 3 Caesarean birth

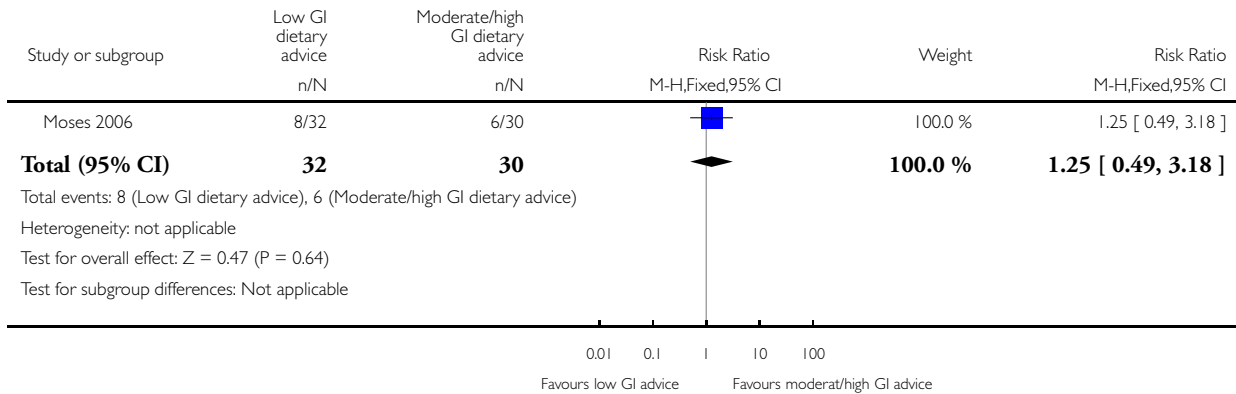


### Analysis 2.4. Comparison 2 Low-GI dietary advice versus moderate- to high-GI dietary advice, Outcome 4 Operative vaginal birth.

Review: Dietary advice interventions in pregnancy for preventing gestational diabetes mellitus

Comparison: 2 Low-GI dietary advice versus moderate- to high-GI dietary advice

Outcome: 4 Operative vaginal birth

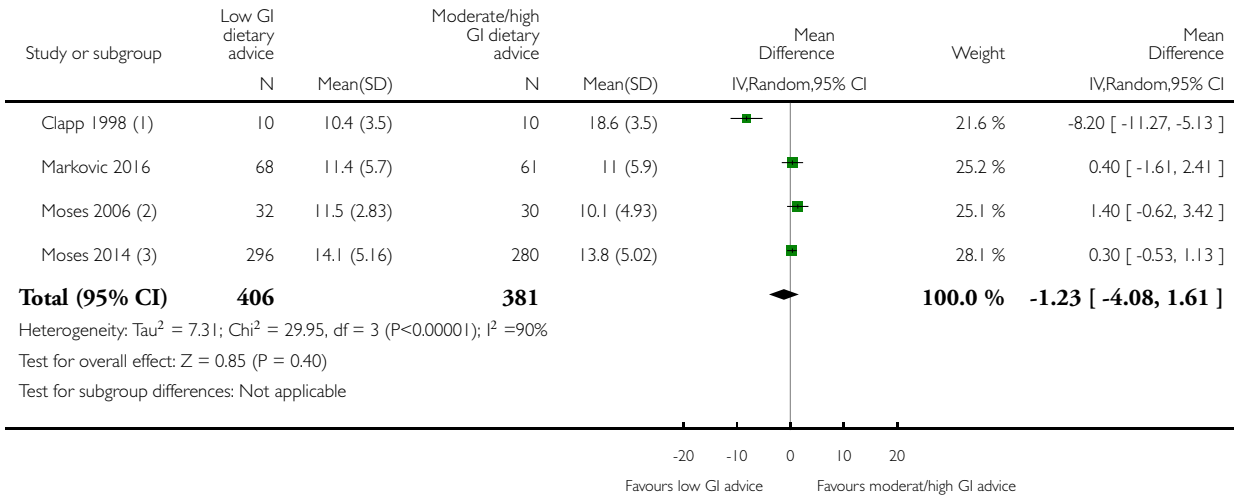


**Analysis 2.5. Comparison 2 Low-GI dietary advice versus moderate- to high-GI dietary advice, Outcome 5 Gestational weight gain (kg).**

Review: Dietary advice interventions in pregnancy for preventing gestational diabetes mellitus

Comparison: 2 Low-GI dietary advice versus moderate- to high-GI dietary advice

Outcome: 5 Gestational weight gain (kg)



(1) SDs calculated based on SEs provided

(2) SDs calculated based on SEs provided

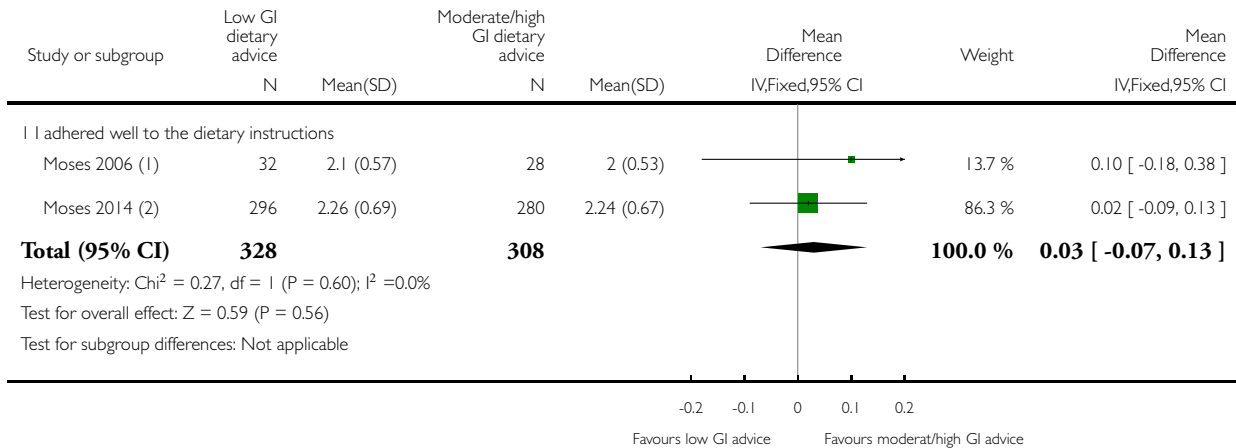
(3) SDs calculated based on SEs provided

### Analysis 2.6. Comparison 2 Low-GI dietary advice versus moderate- to high-GI dietary advice, Outcome 6 Adherence to the intervention.

Review: Dietary advice interventions in pregnancy for preventing gestational diabetes mellitus

Comparison: 2 Low-GI dietary advice versus moderate- to high-GI dietary advice

Outcome: 6 Adherence to the intervention



(1) 5-point Likert scale (1 being "all of the time" and 5 being "none of the time"); SDs calculated based on SEs provided

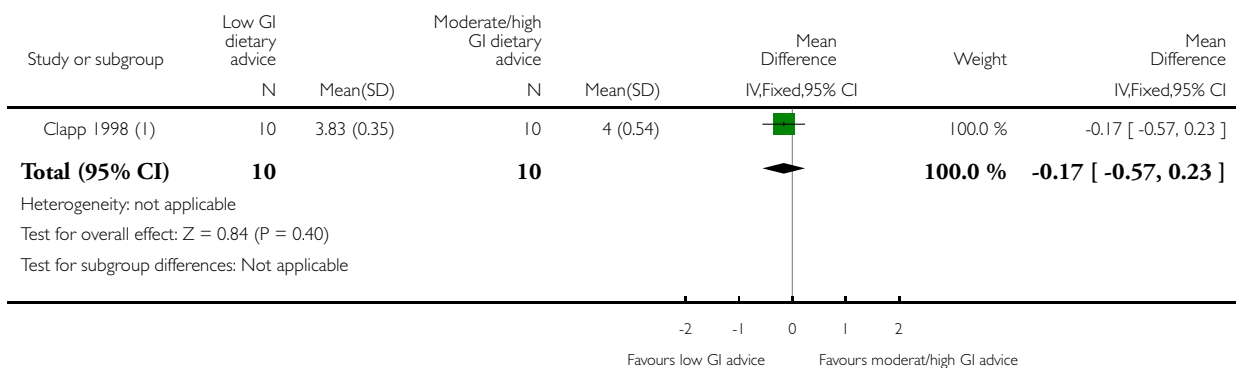
(2) 5-point Likert scale (1 being "all of the time" and 5 being "none of the time"); SDs calculated based on SEs provided

### Analysis 2.7. Comparison 2 Low-GI dietary advice versus moderate- to high-GI dietary advice, Outcome 7 Fasting glucose at 24-28 weeks (mmol/L).

Review: Dietary advice interventions in pregnancy for preventing gestational diabetes mellitus

Comparison: 2 Low-GI dietary advice versus moderate- to high-GI dietary advice

Outcome: 7 Fasting glucose at 24-28 weeks (mmol/L)





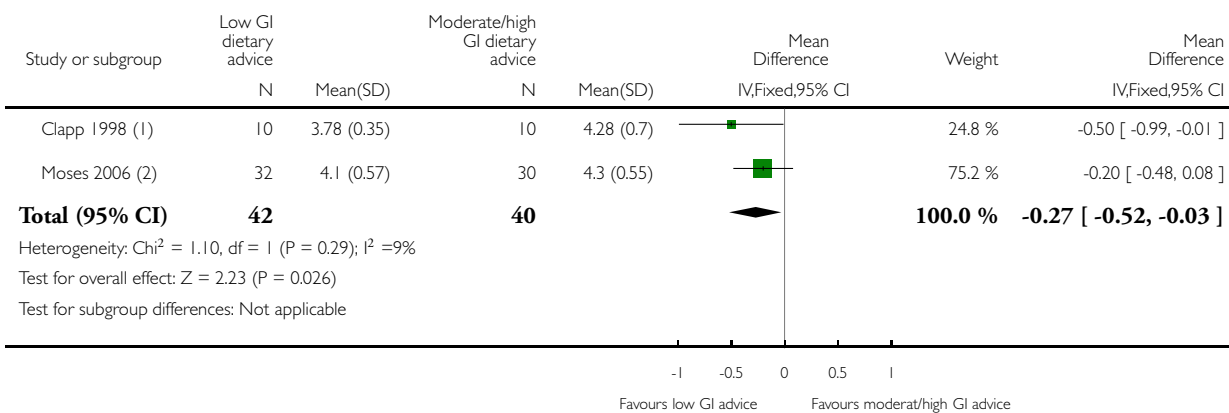
(1) SDs calculated based on SEs provided

### Analysis 2.8. Comparison 2 Low-GI dietary advice versus moderate- to high-GI dietary advice, Outcome 8 Fasting glucose at 32-36 weeks (mmol/L).

Review: Dietary advice interventions in pregnancy for preventing gestational diabetes mellitus

Comparison: 2 Low-GI dietary advice versus moderate- to high-GI dietary advice

Outcome: 8 Fasting glucose at 32-36 weeks (mmol/L)



(1) SDs calculated based on SEs provided

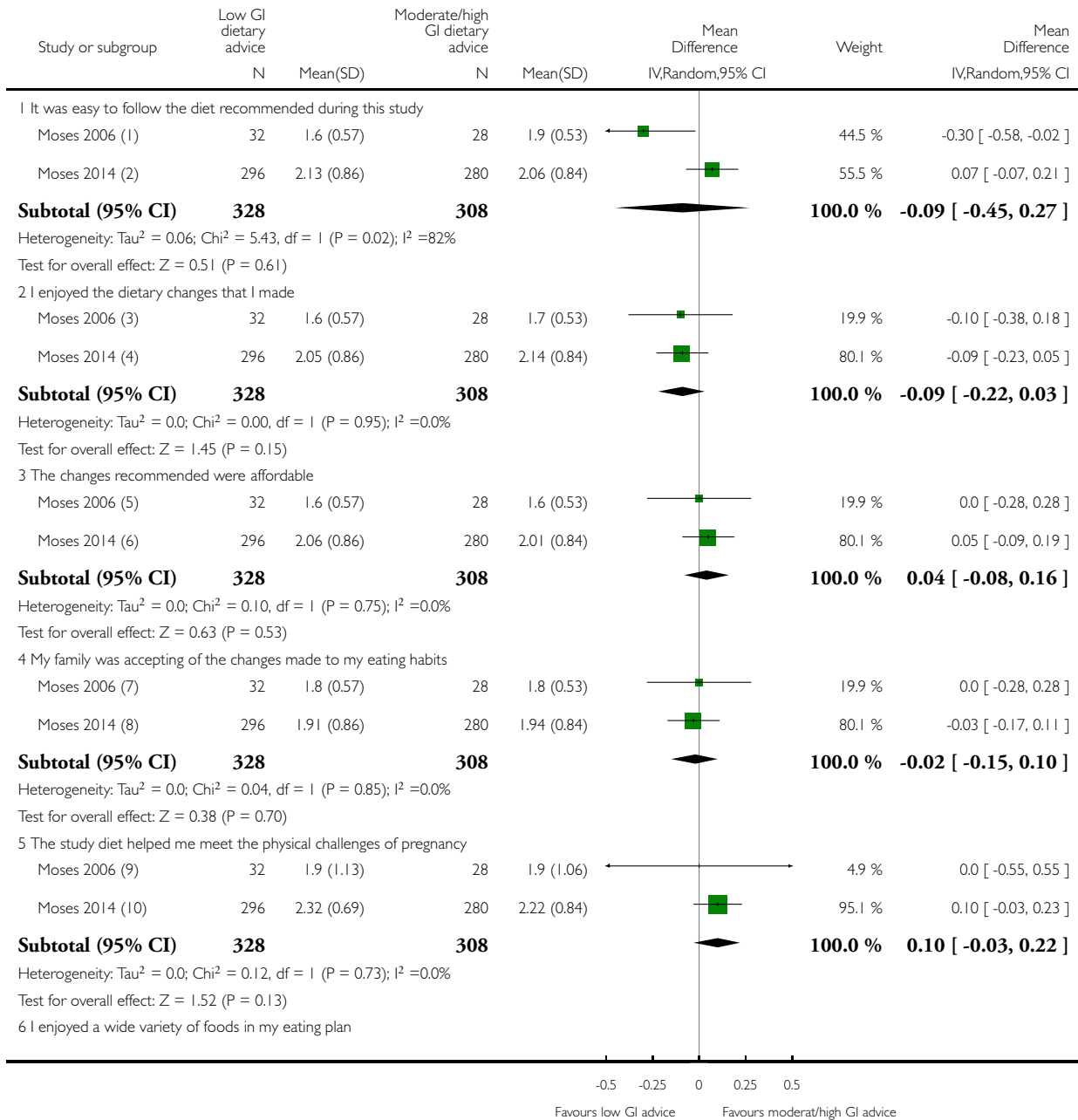
(2) SDs calculated based on SEs provided

**Analysis 2.9. Comparison 2 Low-GI dietary advice versus moderate- to high-GI dietary advice, Outcome 9 Views of the intervention.**

Review: Dietary advice interventions in pregnancy for preventing gestational diabetes mellitus

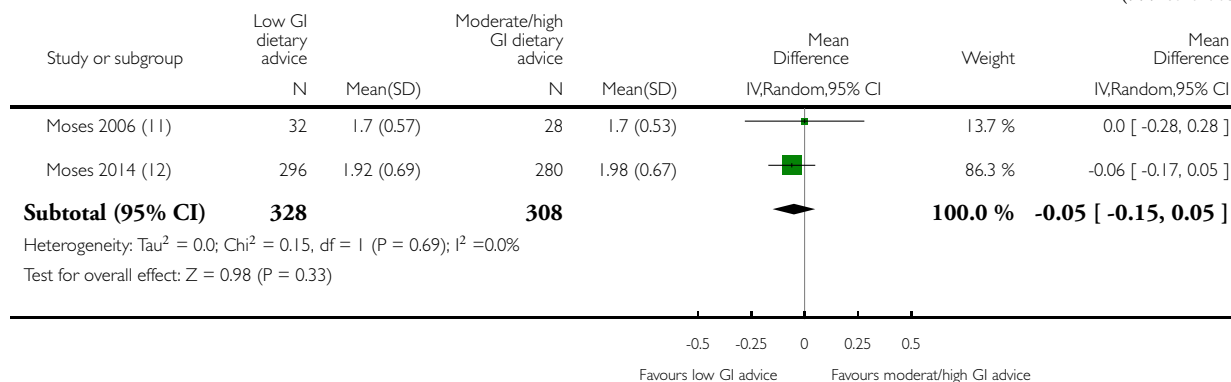
Comparison: 2 Low-GI dietary advice versus moderate- to high-GI dietary advice

Outcome: 9 Views of the intervention



(Continued ...)

(... Continued)



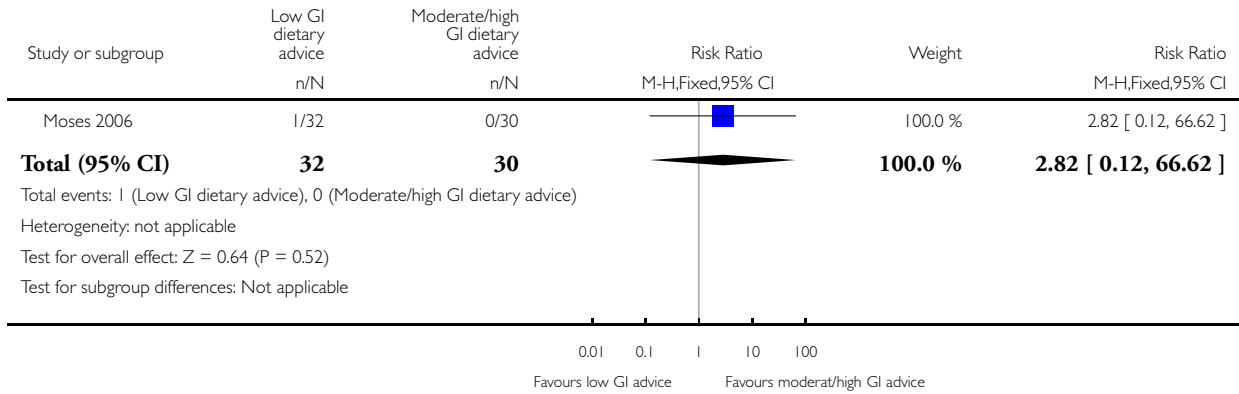
- (1) SDs calculated based on SEs provided
- (2) SDs calculated based on SEs provided
- (3) SDs calculated based on SEs provided
- (4) SDs calculated based on SEs provided
- (5) SDs calculated based on SEs provided
- (6) SDs calculated based on SEs provided
- (7) SDs calculated based on SEs provided
- (8) SDs calculated based on SEs provided
- (9) SDs calculated based on SEs provided
- (10) SDs calculated based on SEs provided
- (11) SDs calculated based on SEs provided
- (12) SDs calculated based on SEs provided

**Analysis 2.10. Comparison 2 Low-GI dietary advice versus moderate- to high-GI dietary advice, Outcome 10 Apgar score less than 7 at 5 minutes.**

Review: Dietary advice interventions in pregnancy for preventing gestational diabetes mellitus

Comparison: 2 Low-GI dietary advice versus moderate- to high-GI dietary advice

Outcome: 10 Apgar score less than 7 at 5 minutes

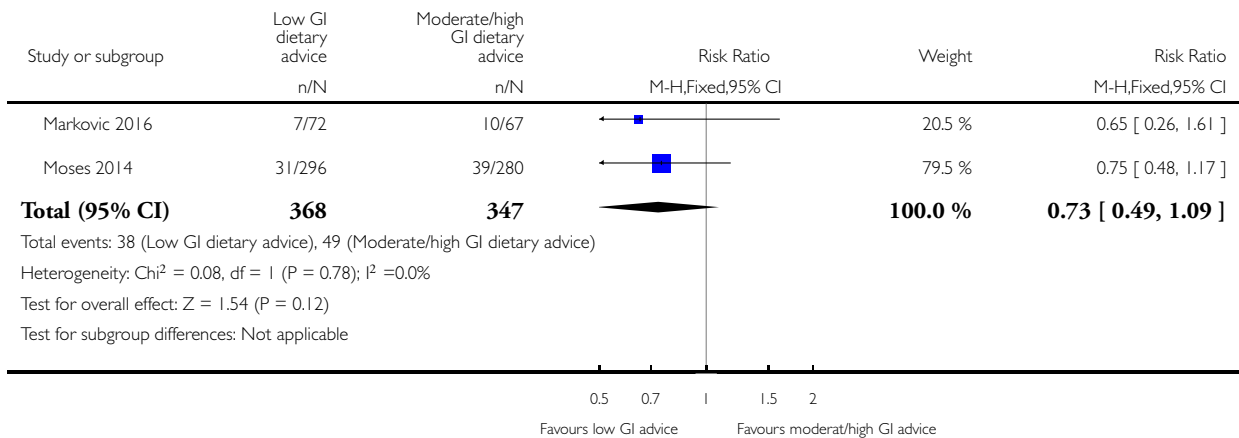


**Analysis 2.11. Comparison 2 Low-GI dietary advice versus moderate- to high-GI dietary advice, Outcome 11 Macrosomia (> 4000 g).**

Review: Dietary advice interventions in pregnancy for preventing gestational diabetes mellitus

Comparison: 2 Low-GI dietary advice versus moderate- to high-GI dietary advice

Outcome: 11 Macrosomia (> 4000 g)

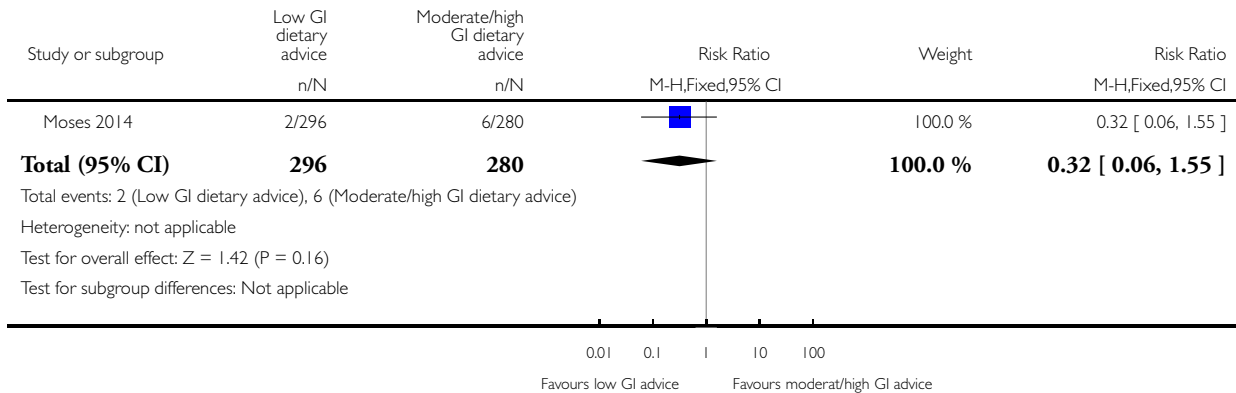


### Analysis 2.12. Comparison 2 Low-GI dietary advice versus moderate- to high-GI dietary advice, Outcome 12 Macrosomia (> 4500 g).

Review: Dietary advice interventions in pregnancy for preventing gestational diabetes mellitus

Comparison: 2 Low-GI dietary advice versus moderate- to high-GI dietary advice

Outcome: 12 Macrosomia (> 4500 g)

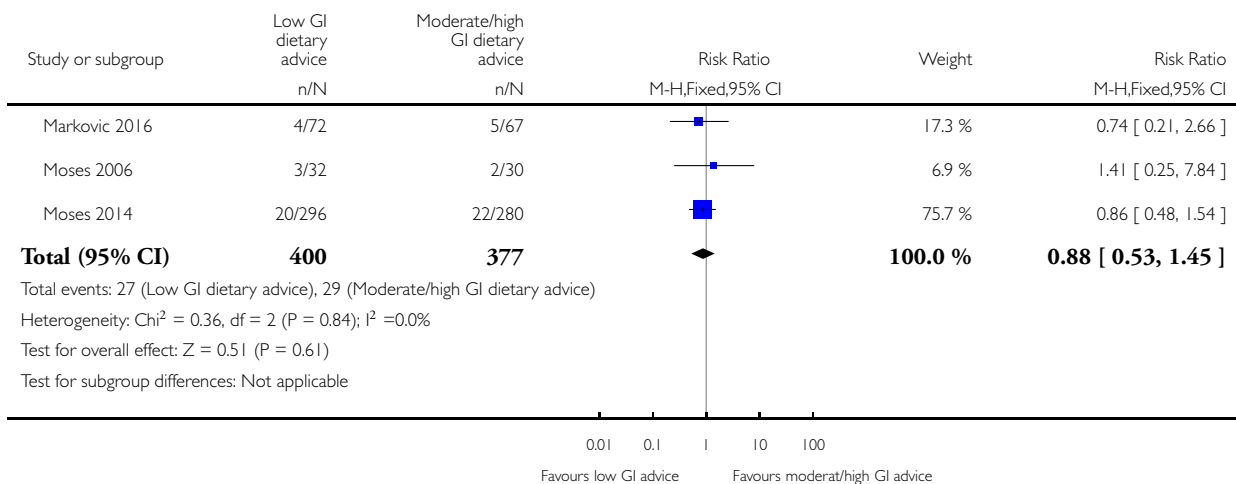


### Analysis 2.13. Comparison 2 Low-GI dietary advice versus moderate- to high-GI dietary advice, Outcome 13 Small-for-gestational age.

Review: Dietary advice interventions in pregnancy for preventing gestational diabetes mellitus

Comparison: 2 Low-GI dietary advice versus moderate- to high-GI dietary advice

Outcome: 13 Small-for-gestational age

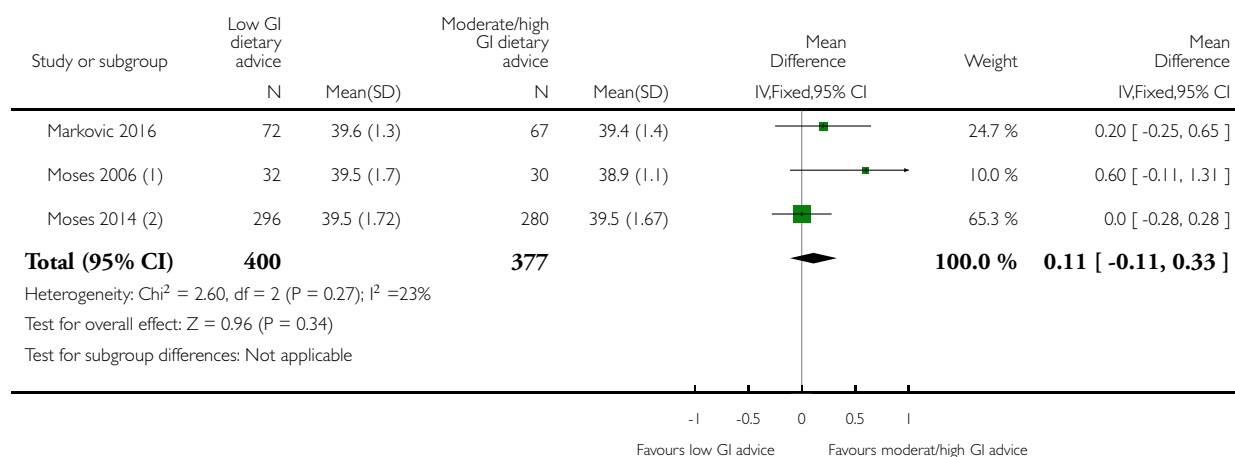


**Analysis 2.14. Comparison 2 Low-GI dietary advice versus moderate- to high-GI dietary advice, Outcome 14 Gestational age at birth (weeks).**

Review: Dietary advice interventions in pregnancy for preventing gestational diabetes mellitus

Comparison: 2 Low-GI dietary advice versus moderate- to high-GI dietary advice

Outcome: 14 Gestational age at birth (weeks)



(1) SDs calculated based on SEs provided

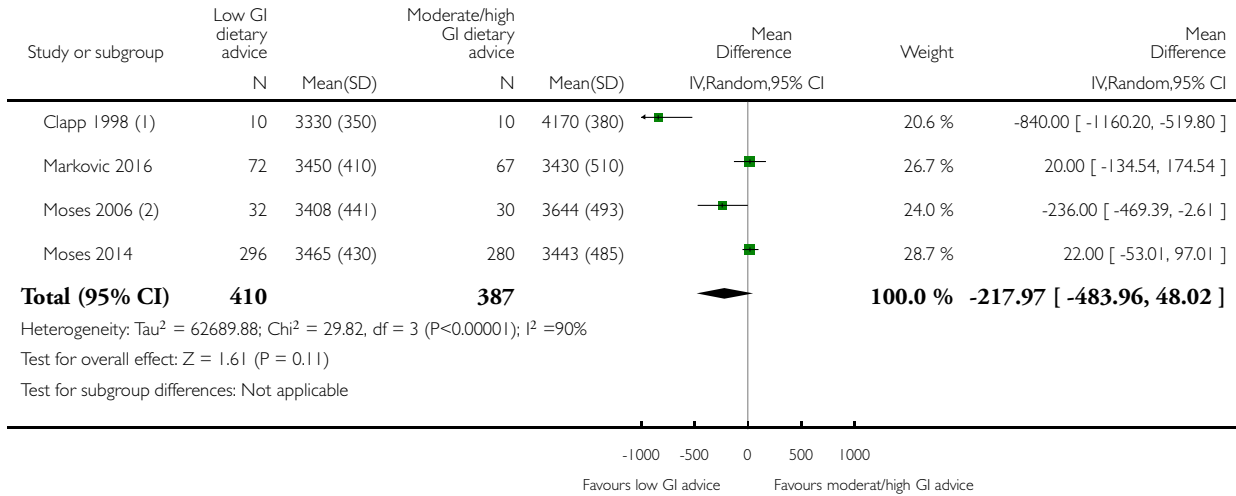
(2) SDs calculated based on SEs provided

**Analysis 2.15. Comparison 2 Low-GI dietary advice versus moderate- to high-GI dietary advice, Outcome 15 Birthweight (g).**

Review: Dietary advice interventions in pregnancy for preventing gestational diabetes mellitus

Comparison: 2 Low-GI dietary advice versus moderate- to high-GI dietary advice

Outcome: 15 Birthweight (g)



(1) SDs calculated based on SEs provided

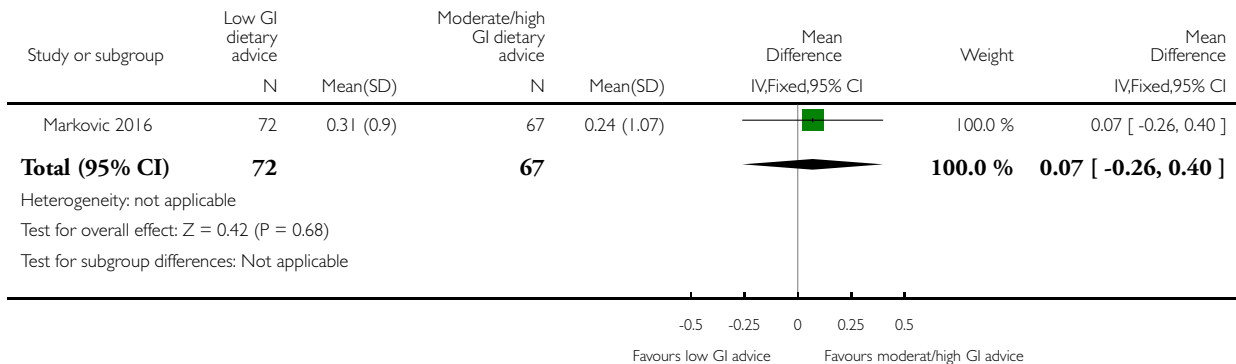
(2) SDs calculated based on SEs provided

**Analysis 2.16. Comparison 2 Low-GI dietary advice versus moderate- to high-GI dietary advice, Outcome 16 Birthweight (z score).**

Review: Dietary advice interventions in pregnancy for preventing gestational diabetes mellitus

Comparison: 2 Low-GI dietary advice versus moderate- to high-GI dietary advice

Outcome: 16 Birthweight (z score)

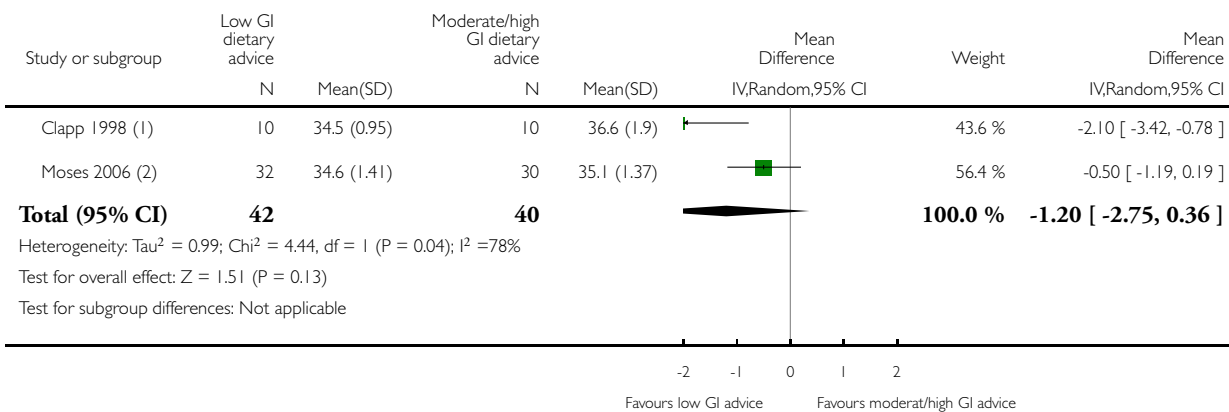


**Analysis 2.17. Comparison 2 Low-GI dietary advice versus moderate- to high-GI dietary advice, Outcome 17 Head circumference at birth (cm).**

Review: Dietary advice interventions in pregnancy for preventing gestational diabetes mellitus

Comparison: 2 Low-GI dietary advice versus moderate- to high-GI dietary advice

Outcome: 17 Head circumference at birth (cm)



(1) SDs calculated based on SEs provided

(2) SDs calculated based on SEs provided

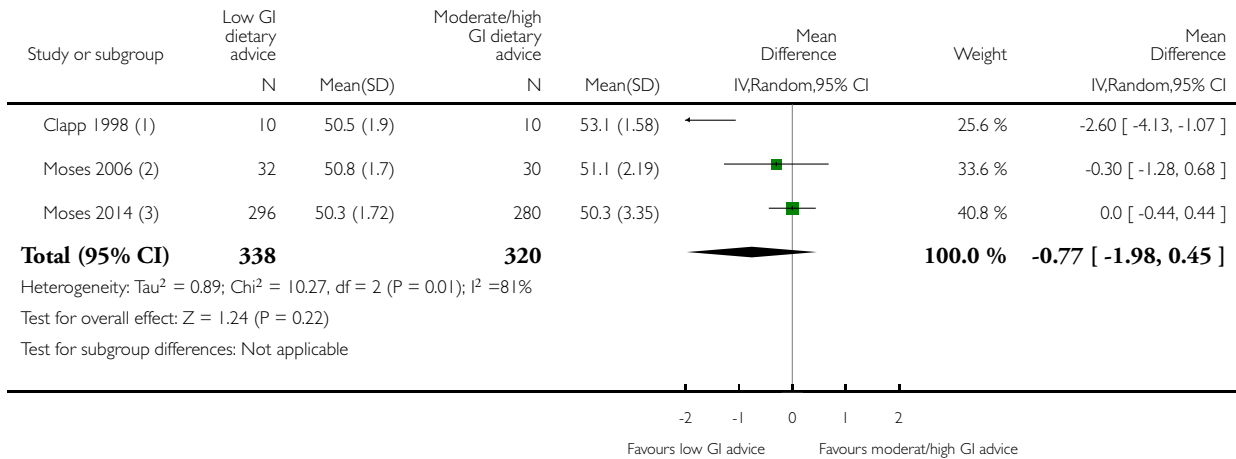


**Analysis 2.18. Comparison 2 Low-GI dietary advice versus moderate- to high-GI dietary advice, Outcome 18 Length at birth (cm).**

Review: Dietary advice interventions in pregnancy for preventing gestational diabetes mellitus

Comparison: 2 Low-GI dietary advice versus moderate- to high-GI dietary advice

Outcome: 18 Length at birth (cm)



(1) SDs calculated based on SEs provided

(2) SDs calculated based on SEs provided

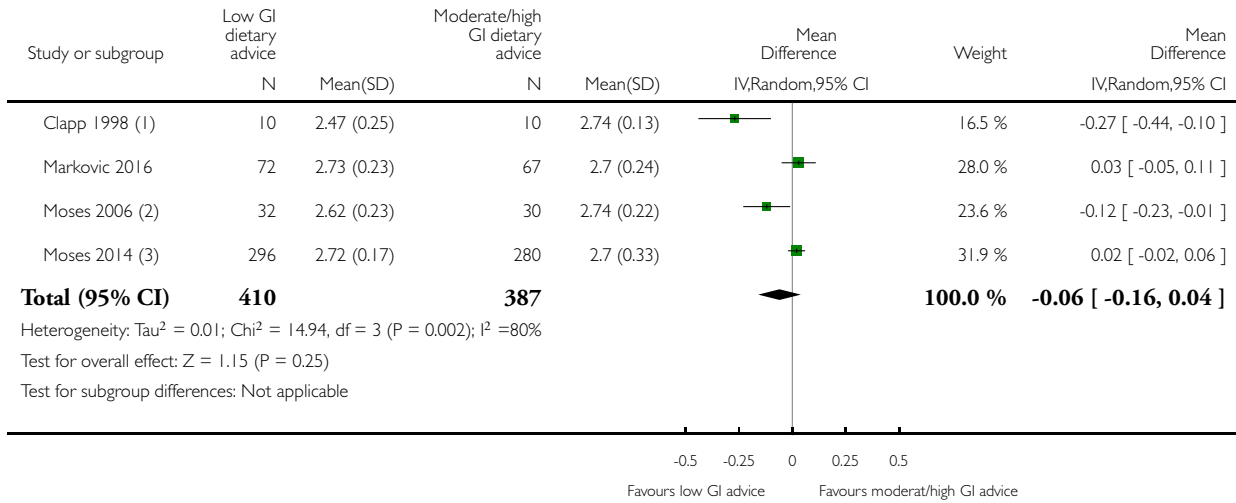
(3) SDs calculated based on SEs provided

**Analysis 2.19. Comparison 2 Low-GI dietary advice versus moderate- to high-GI dietary advice, Outcome 19 Ponderal index at birth.**

Review: Dietary advice interventions in pregnancy for preventing gestational diabetes mellitus

Comparison: 2 Low-GI dietary advice versus moderate- to high-GI dietary advice

Outcome: 19 Ponderal index at birth



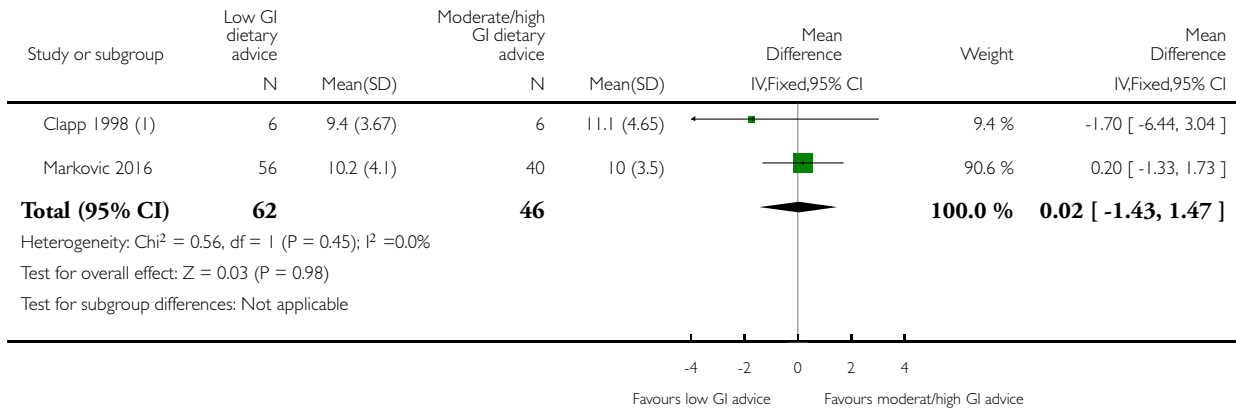
- (1) SDs calculated based on SEs provided
- (2) SDs calculated based on SEs provided
- (3) SDs calculated based on SEs provided

**Analysis 2.20. Comparison 2 Low-GI dietary advice versus moderate- to high-GI dietary advice, Outcome 20 Adiposity at birth: % body fat.**

Review: Dietary advice interventions in pregnancy for preventing gestational diabetes mellitus

Comparison: 2 Low-GI dietary advice versus moderate- to high-GI dietary advice

Outcome: 20 Adiposity at birth: % body fat



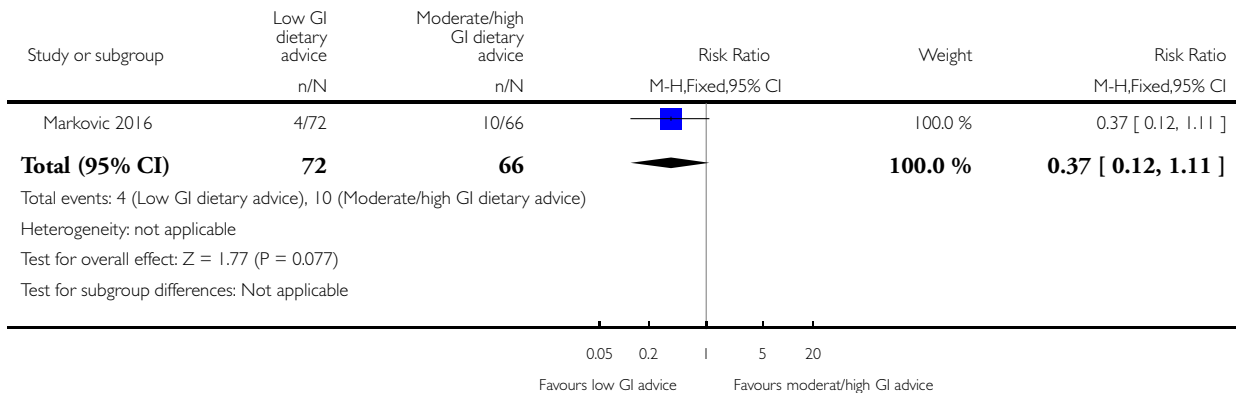
(1) SDs calculated based on SEs provided

**Analysis 2.21. Comparison 2 Low-GI dietary advice versus moderate- to high-GI dietary advice, Outcome 21 Neonatal intensive care unit admission.**

Review: Dietary advice interventions in pregnancy for preventing gestational diabetes mellitus

Comparison: 2 Low-GI dietary advice versus moderate- to high-GI dietary advice

Outcome: 21 Neonatal intensive care unit admission

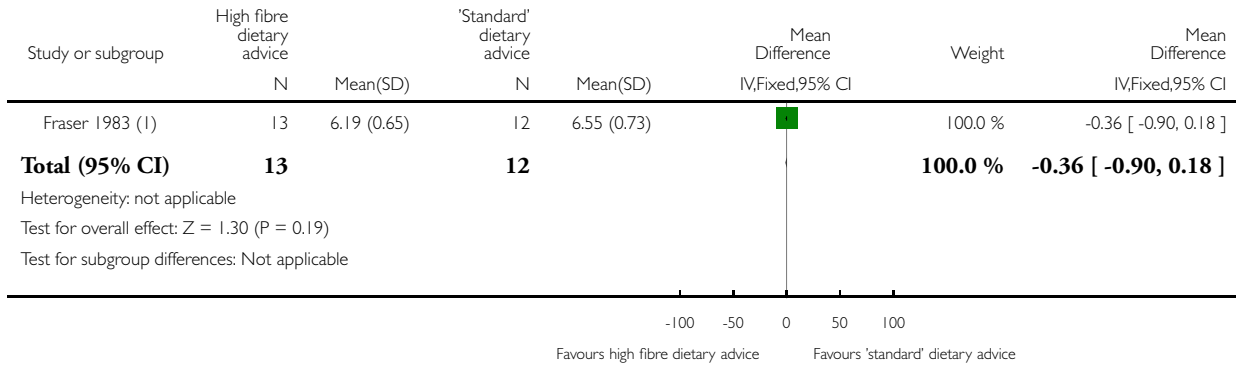


### Analysis 3.1. Comparison 3 High-fibre dietary advice versus 'standard' dietary advice, Outcome 1 OGTT at 35 weeks (mmol/L).

Review: Dietary advice interventions in pregnancy for preventing gestational diabetes mellitus

Comparison: 3 High-fibre dietary advice versus 'standard' dietary advice

Outcome: 1 OGTT at 35 weeks (mmol/L)



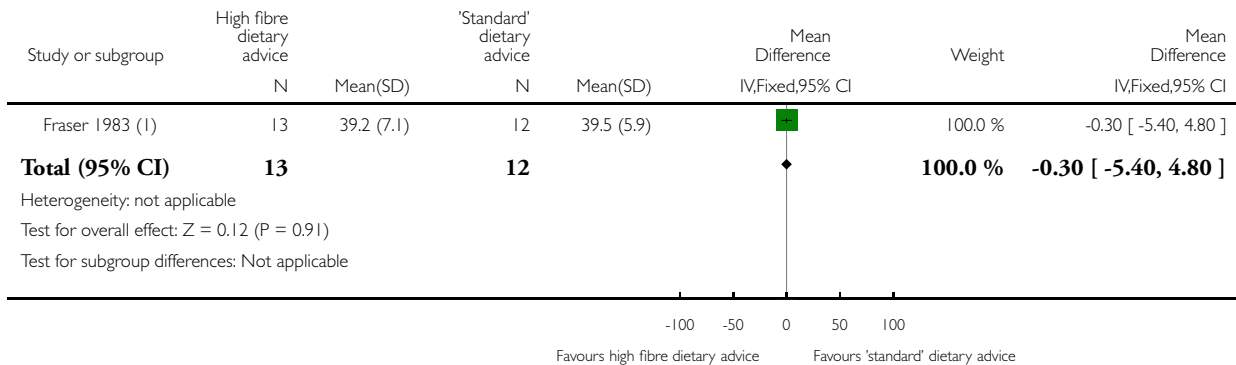
(1) SDs calculated based on SEs provided

### Analysis 3.2. Comparison 3 High-fibre dietary advice versus 'standard' dietary advice, Outcome 2 Birthweight centile.

Review: Dietary advice interventions in pregnancy for preventing gestational diabetes mellitus

Comparison: 3 High-fibre dietary advice versus 'standard' dietary advice

Outcome: 2 Birthweight centile



(1) SDs calculated based on SEs provided

## WHAT'S NEW

Last assessed as up-to-date: 3 January 2016.

Date	Event	Description
3 January 2016	New search has been performed	Searched updated. Eight new trials have been included ( <a href="#">Laitinen 2009</a> ; <a href="#">Markovic 2016</a> ; <a href="#">Moses 2014</a> ; <a href="#">Quinlivan 2011</a> ; <a href="#">Thornton 2009</a> ; <a href="#">Vitolo 2011</a> ; <a href="#">Walsh 2012</a> ; <a href="#">Wolff 2008</a> ) in this update. Methods updated, including four 'Summary of findings' tables. An additional 'dietary advice interventions versus standard care' comparison has been added, and outcomes have been revised (see <a href="#">Differences between protocol and review</a> ). One new author (Emily Shepherd) was involved in this update
3 January 2016	New citation required and conclusions have changed	Dietary advice interventions versus standard care (six new trials): all conclusions are new Low GI dietary advice versus moderate/high GI dietary advice (four trials, two of which were included in previous version): in regards to primary outcomes: the reduction in large-for-gestational age infants with low GI dietary advice observed in the previous version of the review, was no longer apparent in this update. Considering secondary outcomes: the lower ponderal indices and birthweights for infants born to mothers who received low GI dietary advice seen in the previous version of the review, were no longer observed in this update High fibre dietary advice versus standard dietary advice comparison (one trial, which was included in previous version): no change to conclusions

## HISTORY

Protocol first published: Issue 3, 2007

Review first published: Issue 2, 2008

Date	Event	Description
10 January 2011	Amended	Contact details updated.
13 February 2009	Amended	Contact details updated.
15 February 2008	Amended	Converted to new review format.

## CONTRIBUTIONS OF AUTHORS

In this update of the review Joanna Tieu and Emily Shepherd, assessed studies for eligibility and extracted data. Emily Shepherd drafted the first version of the update and all authors made comments on subsequent drafts and contributed to the final version.

In the previous version of this review Joanna Tieu and Philippa Middleton assessed studies for inclusion and extracted data. Caroline Crowther also consulted on study inclusion and data extraction. The review was written by Joanna Tieu with help from Caroline Crowther and Philippa Middleton.

Joanna Tieu researched and wrote the protocol with aid and regular feedback from Professor Caroline Crowther and Philippa Middleton.

## DECLARATIONS OF INTEREST

Joanna Tieu: none known.

Emily Shepherd: none known.

Philippa Middleton: none known.

Caroline A Crowther: none known.

## SOURCES OF SUPPORT

### Internal sources

- ARCH: Australian Research Centre for Health of Women and Babies, Robinson Research Institute, The University of Adelaide, Australia.

### External sources

- NHMRC: National Health and Medical Research Council, Australia Funding for the PCG Australian and New Zealand Satellite, Australia.
- National Institute for Health Research (NIHR), UKNIHR Cochrane Programme Grant Project: 13/89/05 - Pregnancy and childbirth systematic reviews to support clinical guidelines, UK.

## DIFFERENCES BETWEEN PROTOCOL AND REVIEW

In this update of the review:

- We have updated our primary and secondary review outcomes to be in line with those core outcomes that are/will be used in other Cochrane Pregnancy and Childbirth GDM reviews.
- We have updated the methods in line with those in the standard template used by the Cochrane Pregnancy and Childbirth Group.
- We have used the GRADE approach to assess the quality of the body of evidence and we have included 'Summary of findings' tables.

## **INDEX TERMS**

### **Medical Subject Headings (MeSH)**

\*Diet; \*Glycemic Index; Adiposity; Birth Weight; Cesarean Section [statistics & numerical data]; Diabetes, Gestational [\*prevention & control]; Dietary Fiber [\*administration & dosage]; Randomized Controlled Trials as Topic

### **MeSH check words**

Female; Humans; Infant, Newborn; Pregnancy