Lifestyle interventions for the treatment of women with gestational diabetes

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Authors

Julie Brown[1], Nisreen A Alwan[2], Jane West[3], Stephen Brown[4], Christopher JD McKinlay[1], Diane Farrar[5], Caroline A Crowther[1]

[1]Liggins Institute, The University of Auckland, Auckland, New Zealand
[2]Academic Unit of Primary Care and Population Sciences, Faculty of Medicine, University of Southampton, Southampton, UK
[3]Bradford Institute for Health Research, Bradford Teaching Hospitals NHS Foundation Trust, Bradford, UK
[4]School of Interprofessional Health Studies, Auckland University of Technology, Auckland, New Zealand
[5]Maternal and Child Health, Bradford Institute for Health Research, Bradford, UK

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Contact person

Julie Brown

Cochrane Sytematic Reviewer
Liggins Institute
The University of Auckland
Park Rd
Grafton
Auckland
1142
New Zealand

E-mail: j.brown@auckland.ac.nz

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Abstract

Background

Gestational diabetes (GDM) is glucose intolerance, first recognised in pregnancy and usually resolving after birth. GDM is associated with both short- and long-term adverse effects for the mother and her infant. Lifestyle interventions are the primary therapeutic strategy for many women with GDM.

Objectives

To evaluate the effects of combined lifestyle interventions with or without pharmacotherapy in treating women with gestational diabetes.

Search methods

We searched the Pregnancy and Childbirth Group's Trials Register (14 May 2016), ClinicalTrials.gov, WHO International Clinical Trials Registry Platform (ICTRP) (14th May 2016) and reference lists of retrieved studies.

Selection criteria

We included only randomised controlled trials comparing a lifestyle intervention with usual care or another intervention for the treatment of pregnant women with GDM. Quasi-randomised trials were excluded. Cross-over trials were not eligible for inclusion. Women with pre-existing type 1 or type 2 diabetes were excluded.

Data collection and analysis

We used standard methodological procedures expected by the Cochrane Collaboration. All selection of studies, data extraction was conducted independently by two review authors.

Main results

Fifteen trials (in 45 reports) are included in this review (4501 women, 3768 infants). None of the trials were funded by a conditional grant from a pharmaceutical company. The lifestyle interventions included a wide variety of components such as education, diet, exercise and self-monitoring of blood glucose. The control group included usual antenatal care or diet alone. Using GRADE methodology, the quality of the evidence ranged from high to very low quality. The main reasons for downgrading evidence were inconsistency and risk of bias. We summarised the following data from the important outcomes of this review.

Lifestyle intervention versus control group

For the mother:

There was no clear evidence of a difference between lifestyle intervention and control groups for the risk of **hypertensive disorders of pregnancy (pre-eclampsia)** (average risk ratio (RR) 0.70; 95% confidence interval (CI) 0.40 to 1.22; four trials, 2796 women; I^2 = 79%, Tau^2 = 0.23; *low-quality evidence*); **caesarean section** (average RR 0.90; 95% CI 0.78 to 1.05; 10 trials, 3545 women; I^2 = 48%, Tau^2 = 0.02; *low-quality evidence*); **development of type 2 diabetes** *(*up to a maximum of 10 years follow-up) (RR 0.98, 95% CI 0.54 to 1.76; two trials, 486 women; I^2 = 16%; *low-quality evidence*); **perineal trauma/tearing** (RR 1.04, 95% CI 0.93 to 1.18; one trial, n = 1000 women; *moderate-quality evidence*) or **induction of labour** (average RR 1.20, 95% CI 0.99 to 1.46; four trials, n = 2699 women; I^2 = 37%; *high-quality evidence*).

More women in the lifestyle intervention group had **met postpartum weight goals** one year after birth than in the control group (RR 1.75, 95% CI 1.05 to 2.90; 156 women; one trial, *low-quality evidence*). Lifestyle interventions were associated with a decrease in the risk of **postnatal depression** compared with the control group (RR 0.49, 95% CI 0.31 to 0.78; one trial, n = 573 women; *low-quality evidence*).

For the infant/child/adult:

Lifestyle interventions were associated with a reduction in the risk of being born **large-for-gestational age (LGA)** (RR 0.60, 95% CI 0.50 to 0.71; six trials, 2994 infants; I^2 = 4%; *moderate-quality evidence*). Birthweight and the incidence of macrosomia were lower in the lifestyle intervention group.

Exposure to the lifestyle intervention was associated with decreased **neonatal fat mass** compared with the control group (mean difference (MD) -37.30 g, 95% CI -63.97 to -10.63; one trial, 958 infants; *low-quality evidence*). In childhood, there was no clear evidence of a difference between groups for **body mass index (BMI)** ≥ 85th percentile (RR 0.91, 95% CI 0.75 to 1.11; three trials, 767 children; I^2 = 4%; *moderate-quality evidence*).

There was no clear evidence of a difference between lifestyle intervention and control groups for the risk of **perinatal death** (RR 0.09, 95% CI 0.01 to 1.70; two trials, 1988 infants; *low-quality evidence*). Of 1988 infants, only five events were reported in total in the control group and there were no events in the lifestyle group. There was no clear evidence of a difference between lifestyle intervention and control groups for a **composite of serious infant outcome/s** (average RR 0.57, 95% CI 0.21 to 1.55; two trials, 1930 infants; I^2 = 82%, Tau^2 = 0.44; *very low-quality evidence*) or **neonatal hypoglycaemia** (average RR 0.99, 95% CI 0.65 to 1.52; six trials, 3000 infants; I^2 = 48%, Tau^2 = 0.12; *moderate-quality evidence*).

Diabetes and adiposity in adulthood and **neurosensory disability in later childhood** were not prespecified or reported as outcomes for any of the trials included in this review.

Authors' conclusions

Lifestyle interventions are the primary therapeutic strategy for women with GDM. Women receiving lifestyle interventions were less likely to have postnatal depression and were more likely to achieve postpartum weight goals. Exposure to lifestyle interventions was associated with a decreased risk of the baby being born LGA and decreased neonatal adiposity. Long-term maternal and childhood/adulthood outcomes were poorly reported.

The value of lifestyle interventions in low-and middle-income countries or for different ethnicities remains unclear. The longer-term benefits or harms of lifestyle interventions remains unclear due to limited reporting.

The contribution of individual components of lifestyle interventions could not be assessed. Ten per cent of participants also received some form of pharmacological therapy. Lifestyle interventions are useful as the primary therapeutic strategy and most commonly include healthy eating, physical activity and self-monitoring of blood glucose concentrations.

Future research could focus on which specific interventions are most useful (as the sole intervention without pharmacological treatment), which health professionals should give them and the optimal format for providing the information. Evaluation of long-term outcomes for the mother and her child should be a priority when planning future trials. There has been no in-depth exploration of the costs ‘saved’ from reduction in risk of LGA/macrosomia and potential longer-term risks for the infants.

Plain language summary

Lifestyle interventions for treating women with gestational diabetes (or diabetes in pregnancy)

What is the issue?

Gestational diabetes (GDM), is a glucose intolerance leading to high blood glucose levels that is first recognised during pregnancy and which usually normalises after giving birth. Diabetes during pregnancy has been linked to many short-term and long-term health problems for the mother and her baby. The main way to treat GDM is through lifestyle changes such as diet, exercise and checking blood glucose levels.

Why is this important?

Women with GDM have an increased risk of developing high blood pressure during pregnancy (pre-eclampsia) and are more likely to have their labour induced. The babies of women with GDM are more likely to be large when born and this can be linked to babies having birth trauma (bones broken or nerves damaged during the birth) and the need for giving birth by caesarean section. Lifestyle interventions that include two or more components of dietary advice, physical activity, education, and self-monitoring of blood glucose are the first-line treatment for most women diagnosed with GDM. Interventions such as healthy eating and physical activity aim to help women maintain their blood glucose levels within a target range and to improve health outcomes for the mother and baby.

What evidence did we find?

We searched the literature (May 2016) for controlled trials comparing lifestyle intervention with a control group of women receiving usual care or another intervention. Fifteen randomised controlled trials (45 publications) are included in this review, involving 4501 women and 3768 infants. None of the trials were funded by a conditional grant from a pharmaceutical company.

For the baby, lifestyle interventions were associated with a reduction in the risk of being born large-for-gestational age (six trials, 2994 infants). The number of babies with birthweight over 4000 g (macrosomia) was lower with the lifestyle intervention, with no clear difference in the number of newborn babies experiencing low blood glucose levels (six trials, 3000 infants). The evidence was of moderate quality for these findings. Birthweight was also lower in the lifestyle intervention group.

For the mothers, introducing lifestyle interventions made no clear difference in the number of women with pregnancy-induced high blood pressure (four trials, 2796 women) or having a caesarean section (10 trials, 3545 women) based on low-quality evidence or on induction of labour (four trials, 2699 women, high-quality evidence). Similar numbers of women experienced perineal trauma or tearing (one trial, 1000 women) or developed type 2 diabetes at a maximum of 10 years after giving birth (two trials, 486 women). These findings were supported by low- to moderate-quality evidence.

More women in the lifestyle group had met their weight goals one year after giving birth, and lifestyle interventions were associated with a decrease in the risk of depression after birth, from single trials. These findings were supported by low quality evidence.

What does this mean?

Lifestyle interventions provide benefits to women with GDM and their babies. The interventions are useful as the primary therapeutic strategy and generally include, as a minimum, healthy eating, physical activity and self-monitoring of blood sugar levels.

Furture research could focus on the effective components of lifestyle interventions and the use of lifestyle interventions as the sole intervention without pharmacological treatment. Future studies also need to consider long-term outcomes for the mother and her child as a priority when planning future trials.

Background

The original review on *Treatments for gestational diabetes* (Alwan 2009) has been split into three new reviews due to the complexity of the included interventions. The following new review protocols are published.

Lifestyle interventions for the treatment of women with gestational diabetes (this review) (Brown 2015)

Oral anti-diabetic pharmacological therapies for the treatment of women with gestational diabetes (Brown 2015b)

Insulin for the treatment of women with gestational diabetes (Brown 2016)

There will be similarities in the background, methods and outcomes between these three systematic reviews. Portions of the methods section of this protocol are based on a standard template used by the Cochrane Pregnancy and Childbirth Review Group.

Description of the condition

Gestational diabetes mellitus (GDM), often referred to as gestational diabetes can be defined as 'glucose intolerance or hyperglycaemia (high blood glucose concentration) with onset or first recognition during pregnancy' (WHO 1999).
GDM occurs when the body is unable to make enough insulin to meet the extra needs in pregnancy. The high blood sugars associated with GDM will usually return to normal after the birth of the baby. However, there is currently no universally accepted diagnostic criteria (ACOG 2013; Coustan 2010; HAPO 2008; Hoffman 1998; IADPSG 2010; Metzger 1998; NICE 2015). GDM may include previously undetected glucose intolerance (IADPSG 2010; Nankervis 2014; WHO 2013). In an attempt to distinguish women with diabetes mellitus in pregnancy from women with gestational diabetes, WHO 2013 provides separate diagnostic criteria. Some countries such as New Zealand have recommended early

screening in the first trimester using glycated or glycosylated haemoglobin - HbA1C (glycated or glycosylated haemoglobin is a form of haemoglobin measured primarily to identify the average plasma glucose concentration over a period of time), with the aim that more women with overt diabetes will be diagnosed and treated appropriately (Ministry of Health 2014 - New Zealand). It should be noted that this screening is not used globally.

GDM is one of the most common pregnancy complications and the prevalence is rising worldwide with 1% to 36% of pregnancies being affected (Bottalico 2007; Cundy 2014; Duran 2014; Ferrara 2007; Kleinwechter 2014; NICE 2015; Tran 2013). The prevalence of GDM is likely to continue to increase along with the increasing prevalence of maternal obesity and associated type 2 diabetes mellitus (Bottalico 2007; Mulla 2010).

Screening and diagnosis of GDM

There are global variations in screening for GDM with some countries, such as the UK, using an assessment of risk for GDM based on maternal characteristics (NICE 2015), some countries, such as the USA, use either an assessment based on maternal risk factors or a 50 g oral glucose challenge test. In New Zealand all women with an HbA1c value in the normal range at the time of booking are offered the 50 g oral glucose challenge test at 24 to 28 weeks' gestation (Ministry of Health 2014).

Diagnosis of GDM is usually based on either a 75 g two-hour oral glucose tolerance test (OGTT) or a 100 g three-hour OGTT (ADA 2013; IADPSG 2010; Nankervis 2014; NICE 2015; WHO 1999; WHO 2013). Recommendations regarding diagnostic criteria vary nationally and internationally (Table 1), and these diagnostic criteria have changed over time, sometimes due to changing understanding about the effects of hyperglycaemia on pregnancy and infant outcomes (Coustan 2010), but also because of a lack of evidence clearly demonstrating the clinical and cost-effectiveness of one criterion over another.

The Hyperglycaemia and Adverse Pregnancy Outcomes (HAPO) study (HAPO 2008) was a large, international observational study that reported graded linear associations in the odds of several GDM-associated adverse outcomes and glucose concentrations at OGTT, with no clear threshold identified at which risk increased substantially. The International Association of the Diabetes and Pregnancy Study Groups (IADPSG) recommended diagnostic criteria using data from the HAPO study (IADPSG 2010) (Table 1). Applying the IADPSG criteria in most health environments will increase the number of women diagnosed with GDM. A study conducted in Vietnam showed that depending on the criteria used, the diagnosis of GDM varied between 5.9% (American Diabetes Association - ADA), 20.4% (IADPSG), 20.8% (Australasian Diabetes in Pregnancy Society - ADIPS), and up to 24.3% (World Health Organization - WHO) (Tran 2013). A Bulgarian study also reported differences in prevalence based on the different diagnostic criteria used ranging from 10.8% (European Association for the Study of Diabetes - EASD), 13.5% (ADA), 16.2% (New Zealand Society for the Study of Diabetes - NZSSD), 17.1% (WHO), 21.2% (ADIPS), 31.6% (IADPSG) (Boyadzhieva 2012).

Pathophysiology of GDM

Normal pregnancy is associated with significant changes in maternal metabolism (Lain 2007). In early pregnancy, oestrogen and progesterone stimulate maternal beta-cell hyperplasia and insulin secretion, which promotes maternal nutrient storage (adipose and hepatic glycogen) to support later fetal growth. At this stage, insulin sensitivity is maintained or may even increase. However, as pregnancy progresses, whole-body insulin sensitivity steadily decreases, such that by the third trimester it is reduced by almost half (Barbour 2007). Several factors contribute to this, including placental hormones (human placental lactogen and placental growth hormone), cytokines released from adipocytes (e.g. IL-6, TNF-alpha), increased free fatty acids and lower adiponectin concentrations (Clapp 2006; Devlieger 2008). This results in decreased post-prandial peripheral glucose disposal by up to 40% to 60% (Barbour 2007). Because glucose is transported across the placenta to the fetus by facilitated diffusion, this state of physiological insulin resistance promotes fetal glucose uptake, a principal oxidative fuel and carbon source for the growing fetus. In normal pregnancy, maternal glycaemia is maintained by a significant increase in insulin secretion of up to 200% to 250% (Barbour 2007; Lain 2007; Suman Rao 2013).

Women who develop GDM have greater reductions in insulin sensitivity in pregnancy and are unable to increase insulin secretion sufficiently to maintain euglycaemia, especially after meals. Glucose intolerance results from both reduced insulin action in skeletal muscle, leading to decreased peripheral glucose disposal, and in the liver, resulting in inadequate suppression of endogenous glucose production. GDM is associated with impaired insulin signalling, with disruption of several components of the signalling cascade. Subclinical inflammation (TNF-alpha) and decreased secretion of adiponectin from adipocytes contribute to altered insulin signalling in women with GDM (Barbour 2007). The net effect in skeletal muscle is reduced insulin-mediated glucose uptake due to decrease translocation of the glucose transporter GLUT4 to the cell membrane.

In GDM, the steeper maternal-fetal glucose gradient, especially post-prandial, leads to increased fetal glucose uptake, which stimulates fetal insulin secretion. Insulin is a key fetal anabolic hormone and hyperinsulinaemia promotes fetal overgrowth, especially of fat, leading to large-for-gestational age (LGA) infants, macrosomia (larger than average baby), and possible organ damage (Catalano 2003; Ju 2008; Metzger 2008; Reece 2009).

Women with GDM have increased circulating inflammatory cytokines and lower adiponectin concentrations which can lead to insulin resistance in adipose tissue, which in turn results in increased lipolysis and fatty acid concentrations. Placental transfer of free fatty acids contributes to increased fetal adiposity, independent of glucose uptake (Knopp 1985). Thus, even women with well-controlled GDM still have an increased risk of fetal macrosomia (Langer 2005).

Risk factors associated with GDM

A variety of factors have been associated with an increased risk of developing GDM. Non-modifiable risk factors include advanced maternal age (Chamberlain 2013; Morisset 2010), high parity, non-Caucasian race or ethnicity (in particular South Asian, Middle Eastern), family history of diabetes mellitus, maternal high or low birthweight, polycystic ovarian syndrome (Cypryk 2008; Petry 2010; Solomon 1997), a history of having a previous macrosomic infant (birthweight 4000 g or more) and previous history of GDM (Petry 2010).

Modifiable risk factors include physical inactivity (Chasan-Taber 2008), having a low-fibre and high-glycaemic load diet (Zhang 2006), maternal overweight (body mass index (BMI) equal to or greater than 25 kg/m²) or obesity (BMI equal to or greater than 30 kg/m²) (Kim 2010a), and excessive weight gain during pregnancy, especially for those who are already overweight or obese (Hedderson 2010).

Clinical outcomes for women with pregnancy hyperglycaemia

Adverse outcomes have been consistently reported at higher rates in women diagnosed with GDM, and their infants, compared with women without GDM (Crowther 2005; Landon 2009; Metzger 2008; Reece 2009).

Women with GDM have an increased risk of developing pre-eclampsia, are more likely to have their labour induced (Anderberg 2010; Crowther 2005; Ju 2008; Landon 2009; Metzger 2008), and to give birth by caesarean section (Landon 2009; Metzger 2008). The incidence of uterine rupture, shoulder dystocia and perineal lacerations is increased in women with GDM due to the increased likelihood of having a LGA or macrosomic baby (Jastrow 2010). Women who have experienced GDM are at a greater risk of metabolic dysfunction in later life (Shah 2008; Vohr 2008), with a crude cumulative incidence of type 2 diabetes of 10% to 20% within 10 years (Bellamy 2009; Kim 2002), but up to 50% when adjusted for retention and length of follow-up (Kim 2002).

Neonatal, infant and later outcomes related to pregnancy hyperglycaemia

A significant adverse health outcome for babies born to mothers with GDM is being born LGA or macrosomic (Catalano 2003; Crowther 2005; Landon 2009; Metzger 2008; Reece 2009), which increases the risk of birth injury, including shoulder dystocia, perinatal asphyxia, bone fractures and nerve palsies (Esakoff 2009; Henriksen 2008; Langer 2005; Metzger 2008). Other adverse outcomes which are increased for babies born to women with GDM include respiratory distress syndrome, hypoglycaemia (which if prolonged can cause brain injury), hyperbilirubinaemia, hypertrophic cardiomyopathy, hypocalcaemia, hypomagnesaemia, polycythaemia and admission to the neonatal nursery (Metzger 2008; Reece 2009).

Babies born to women with GDM, compared with babies born to women without GDM, have significantly greater skinfold measures and fat mass (Catalano 2003), have greater adiposity (Pettitt 1985; Pettitt 1993), and are more likely to develop early overweight or obesity, type 2 diabetes (Hillier 2007; Pettitt 1993; Whincup 2008), and metabolic syndrome in childhood, adolescence or adulthood. Metabolic syndrome is a cluster of risk factors defined by the occurrence of three of the following: obesity, hypertension, hypertriglyceridaemia and low concentration of high-density lipoprotein (HDL) cholesterol (Guerrero-Romero 2010; Harder 2009).

The development of the metabolic syndrome during childhood is a risk factor for the development of adult type 2 diabetes at 25 to 30 years of age (Morrison 2008). These health problems repeat across generations (Dabelea 2005; Mulla 2010) and are important from a public health perspective, because with each generation the prevalence of diabetes increases.

Description of the intervention

GDM management aims to optimise glycaemic control and consequently improve pregnancy outcomes (Kim 2010b). Providing dietary and lifestyle advice is usually recommended as the primary therapeutic strategy for women with GDM (ACOG 2013; ADA 2015a; Hoffman 1998; NICE 2015). If diet and lifestyle management alone are insufficient to achieve targets for maternal glycaemic control, insulin therapy or oral anti-diabetic pharmacological therapies such as glibenclamide and metformin can be added (ACOG 2013; ADA 2013; Hoffman 1998; NICE 2015; Silva 2010; Simmons 2004). As part of GDM management, maternal glucose monitoring and ultrasonography are advised to monitor the effectiveness of treatment and to guide care for birth (ACOG 2013; Hoffman 1998; NICE 2015). However, treatment recommendations differ across countries, for example, serial ultrasonography is not recommended to guide treatment management in the New Zealand Ministry of Health guidelines (Ministry of Health 2014).

Dietary intervention for managing GDM

Diet therapy is the primary strategy for managing GDM. Elevated blood glucose concentrations, in particular elevations in post-prandial glucose are associated with adverse pregnancy outcomes in GDM (de Veciana 1995). The role of different dietary interventions for treatment of women with GDM, assessed by head-to-head trials, has been described in the Cochrane systematic review by Han 2013 and will not be included in this systematic review.

Carbohydrate-containing foods are important sources of energy, vitamins, minerals and fibre and are the main nutrient affecting blood glucose concentrations (Reader 2007). Blood glucose concentrations are affected by both total amount and type of carbohydrates consumed (Reader 2007). Glycaemic index (GI) is a ranking of the effects of carbohydrates on blood glucose concentrations (Jenkins 1981). Foods with a low GI (less than 55) produce a lower post-prandial glucose elevation and area under the glucose curve; foods with a high GI (more than 70) produce a rapid increase in post-prandial blood glucose concentrations (Jenkins 1981). Outside of pregnancy, consumption of low-GI diets by people with diabetes seems to help lower glycated or glycosylated haemoglobin - HbA1C (Thomas 2010

). Use of low-GI diets in GDM management seems to be beneficial in reducing the need for insulin, though the evidence is limited (Moses 2009). Polyunsaturated fatty acids may be protective against impaired glucose tolerance, while saturated fatty acids can increase glucose and insulin concentrations in women with GDM (Ilic 1999). Reducing blood lipid concentrations may improve glycaemic control and pregnancy outcomes in GDM (Barrett 2014). However, the specific amount and sources of fat that are beneficial for GDM management are not clear (Kim 2010b). Therefore, recommendations on the fat intake for women with GDM have not yet been promulgated (ACOG 2013; Hoffman 1998; Metzger 2007; (New Zealand) Ministry of Health 2014; NICE 2015). Recommendations on the intake of other nutrients for women with GDM are usually based on the general recommendations for people with diabetes mellitus outside pregnancy (Cheung 2009).

Physical activity during pregnancy for managing GDM

The role of supplementary physical activity interventions for the management of glycaemic control in women with diabetes in pregnancy (including GDM) was one of the comparisons described in the Cochrane review by Ceysens 2006. In non-pregnant women with type 2 diabetes, physical activity (in addition to diet and insulin) helped to normalise blood glucose levels (Tuomilehto 2001). Caution is required when generalising this evidence to pregnant women, but it potentially suggests that during pregnancy mild exercise could reduce the risk of complications related to high blood glucose and high insulin levels, including macrosomia, birth trauma, respiratory distress, neonatal hypoglycaemia and hypocalcaemia. Exercise interventions alone for treating women with gestational diabetes will not be included in this systematic review.

Appropriate weight gain during pregnancy

Interventions for preventing excessive weight gain in pregnancy (diet or exercise or both) have been described in the Cochrane systematic review by Muktabhant 2015, which included 65 randomised controlled trials, of which seven recruited women who were at high risk of gestational diabetes. Given the high prevalence of overweight and obesity in women with GDM, dietary interventions for appropriate pregnancy weight gain are routinely included as a part of nutritional management of GDM (Kim 2010b). Small reductions in weight improve glycaemic control (ACOG 2005), but the implications in pregnancy for the mother and fetus are unclear.

In 2009, the American Institute of Medicine updated their guidelines for weight gain during pregnancy. Guidance is stratified by pre-pregnancy BMI, i.e. women with a pre-pregnancy BMI between 25 kg/m² and 29.9 kg/m² should aim for 6.8 kg to 11.4 kg weight gain and those with pre-pregnancy BMI of 30 kg/m² or more should aim for 5 kg to 9 kg weight gain (IOM 2009). However, the degree of energy restriction for pre-pregnancy overweight and obese women to achieve these weight gain goals is unknown and based on observational data (Kim 2010b).

Dietary interventions provided for women with GDM should ensure adequate nutrients for normal fetal growth and maternal health, but not induce weight loss or excessive weight gain during pregnancy; the main aim however is to promote optimal glycaemic control (ACOG 2013; Hoffman 1998; Metzger 2007; NICE 2015).

Combined dietary intervention and physical activity during pregnancy for managing GDM

Some interventions may involve a combination of dietary and physical activity modalities. Regular physical activity may help normalise maternal blood glucose for pregnant women with gestational diabetes and in combination with dietary interventions may reduce the need for oral anti-diabetic agents or insulin. As women with gestational diabetes are at increased risk of developing type 2 diabetes in the future, regular physical activity may also help reduce the risk of this long-term complications (Tuomilehto 2011).

Other interventions during pregnancy for managing GDM

There may be other interventions, including psychological approaches that could be used independently or alongside physical activity or dietary modalities such as mindfulness eating, yoga or spiritual support.

How the intervention might work

Role of diet

A carbohydrate-controlled diet (with carbohydrates distributed evenly throughout the day) that provides adequate nutrition, alongside glycaemic control and avoids ketonuria (ketones are produced when stored fat is utilised to produce energy in the absence of glucose) is thought to be optimal to reduce complications associated with gestational diabetes (Dornhorst 2002). Other elements of diet such as fat and fibre are also thought to influence maternal blood glucose concentrations (Zhang 2006). Excess fetal growth is most effectively limited by normal post-prandial maternal glucose concentrations (de Veciana 1995; Dornhorst 2002; Harmon 2011; Rowan 2011; Weisz 2005). Dietary advice in the second trimester, when insulin resistance is increasing, may help reduce the risk of adverse outcomes associated with GDM (Dornhorst 2002).

Role of physical activity

Insulin sensitivity in skeletal muscle is related to the degree of physical activity, and therefore, physical activity interventions may improve insulin sensitivity and glucose control in individuals with diabetes (Asano 2014).

Glucose enters skeletal muscle cells via facilitated diffusion through a glucose transporter (GLUT4). Peripheral clearance of glucose in skeletal muscle depends on blood flow to muscle, expression of GLUT4 transporters and intracellular utilisation of glucose through glycolysis and glycogenesis. Translocation of the GLUT4 transporter is induced by insulin and insulin-independent mechanisms (Richter 2001). Exercise increases glucose uptake in skeletal

muscle (Asano 2014), and improves glucose homeostasis and insulin sensitivity in skeletal muscle. Exercise potentiates most of the insulin-mediated post-receptor events that lead to an increased expression of GLUT4, and GLUT4 translocation from intracellular stores to the muscle membrane. These exercise-induced improvements in glucose uptake, however, are not limited to changes in GLUT4 expression. The improvements in insulin sensitivity after regular exercise may be related to changes in expression and/or activity of proteins involved in insulin signal transduction in skeletal muscle. As such, the enhanced glucose uptake in skeletal muscle attributed to exercise might be related to an increased expression and activity of key proteins for insulin signalling such as insulin receptor, insulin receptor substrate, and phosphatidylinositol 3-kinase (Chibalin 2000; Dela 1993; Hjeltnes 1998). Physical activity improves blood supply to the active skeletal muscles (Jensen 2004), counteracts the ability of lipids to induce insulin resistance (Schenk 2005), and modifies the hormonal regulation of hepatic glucose output. These exercise-induced alterations in muscle glucose handling explain most of the insulin-sensitising and diabetes-preventing effects of exercise, and partly explain why the many defects of insulin action observed in type 2 diabetes and insulin resistance are reversed by the effects of exercise (Zierath 2002).

Self-monitoring of blood glucose

Self-monitoring of blood glucose is performed by most women with GDM. Evidence has suggested that self-monitoring between four and seven times per day (including fasting and post-prandial measurements) can contribute to improved maternal and perinatal outcomes (ADA 2015a), and is likely to be most effective when combined with effective treatment.

Why it is important to do this review

GDM affects a significant proportion of pregnant women and the prevalence is increasing worldwide (Bottalico 2007; Dabelea 2005; Mulla 2010). GDM is associated with an increased risk of a range of adverse pregnancy outcomes and these adverse health outcomes repeat across generations (Metzger 2008; Mulla 2010), which has important implications for the future. Providing dietary and lifestyle advice is usually recommended as the primary therapeutic strategy for women with GDM (ACOG 2013; Hoffman 1998; (New Zealand) Ministry of Health 2014; NICE 2015).

Two other Cochrane reviews cover comparisons of individual lifestyle components (diet and exercise) '*Different types of dietary advice for women with gestational diabetes mellitus*' (Han 2013). This review examined the effects of two or more modalities of dietary interventions compared with each other for treating women with GDM, i.e. standard dietary advice compared with individualised dietary advice, individual dietary education sessions compared with group dietary education sessions, single dietary counselling session compared with multiple dietary counselling sessions. '*Exercise for diabetic pregnant women*' (Ceysens 2006). This review evaluated the effects of physical activity interventions with or without dietary interventions compared with no additional physical activity intervention for women with diabetes in pregnancy and the trials included in the review recruited women with gestational diabetes.

This review focuses on the potential effectiveness of multi-component lifestyle interventions.

Objectives

To evaluate the effects of combined lifestyle interventions with or without pharmacotherapy in treating women with gestational diabetes.

Methods

Criteria for considering studies for this review

Types of studies

We included published or unpublished randomised controlled trials in full text or abstract format. If identified, we planned to include cluster-randomised trials. Quasi-randomised trials were excluded. Cross-over trials were not eligible for inclusion. Conference abstracts were handled in the same way as full-text publications.

Types of participants

Participants were pregnant women diagnosed with gestational diabetes (diagnosis as defined by the individual trial). Women with known type 1 or type 2 diabetes were excluded.

Types of interventions

We included randomised trials comparing lifestyle interventions (as defined by trialists) with:

- expectant management, standard care;
- other lifestyle intervention or combination of lifestyle interventions not described below.

The aim of the interventions was to maintain maternal glycaemic targets during pregnancy in women with gestational diabetes.

Lifestyle interventions could include a combination of at least two or more of the following interventions:

- diet;
- physical activity;
- education;
- behavioural change techniques;
- regimens of self-monitoring of blood glucose;
- other intervention not previously specified.

These interventions may or may not require adjunctive pharmacotherapy (oral anti-diabetic pharmacological therapies, insulin) used to treat women with gestational diabetes.

Interventions examining the comparison of different dietary interventions or the effects of exercise alone are not be included in this review as they are already included in other Cochrane systematic reviews (Han 2013 and Ceysens 2006, respectively).

Types of outcome measures

The following standardised outcomes have been developed through a process involving authors of Cochrane reviews for treatment interventions for women with gestational diabetes mellitus (GDM).

Primary outcomes

Maternal

- Hypertensive disorders of pregnancy (including pre-eclampsia, pregnancy-induced hypertension, eclampsia as defined by trialists)
- Caesarean section
- Development of type 2 diabetes

Neonatal

- Perinatal (fetal and neonatal death) and later infant mortality
- Large-for-gestational age (LGA) (as defined by trialists)
- Death or serious morbidity composite (variously defined by trials, e.g. perinatal or infant death, shoulder dystocia, bone fracture or nerve palsy)
- Neurosensory disability in later childhood (as defined by trialists)

Secondary outcomes

Maternal

- Use of additional pharmacotherapy
- Maternal hypoglycaemia (as defined by trialists)
- Glycaemic control during/end of treatment (as defined by trialists)
- Weight gain in pregnancy
- Adherence to the intervention
- Induction of labour
- Placental abruption
- Postpartum haemorrhage (as defined by trialists)
- Postpartum infection
- Perineal trauma/tear
- Breastfeeding at discharge, six weeks postpartum, six months or longer
- Maternal mortality
- Sense of well-being and quality of life
- Behavioural changes associated with the intervention
- Views of the intervention
- Relevant biomarker changes associated with the intervention (including adiponectin, free fatty acids, triglycerides, high-density lipoproteins (HDL), low-density lipoproteins (LDL), insulin)

Long-term outcomes for mother

- Postnatal depression
- Body mass index (BMI)
- Postnatal weight retention or return to pre-pregnancy weight
- Type 1 diabetes
- Impaired glucose tolerance
- Subsequent gestational diabetes
- Cardiovascular health (as defined by trialists including blood pressure, hypertension, cardiovascular disease, metabolic syndrome)

Fetal/neonatal outcomes

- Stillbirth
- Neonatal death
- Macrosomia (greater than 4000 g; or as defined by individual study)
- Small-for-gestational (SGA) age (as defined by trialists)
- Birth trauma (shoulder dystocia, bone fracture, nerve palsy)
- Gestational age at birth
- Preterm birth (< 37 weeks' gestation; and < 32 weeks' gestation)
- Five-minute Apgar less than seven
- Birthweight and z score
- Head circumference and z score
- Length and z score

- Ponderal index
- Adiposity (including skinfold thickness measurements (mm); fat mass as defined by trialists)
- Neonatal hypoglycaemia (as defined by trialists)
- Respiratory distress syndrome
- Neonatal jaundice (hyperbilirubinaemia) (as defined by trialists)
- Hypocalcaemia (as defined by trialists)
- Polycythaemia (as defined by trialists)
- Relevant biomarker changes associated with the intervention (including insulin, cord c-peptide)

Later infant/childhood outcomes

- Weight and z score
- Height and z score
- Head circumference and z score
- Adiposity (including BMI, skinfold thickness, fat mass)
- Educational attainment
- Blood pressure
- Type 1 diabetes
- Type 2 diabetes
- Impaired glucose tolerance
- Dyslipidaemia or metabolic syndrome

Child as an adult outcomes

- Weight
- Height
- Adiposity (including BMI, skinfold thickness, fat mass)
- Employment, education and social status/achievement
- Dyslipidaemia or metabolic syndrome
- Type 1 diabetes
- Type 2 diabetes
- Impaired glucose tolerance
- Cardiovascular health (as defined by trialists including blood pressure, hypertension, cardiovascular disease, metabolic syndrome)

Health service use

- Number of antenatal visits or admissions
- Number of hospital or health professional visits (including midwife, obstetrician, physician, dietician, diabetic nurse)
- Admission to neonatal intensive care unit/nursery
- Duration of stay in neonatal intensive care unit or special care baby unit
- Length of antenatal stay
- Length of postnatal stay (maternal)
- Length of postnatal stay (baby)
- Cost of maternal care
- Cost of offspring care
- Costs associated with the intervention
- Costs to families associated with the management provided
- Cost of dietary monitoring (e.g. diet journals, dietician, nurse visits, etc)
- Costs to families - change of diet, extra antenatal visits
- Extra use of healthcare services (consultations, blood glucose monitoring, length and number of antenatal visits)
- Women's view of treatment advice

Search methods for identification of studies

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

Electronic searches

We searched Cochrane Pregnancy and Childbirth's Trials Register by contacting their Information Specialist (14 May 2016).

The Register is a database containing over 23,000 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).

In addition, we searched ClinicalTrials.gov and the WHO International Clinical Trials Registry Platform (ICTRP) (14 May 2016) for unpublished, planned and ongoing trial reports. The search terms we used are given in (Appendix 1).

Searching other resources

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

Data collection and analysis

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

Selection of studies

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

We created a study flow diagram to map out the number of records identified, included and excluded.

Data extraction and management

We designed a form to extract data. For eligible studies, two review authors extracted the data using the agreed form. We resolved discrepancies through discussion or, if required, we consulted a third person. We entered data into Review Manager software (RevMan 2014) and checked for accuracy. When information regarding any of the above was unclear, we attempted to contact 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 randomised study using the criteria outlined in the *Cochrane Handbook for Systematic Reviews of Interventions* (Higgins 2011). We resolved any disagreement by discussion or by involving a third assessor. For cluster-randomised trials, we planned to refer to the *Handbook* sections 16.3.2 and 16.4.3 for assessing bias. No cluster-randomised trials were identified in this version of the review.

(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 will assess 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 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 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 trial authors, we re-included 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 had been reported);
- high risk of bias (where not all the study's pre-specified outcomes had been reported; one or more reported primary outcomes were not pre-specified; outcomes of interest were reported incompletely and so could 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 (5) above)

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

We assessed whether each study was free of other problems that could put it at risk of bias:

- low risk of other bias;
- high risk of other bias;
- unclear whether there is risk of other 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

We assessed the quality of the evidence using the GRADE approach as outlined in the GRADE handbook in order to assess the quality of the body of evidence relating to the following outcomes. We selected up to a maximum of seven outcomes for the mother and seven for the infant covering both short- and long-term outcomes for the main comparisons.

Maternal outcomes

- Hypertensive disorders of pregnancy (including pre-eclampsia, pregnancy-induced hypertension, eclampsia)
- Perineal trauma/tear
- Induction of labour
- Caesarean section
- Postnatal depression
- Return to pre-pregnancy weight
- Development of type 2 diabetes

Neonatal/child/adult outcomes

- LGA (neonatal)
- Perinatal mortality (neonatal)
- Death or serious morbidity composite (variously defined by trials, e.g. perinatal or infant death, shoulder dystocia, bone fracture or nerve palsy) (neonatal)
- Neonatal hypoglycaemia (neonatal)
- Adiposity (neonatal, child, adult)
- Diabetes (type 2) (child or adult)

- Neurosensory disability (child, adult)

We used the GRADEpro Guideline Development Tool to import data from Review Manager 5.3 (RevMan 2014) in order to create 'Summary of findings' tables. A summary of the intervention effect and a measure of quality for each of the above outcomes was produced using the GRADE approach. 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 each outcome. 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, serious inconsistency, imprecision of effect estimates or potential publication bias.

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 if outcomes are measured in the same way between trials. 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

No cluster-randomised trials were identified in this version of the review. In future updates, if identified, we will include cluster-randomised trials in the analyses along with individually-randomised trials. We will make adjustments using the methods described in the *Handbook* [Section 16.3.4 or 16.3.6] using an estimate of the intra-cluster correlation co-efficient (ICC) derived from the trial (if possible), from a similar trial or from a study of a similar population. If we use ICCs from other sources, we will report this and conduct sensitivity analyses to investigate the effect of variation in the ICC. We will consider it reasonable to combine the results from both cluster-randomised trials and individually-randomised trials 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. If cluster-randomised trials are included, we will seek statistical advice on appropriate analysis to enable inclusion of data in the meta-analyses.

Other unit of analysis issues

Multiple pregnancy

We presented maternal data as per woman randomised and neonatal data per infant.

Multiple-arm studies

If in future versions of the review a trial has multiple intervention arms we will avoid 'double counting' of participants by combining groups to create a single pair-wise comparison if possible. Where this is not possible, we will split the 'shared' group into two or more groups with smaller sample size and include two or more (reasonably independent) comparisons.

Dealing with missing data

For included studies, we noted levels of attrition. We planned to explore the impact of including studies with high levels of missing data (> 20%) in the overall assessment of treatment effect by using sensitivity analysis.

For all outcomes, we carried out analyses, 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, and all participants were analysed in the group to which they were allocated, regardless of whether or not they received the allocated intervention. The denominator for each outcome in each trial was the number randomised minus any participants whose outcomes are known to be missing.

Assessment of heterogeneity

We assessed statistical heterogeneity in each meta-analysis using the Tau², I² and Chi² statistics. We regarded heterogeneity as substantial if an I² was greater than 30% and either a Tau² was greater than zero, or there was a low P value (less than 0.10) in the Chi² test for heterogeneity.

Assessment of reporting biases

Where there were 10 or more studies in the meta-analysis, we investigated reporting biases (such as publication bias) using funnel plots. We assessed funnel plot asymmetry visually. If asymmetry was suggested by a visual assessment, we performed 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 trials examined the same intervention, and the trials' populations and methods were judged sufficiently similar. If clinical heterogeneity was sufficient to expect that the underlying treatment effects differed between trials, or if 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 was treated as the average of the range of possible treatment effects and we 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 were presented as the average treatment effect with 95% confidence intervals, and the estimates of Tau² and I².

Subgroup analysis and investigation of heterogeneity

If we identified substantial heterogeneity, we investigated it using subgroup analyses and sensitivity analyses. We considered whether an overall summary was meaningful, and if it was, used random-effects analysis to produce it.

- Diagnostic test used: ADA 2013, IADPSG 2010, Nankervis 2014 versus ACOG 2013 versus NICE 2015 versus NICE 2008; WHO 1999; WHO 2013 or Hoffman 1998 versus New Zealand Ministry of Health 2014 versus other not previously specified
- Timing of diagnosis: early (< 28 weeks' gestation) versus late (≥ 28 weeks' gestation)

The following outcomes were used in subgroup analysis.

Maternal outcomes

- Pre-eclampsia
- Caesarean section
- Development of type 2 diabetes

Neonatal outcomes

- LGA
- Perinatal mortality
- Death or morbidity composite (variously defined by trials, e.g. infant death, shoulder dystocia, bone fracture or nerve palsy)
- Neurosensory disability in later childhood (as defined by trialists)

We assessed subgroup differences by interaction tests available within RevMan (RevMan 2014). We reported the results of subgroup analyses quoting the Chi^2 statistic and P value, and the interaction test I² value.

Sensitivity analysis

If there was evidence of significant heterogeneity, we explored this by using the quality of the included trials for the primary outcomes. We compared trials that have low risk of bias for allocation concealment with those judged to be of unclear or high risk of bias, and conference abstracts were excluded from the meta-analysis.

We planned to investigate the effect of the randomisation unit (i.e. if we had included cluster-randomised trials along with individually-randomised trials).

Results

Description of studies

Results of the search

We assessed 253 abstracts from the electronic search and an additional 21 potential studies from other sources. Two hundred and one of these abstracts were excluded due to lack of relevance and 73 publications were viewed in full-text format. Of these, 23 studies (28 reports) were excluded. Twenty-five studies (45 reports) were included in the qualitative synthesis (Figure 1).

Three studies are awaiting classification as methodological queries need to be answered or translations into English are required. Where necessary authors have been contacted (Cao 2012; Kaveh 2012; Zhang 2012), See Characteristics of studies awaiting classification.

There are seven studies that are currently ongoing and would appear to meet the inclusion criteria for the review (See Characteristics of ongoing studies). For the next update of this review we will see if any data from these trials have been published.

Included studies

Forty-five publications associated with 15 trials are included in this review (Bancroft 2000; Bo 2014; Crowther 2005; Elnour 2008; Ferrara 2011; Garner 1997; Gillen 2004; Jovanovic-Peterson 1989; Kaviani 2014; Landon 2009; Mendelson 2008; Rahimikian 2014; Yang 2003; Yang 2014; Youngwanichsetha 2014). The 15 trials included a total of 4501 women and 3768 infants. Four trials did not report any neonatal data (Kaviani 2014; Rahimikian 2014; Yang 2003; Youngwanichsetha 2014).

Design

All of the included studies used a parallel design in a randomised controlled trial.

Sample sizes

Sample sizes ranged from 19 (Jovanovic-Peterson 1989) to 1000 (Crowther 2005) women. Twelve studies had a sample size of 300 women or fewer (Bancroft 2000; Bo 2014; Elnour 2008; Ferrara 2011; Garner 1997; Gillen 2004; Jovanovic-Peterson 1989; Kaviani 2014; Mendelson 2008; Rahimikian 2014; Yang 2003; Youngwanichsetha 2014).

Settings

Four studies were conducted in the USA (Ferrara 2011; Landon 2009; Mendelson 2008; Jovanovic-Peterson 1989), two in China (Yang 2003; Yang 2014), two in Iran (Kaviani 2014; Rahimikian 2014), two in Canada (Garner 1997; Gillen 2004), one each in the UK (Bancroft 2000), Italy (Bo 2014), United Arab Emirates (Elnour 2008), Thailand (Youngwanichsetha 2014), and one in Australia and the UK (Crowther 2005).

Population

Eleven trials reported data for maternal age (see Table 2. In the intervention groups the mean age ranged from a minimum of 29.2 ± 5.7 years (Landon 2009) to maximum of 35.9 ± 4.8 years (Bo 2014). In the control groups, the mean age ranged from a minimum of 28.9 ± 5.6 (Landon 2009) to 33.9 ± 5.3 years (Bo 2014). Details on maternal BMI (kg/m^2) at trial entry, reported in seven trials and ethnicity reported in nine of 15 trials are summarised in Table 3 and Table 4, respectively. Gestational age at trial entry and treatment targets are described in Table 5 and Table 6, respectively.

Details of diagnostic criteria used

Criteria used to diagnose the women with gestational diabetes were variable. Six different diagnostic criteria were used in the nine trials that provided details (Table 7).

- World Health Organization (1999) Bancroft 2000; Crowther 2005; Yang 2003
- Carpenter and Coustan criteria Elnour 2008; Landon 2009
- American Diabetes Association (2000) Ferrara 2011
- ADIPS (Hofman 1998) Gillen 2004
- IADPSG criteria Yang 2014
- Hatem (1988) 75 g OGTT > 7.5 mmol (second trimester) and > 9.6 mmol/L (third trimester) (no other details) (Garner 1997)

Six trials did not provide details on the criteria used to diagnose the women with gestational diabetes (Bo 2014; Jovanovic-Peterson 1989; Kaviani 2014; Mendelson 2008; Rahimikian 2014; Youngwanichsetha 2014).

Interventions

The types of interventions used varied, as can be seen below.

Bancroft 2000: Intensive intervention (standard dietary advice, glucose monitoring five days a week, HbA1c monthly, serial ultrasound, Doppler studies, cardiotocography (CTG monitoring) compared with usual care (dietary advice, HbA1c monthly).

Bo 2014: Reported on a multiple-arm trial that included a) Individualised-dietary advice alone, b) Exercise alone, c) Behavioural intervention and d) Behavioural intervention and exercise. We used the combined behavioural and exercise group as the intervention arm for this review and the Individualised-dietary advice alone as the control group.

Crowther 2005: Intensive intervention (individualised-dietary advice, advice on self-monitoring of blood glucose) compared with usual care (women and caregivers unaware of diagnosis).

Elnour 2008: Intensive intervention (structured pharmaceutical care, structured education, self-monitoring of blood glucose) compared with usual care (no additional education or pharmacist counselling).

Ferrara 2011: Intensive intervention (individualised advice on diet, exercise and breastfeeding) compared with usual care (printed material only in prenatal and postnatal period).

Garner 1997: Intensive intervention (dietary counselling, self-glucose monitoring, biweekly review, monitoring of fetal growth, amniotic volume and cardiac size) versus usual care (no dietary counselling).

Gillen 2004: Group session on education and diet followed by specific dietary advice compared with group session on education and diet followed by standard clinical care and advice.

Jovanovic-Peterson 1989: Diet alone compared with diet plus supervised exercise.

Kaviani 2014: Relaxation training (education, breathing, muscle relaxation, mental imagery, and contacted by telephone by the researcher three times per week) compared with usual care (no details).

Landon 2009: Nutritional counselling and diet therapy +/- insulin plus self-monitoring of blood glucose compared with usual care +/- insulin plus self-monitoring of blood glucose.

Mendelson 2008: Intensive education and spiritual intervention compared with standard education.

Rahimikian 2014: Face-to-face education (risks of GDM, training on glycaemic control, exercise, diet, medication and follow-up) compared with usual care (no details).

Yang 2003: Intensive intervention (including diet and exercise advice, self-monitoring of blood glucose, insulin if required, fortnightly specialist review) versus usual care (no details).

Yang 2014: Shared care protocol adapted from Crowther 2005. Individualised and group dietary and physical activity counselling, self-monitoring blood glucose compared with usual care (group education on exercise and physical activity, not specifically taught blood glucose self-monitoring).

Youngwanichsetha 2014: Mindfulness eating and yoga compared with standard diabetes care (no details).

Outcomes

For the maternal primary outcomes: pregnancy-induced hypertension was reported in four trials (Crowther 2005; Elnour 2008; Landon 2009; Yang 2014), and caesarean section in 10 trials (Bancroft 2000; Bo 2014; Crowther 2005; Elnour 2008; Garner 1997; Gillen 2004; Landon 2009; Mendelson 2008; Yang 2003; Yang 2014). Development of type 2 diabetes was reported in two trials (Bancroft 2000; Landon 2009).

For the neonatal primary outcomes: Perinatal death was reported in two trials (Crowther 2005; Landon 2009); large-for-gestational age (LGA) in six trials (Bancroft 2000; Bo 2014; Crowther 2005; Elnour 2008; Landon 2009; Yang 2014), and a composite of serious neonatal outcomes in two trials (Crowther 2005; Landon 2009). Neurosensory disability in later childhood was not a pre-specified outcome, nor reported for any of the included trials.

Data were available for the following maternal secondary outcomes: need for supplementary medication, maternal hypoglycaemia, fasting plasma glucose concentration, postprandial glucose concentration, HbA1c, weight gain in pregnancy, induction of labour, postpartum haemorrhage, postnatal infection/pyrexia, perineal trauma/tear, breastfeeding, postnatal depression, quality of life, impaired glucose tolerance, metabolic syndrome and return to prepregnancy weight.

Data were available for the following neonatal secondary outcomes: stillbirth, neonatal death, macrosomia, small-for-gestational age (SGA), birth trauma (shoulder dystocia, bone fracture, nerve palsy), gestational age at birth, preterm birth, congenital anomaly, five-minute Apgar less than seven, birthweight, length, neonatal fat mass, neonatal hypoglycaemia, respiratory distress syndrome, hyperbilirubinaemia, hypocalcaemia, polycythaemia, childhood growth, childhood cholesterol and childhood impaired glucose tolerance.

Data were available for the following health service outcomes: visits to health professionals, antenatal hospital admissions and admission to neonatal intensive care unit.

Funding sources

Funding sources were reported in seven trials (Bo 2014; Crowther 2005; Ferrara 2011; Kaviani 2014; Landon 2009; Mendelson 2008; Yang 2003). None of the sources were conditional grants from pharmaceutical companies. PLEASE STATE WHICH TRIALS DID NOT REPORT SOURCES OF FUNDING (WITH LINKS TO TRIALS).

Declarations of interest

Declarations of conflicts of interest were made in four trials (Bo 2014; Ferrara 2011; Landon 2009; Yang 2003). Three reported that there were no conflicts of interest for any of the authors (Bo 2014; Ferrara 2011; Landon 2009). One trial (Yang 2003) reported that there was a conflict of interest for one of the 12 authors. please add text to the relevant section of Yang 2003's characteristics of included studies to clarify what the conflict of interest was). PLEASE ALSO LIST THE TRIALS THAT DID NOT MENTION DECLARATIONS OF INTEREST (WITH LINKS TO THOSE TRIALS).

Excluded studies

Twenty-three trials (28 reports) were excluded. Six studies were not randomised (Abirami 2014; O'Sullivan 1980; Reader 2006) or were quasi-randomised trials (O'Sullivan 1971; O'Sullivan 1974; Perichart-Perera 2009).

Twelve trials did not use an intervention/comparison included in this review (Adam 2014; Bastani 2015; Berry 2013; Fadl 2015; Ford 1997; Grant 2011; Homko 2002; Holmes 2012; Langer 1989; Li 1987; Mirzamoradi 2015; Rey 1997).

Three trials included women not meeting the diagnosis of gestational diabetes and representing the wrong population for this review (Bevier 1999; Bonomo 2005; Osmundson 2015).

One trial, although registered, never started recruitment due to insufficient funding (Branch 2010), and a second trial did not start recruitment, although no reason could be found (Kitzmiller 1990).

Risk of bias in included studies

Refer to Figure 2; Figure 3.

Allocation (selection bias)

Randomisation - 10 of the trials were considered to be of low risk of bias for randomisation (Bancroft 2000; Crowther 2005; Elnour 2008; Ferrara 2011; Gillen 2004; Jovanovic-Peterson 1989; Kaviani 2014; Landon 2009; Mendelson 2008; Yang 2014). Seven of these trials (Bancroft 2000; Crowther 2005; Ferrara 2011; Gillen 2004; Kaviani 2014; Mendelson 2008; Yang 2014) used computer-generated randomisation. Elnour 2008 used a restricted randomisation method; Jovanovic-Peterson 1989 randomised by drawing a number; and Landon 2009 used a simple urn method. Method of randomisation was judged as unclear in five trials due to lack of sufficient details (Bo 2014; Garner 1997; Rahimikian 2014; Yang 2003; Youngwanichsetha 2014).

Allocation concealment - five trials were considered to be of low risk of bias for allocation concealment (Bancroft 2000; Bo 2014; Crowther 2005; Gillen 2004; Landon 2009). Bancroft 2000 used a telephone randomisation service that was controlled by a trial centre and Bo 2014 used a website (third person); Crowther 2005; Gillen 2004; Landon 2009 performed randomisation centrally. Allocation concealment was judged as unclear in 10 trials due to lack of sufficient details (Elnour 2008; Ferrara 2011; Garner 1997; Jovanovic-Peterson 1989; Kaviani 2014; Mendelson 2008; Rahimikian 2014; Yang 2003; Yang 2014; Youngwanichsetha 2014).

Blinding (performance bias and detection bias)

Performance bias

Four trials were judged to be of low risk of bias. Bancroft 2000 reported that the obstetrician was blinded to randomisation. Yang 2014 reported that the women with GDM were masked to the allocation although the research staff were not. Crowther 2005 reported women and caregivers were unaware of diagnosis in the control group and Garner 1997 reported that healthcare workers in the control group were blinded to allocation.

In two trials the risk of bias was judged to be unclear: in Gillen 2004 participants were unaware of differences in advice between the intervention and control groups but the researchers were aware and in Mendelson 2008 the women were not blinded to allocation but the diabetes educators were blinded to allocation (personal communication). Nine trials were judged to be of high risk of bias, including six trials (Bo 2014; Jovanovic-Peterson 1989; Kaviani 2014; Rahimikian 2014; Yang 2003; Youngwanichsetha 2014) that provided no details of blinding for participants or researchers. Due to the types of interventions blinding is unlikely. Three trials clearly stated that the researchers and participants were not blinded (Elnour 2008; Ferrara 2011; Landon 2009).

Detection bias

Six trials were considered to be of low risk of detection bias. Bo 2014 reported that dieticians and obstetricians who reported maternal/neonatal complications and laboratory personnel were blinded to allocation. Elnour 2008 reported that nursing and pharmacy staff who assisted with questionnaire administration were blinded to allocation. Ferrara 2011 provided details in the trials registration document that outcome assessors were blinded. Yang 2014 reported that outcome assessors for pregnancy-induced hypertension were blinded to allocation and Youngwanichsetha 2014 reported that HbA1c testing was conducted in a laboratory and the personnel there are likely to have been blinded (no further details). Landon 2009 reported that outcome assessors were blinded for some relevant outcomes (no details).

Nine trials (Bancroft 2000; Crowther 2005; Garner 1997; Gillen 2004; Jovanovic-Peterson 1989; Kaviani 2014; Mendelson 2008; Rahimikian 2014; Yang 2003) provided no details of blinding of outcome assessors and were judged as having an unclear risk of bias.

Incomplete outcome data (attrition bias)

Ten trials were judged to be of low risk for attrition bias. Bo 2014; Crowther 2005; Jovanovic-Peterson 1989; Kaviani 2014 reported that all women who were randomised were analysed or that there were no losses to follow-up. Attrition of less than 10% was reported by Elnour 2008 (9%); Ferrara 2011 (4%); Garner 1997 (< 1%); Gillen 2004 (6%); Landon 2009 (6%) and Youngwanichsetha 2014 (6%). The Crowther 2005 trial, although reporting low attrition levels for clinical data, reported that only 68% of women provided data for maternal health status.

Two trials were judged to be of unclear risk of bias. In the Rahimikian 2014 trial, data appear to be missing for one of the intervention groups but no reasons are provided. Bancroft 2000 reported that 18% of women failed to return for postnatal measurements.

Three trials were judged to be at high risk for attrition bias. Mendelson 2008 reported that only 27% (27/100) of women had an HbA1c value recorded at birth; there is no explanation as to why the remaining women did not have results. Yang 2003 reported that only 51% (48/95) of women in the intervention group completed the management plan. Yang 2014 reported that due to construction work in the building where the intervention took place during the trial, 242 women did not receive the intended intervention and they excluded these women from the analysis.

Selective reporting (reporting bias)

Three trials were judged to be of low risk for reporting bias (Crowther 2005; Elnour 2008; Landon 2009).

One trial was judged to be of unclear risk for reporting bias. Mendelson 2008 reported one additional outcome of caesarean section that was not prespecified in the methods section; all of the other outcomes listed a priori were reported.

Eleven trials were judged to be of high risk for reporting bias. Bancroft 2000; Bo 2014; Jovanovic-Peterson 1989 and Yang 2014 reported additional outcomes in the results section that were not prespecified in the methods. Ferrara 2011 reported data for a pilot study and the full trial is yet to be reported on; the primary trial outcome was postpartum weight gain and there were very limited neonatal outcomes. Garner 1997 did not pre-specify any outcomes; Gillen 2004 did not clearly pre-specify trial outcomes, the trial authors report no differences in pregnancy outcomes or mode of birth but these data are not reported in the paper. Kaviani 2014 reported very limited maternal outcome and no neonatal outcomes were reported. Rahimikian 2014 did not provide any numeric data for any of the specified trial outcomes. Yang 2003 reported the trial as a letter and only data for caesarean section and rupture of membranes were reported. Youngwanichsetha 2014 reported on the effects of glycaemic control but no other neonatal or maternal outcomes were reported.

Other potential sources of bias

Two trials were judged to be at high risk of other bias. Yang 2003 published findings in a letter and we were unable to find a full publication. The sample size was estimated at 200 but only 100 women were randomised. Jovanovic-Peterson 1989 reported that the women in the exercise plus diet group had a significantly higher one-hour plasma glucose in the diagnostic test at baseline, the treatment and control groups are therefore not balanced for an important baseline prognostic variable.

There was no evidence of other risk of bias reported by Bancroft 2000; Bo 2014; Crowther 2005; Elnour 2008; Ferrara 2011; Garner 1997; Gillen 2004; Kaviani 2014; Landon 2009; Mendelson 2008; Rahimikian 2014; Yang 2014; Youngwanichsetha 2014. These studies were judged to be of low risk of other potential sources of bias.

Effects of interventions

1.0 Lifestyle intervention versus usual care or control

Maternal primary outcomes

1.1 Hypertensive disorders of pregnancy (including pre-eclampsia, pregnancy-induced hypertension, eclampsia)

There was no evidence of a difference between lifestyle intervention and control groups for *risk of pre-eclampsia* (average risk ratio (RR) 0.70; 95% confidence interval (CI) 0.40 to 1.22; four trials, 2796 women; I^2 = 79%, Tau^2 = 0.23; *low-quality of evidence*) (Analysis 1.1). The evidence was downgraded (-2) for inconsistency.

We explored the heterogeneity by looking at the diagnostic criteria used and the definitions used in the individual trials. Three different diagnostic criteria were used by studies reporting data for pregnancy-induced hypertension: (i) World Health Organization - WHO (1999) Crowther 2005; (ii) American Diabetes Association - ADA (2013) Elnour 2008; Landon 2009 and (iii) International Association for the Study of Diabetes in Pregnancy - IADPSG (2010) Yang 2014. Subgroup analysis identified a significant differential effect on the risk for pre-eclampsia based on diagnostic criteria (Chi^2 = 9.94, df = 2, P = 0.007, I^2 = 79.9%). Interpretation of these data remains unclear due to the limited number of trials reporting data for each diagnostic criteria.

1.2 Caesarean section

Caesarean section was reported in 10 trials (Bancroft 2000; Bo 2014; Crowther 2005; Elnour 2008; Garner 1997; Gillen 2004; Landon 2009; Mendelson 2008; Yang 2003; Yang 2014). There was no evidence of a difference between lifestyle intervention and control groups for *risk of birth by caesarean section* (average RR 0.90; 95% CI 0.78 to 1.05; 10 trials, 3545 women; I^2 = 48%, Tau^2 = 0.02; *low-quality evidence*) (Analysis 1.2). The evidence was downgraded for selective reporting and inconsistency. There is some suggestion of asymmetry observed in the funnel plot (Figure 4).

1.3 Development of type 2 diabetes

Two trials (Bancroft 2000; Landon 2009) reported no evidence of a difference between lifestyle interventions and control groups for *development of type 2 diabetes* (RR 0.98, 95% CI 0.54 to 1.76; two trials, 486 women; I^2 = 16%; Analysis 1.3; *low-quality evidence*). The evidence was downgraded for risk of bias and attrition bias. Bancroft 2000 only states that diagnosis was postnatally. Bancroft 2000 reports data for postnatal glucose metabolism, but there are no details at what time point the test was conducted. Landon 2009 reported follow-up at 4.5 to 10 years.

Neonatal primary outcomes

1.4 Perinatal (fetal and neonatal death) and later infant mortality

There is substantial uncertainty about the size and he direction of the effect for the outcome of *perinatal death* between lifestyle intervention and control groups reported in two trials (Crowther 2005; Landon 2009) (RR 0.09, 95% CI 0.01 to 1.70; two trials, 1988 infants; Analysis 1.4; *low-quality evidence*). The evidence was downgraded for risk of bias and imprecision. The evidence should be interpreted with caution as no perinatal deaths were reported in either intervention or control group in the Landon 2009 trial. No data were reported for later infant mortality.

1.5 Large-for-gestational age (LGA) (as defined by the trialists)

Lifestyle interventions were associated with a reduction in the risk of being born *large-for-gestational age* reported in six trials (Bancroft 2000; Bo 2014; Crowther 2005; Elnour 2008; Landon 2009; Yang 2014) (RR 0.60, 95% CI 0.50 to 0.71; six trials, 2994 infants; I^2 = 4%; Analysis 1.5; *moderate-quality evidence*). The evidence was downgraded due to unclear and high risk of bias for allocation concealment, lack of blinding and selective reporting.

1.6 Death of serious morbidity composite (variously defined by trials, e.g. perinatal or infant death, should dystocia, bone fracture or nerve palsy)

A composite of serious infant outcome was reported in two trials (Crowther 2005; Landon 2009). There was no evidence of a difference between lifestyle intervention and control groups for risk of a composite of serious infant outcome (average RR 0.57, 95% CI 0.21 to 1.55; two trials, 1930 infants; I^2 = 82%, Tau^2 = 0.44; Analysis 1.6; *very low-quality of evidence*). The evidence was downgraded for inconsistency, risk of bias and imprecision. In the Crowther 2005 trial, the composite included one or more of: death, shoulder dystocia, bone fracture and nerve palsy. In the Landon 2009 trial the composite included: stillbirth, neonatal death, hypoglycaemia, hyperbilirubinaemia, elevated cord-blood C-peptide level and birth trauma. We decided to include the meta-analysis as the direction of the treatment effect is the same in both trials.

Primary outcomes not reported in the included studies

None of the included trials prespecified or reported **neurosensory disability in later childhood** as a trial outcome.

Maternal secondary outcomes

1.7 Use of additional pharmacotherapy

Use of additional pharmacotherapy was reported in eight trials. One trial (Ferrara 2011) found no evidence of a difference between women who had received lifestyle interventions and those in the control groups for the use of additional oral anti-diabetic agents which were required by 28% (27/96) women in the intervention group and 36% (36/101) in the control group (RR 0.79, 95% CI 0.52 to 1.19; one trial, n = 197 women; Analysis 1.7). Eight trials reported on the need for additional insulin (Bancroft 2000; Bo 2014; Crowther 2005; Elnour 2008; Ferrara 2011; Gillen 2004; Landon 2009; Yang 2014). Lifestyle interventions were associated with an increase in the use of supplementary insulin (214/1626; 13%) compared with control interventions (62/1628; 4%) (average RR 2.54; 95% CI 1.19 to 5.42; nine trials, n = 3254 women; I^2 = 80%, Tau^2 = 0.77; Analysis 1.7). We advise caution when interpreting these results due to the observed

heterogeneity (inconsistency). The data suggest a wide spread of treatment effects and incidence of the outcome (Analysis 1.7).

1.8 Maternal hypoglycaemia

One small trial of 19 women (Jovanovic-Peterson 1989) reported no events of *maternal hypoglycaemia* in either the lifestyle intervention or the control groups (Analysis 1.8).

1.9 Glycaemic control during/after treatment

Glycaemic control during/after treatment was reported in seven trials that provided data suitable for meta-analysis. Data from the Kaviani 2014 trial were not suitable for inclusion in the meta-analysis for fasting blood glucose concentration or postprandial blood glucose concentration. Further information has been requested from the authors.

1.9.1 Six trials reported on fasting blood glucose concentrations (Bancroft 2000; Bo 2014; Elnour 2008; Garner 1997; Mendelson 2008; Youngwanichsetha 2014). There was no clear evidence of a difference between lifestyle interventions and control groups in fasting blood glucose concentrations during/at the end of treatment (average standardised mean difference (SMD) -0.32; 95% CI -0.72 to 0.07; six trials; 853 women; I^2 = 85%, Tau^2 = 0.18; data not shown). Bancroft 2000 reported on median and range for postnatal fasting blood glucose concentrations (Table 6). There was no evidence of a difference between the intervention and control group.

1.9.2 Postprandial blood glucose concentration was reported at one hour in two trials (Garner 1997; Jovanovic-Peterson 1989) and at two hours in two trials (Bancroft 2000; Youngwanichsetha 2014); one trial did not provide details of the timing of the postprandial test (Bo 2014). The data from the Bancroft 2000 trial are median and range which could not be included in a meta-analysis and are summarised in Table 6. Lifestyle interventions were associated with a decrease in postprandial blood glucose concentration at the end of treatment (average mean difference (MD) -27.11 mg/dL; 95% CI -44.62 to -9.61; four trials, n = 588 women; I^2 = 97%, Tau^2 = 300.13; data not shown). Visual inspection of the forest plot suggests that the Jovanovic-Peterson 1989 trial is an outlier. This is a very small trial of just 19 women in which the treatment effect suggests very large benefit. The removal of this trial from the meta-analysis does not substantially alter the estimate of treatment effect or benefit (MD -10.95 mg/dL, 95% CI -13.50 to -8.40 - analysis not shown), but observed heterogeneity is reduced to I^2 = 0% (Analysis 1.9).

1.9.3 HbA1c was reported at the end of treatment in six trials (Bancroft 2000; Bo 2014; Elnour 2008; Jovanovic-Peterson 1989; Mendelson 2008; Youngwanichsetha 2014). Lifestyle interventions were associated with a reduction in HbA1c values at the end of treatment (average MD -0.33 mmol/mol; 95% CI -0.47 to -0.19; six trials, n = 532 women; I^2 = 66%, Tau^2 = 0.02; Analysis 1.9).

1.10 Weight gain in pregnancy

Weight gain in pregnancy was reported in four trials (Crowther 2005; Garner 1997; Landon 2009; Yang 2014). Lifestyle interventions were associated with a decrease in weight gain in pregnancy (average MD -1.30 kg, 95% CI -2.26 to -0.35; four trials, n = 2930 women; I^2 = 80%, Tau^2 = 0.75; Analysis 1.10). The largest difference between groups was observed in the Landon 2009 trial (2 kg in the lifestyle intervention group versus 5 kg in the control group), whereas the Yang 2014 trial found no evidence of a difference between groups but also reported a mean increase in weight during pregnancy of approximately 15 kg.

1.11 Induction of labour

Induction of labour was reported in four trials (Bancroft 2000; Crowther 2005; Landon 2009; Yang 2014). There was no evidence of a difference between the lifestyle intervention groups and the control groups (average RR 1.20, 95% CI 0.99 to 1.46; four trials, n = 2699 women; I^2 = 37%, T^2 = 0.01; *high-quality evidence;* Analysis 1.11).

1.12 Postpartum haemorrhage (as defined by trialists)

Two trials reported on *postpartum haemorrhage* (Crowther 2005; Elnour 2008). There was no evidence of a difference for postpartum haemorrhage between women in the lifestyle intervention or the control groups (average RR 0.61, 95% CI 0.20 to 1.89; two trials, n = 1165 women; I^2 = 64%, Tau^2 = 0.46; Analysis 1.12).

1.13 Postpartum infection

Postpartum infection was reported in the Crowther 2005 trial only. There was no evidence of a difference for postpartum infection between women in the lifestyle intervention or the control groups (RR 0.61, 95% CI 0.34 to 1.10; one trial, n = 1000 women; Analysis 1.13).

1.14 Perineal trauma/tearing

Perineal trauma/tearing was reported in the Crowther 2005 trial only. There was no evidence of a difference for perineal trauma/tearing between women in the lifestyle intervention or the control groups (RR 1.04, 95% CI 0.93 to 1.18; one trial, n = 1000 women; *moderate-quality evidence;* Analysis 1.14). Evidence was downgraded due to imprecision as it is based on a single trial.

1.15 Breastfeeding at discharge, six weeks postpartum, six months or longer

Breastfeeding was reported in two trials (Crowther 2005; Ferrara 2011). Crowther 2005 reported no clear difference for rates of breastfeeding at discharge between the lifestyle intervention or the control groups (RR 1.04, 95% CI 0.99 to 1.10; one trial, n = 1000 women). Ferrara 2011 reported on breastfeeding at six weeks' postpartum (RR 0.97, 95% CI 0.87 to 1.07; one trial, n = 188 women) and six months or longer (RR 1.31, 95% CI 0.99 to 1.74; one trial, n = 161 women). At neither six weeks' postpartum nor six months postpartum was there evidence of a difference in breastfeeding rates between lifestyle

intervention and control groups. See Analysis 1.15.

1.16 and 1.17 Sense of well-being and quality of life

Quality of life was reported in two trials (Crowther 2005; Elnour 2008) both during the treatment and at three months postpartum using the SF36 questionnaire. Maternal quality of life was improved during treatment for physical functioning, role physical, bodily pain, general health, vitality, social functioning, role emotional, health state utility and overall physical component for women receiving a lifestyle intervention compared with the control group (Analysis 1.16). There were no clear differences between intervention and control groups for mental health, overall mental component or anxiety. At three months follow-up, only social functioning remained different between intervention and control groups. No other differences between groups were found for quality of life (Analysis 1.17).

No data were reported for adherence to the intervention, placental abruption, maternal mortality, behavioural changes associated with the intervention, views of the intervention or relevant biomarker changes associated with the intervention (including adiponectin, free fatty acids, triglycerides, high-density lipoproteins, (HDL) low-density lipoproteins (LDL), insulin).

Long-term outcomes for mother

1.18 Postnatal depression

Postnatal depression was reported in the Crowther 2005 trial only and defined as a Edinburgh Postnatal Depression Score > 12. Lifestyle interventions were associated with a decrease in the risk of postnatal depression compared with the control group (RR 0.49, 95% CI 0.31 to 0.78; one trial, n = 573 women; *low-quality evidence;* Analysis 1.18). The evidence was downgraded for imprecision as it is based on a single trial and risk of attrition bias as only 68% of randomised women responded to the questionnaire.

1.19 Postnatal weight retention or return to pre-pregnancy weight

Ability to *meet postpartum weight goals* was reported in one study (Ferrara 2011). At six weeks and seven months postpartum there was no evidence of a difference between the lifestyle and control groups for this outcome (RR 1.20, 95% CI 0.67 to 2.17; n = 189 women; RR 1.59, 95% CI 0.99 to 2.57, n = 159 women, respectively; Analysis 1.19). At 12 months postpartum more women in the lifestyle group had met postpartum weight goals than in the control group (RR 1.75, 95% CI 1.05 to 2.90; participants = 156; *low-quality evidence;* Analysis 1.19). The evidence was downgraded due to imprecision and risk of bias.

1.20 and 1.21 Impaired glucose intolerance

Fasting plasma glucose concentration at three months postpartum was reported by Elnour 2008. There was a non-significant trend towards lower fasting glucose concentrations in the women who had received a lifestyle intervention compared with the control group (MD -0.08 mmol/L, 95% CI -0.16 to 0.00; one trial, n = 165 women; Analysis 1.20). At six months postpartum, there was a reduction in fasting blood glucose concentrations in the lifestyle intervention group compared with the control group (MD -0.14 mmol/L, 95% CI -0.22 to -0.06; one trial n = 165 women; Analysis 1.20). Data from Kaviani 2014 were not suitable for inclusion in the meta-analysis for postnatal glycaemic blood glucose concentrations. The authors have been contacted for further information. Bancroft 2000 found no evidence of a difference between lifestyle intervention and control groups for diagnosis of postnatal *impaired glucose tolerance* (RR 0.67, 95% CI 0.12 to 3.69; one trial, n = 56 women; Analysis 1.21).

1.22 Cardiovascular health (as defined by the trialists including blood pressure, hypertension, cardiovascular disease, metabolic syndrome)

Landon 2009 reported no evidence of a difference between lifestyle and interventions groups for the risk of maternal *metabolic syndrome* at follow-up at between 4.5 to 10 years after diagnosis of gestational diabetes mellitus (GDM) (RR 0.93, 95% CI 0.71 to 1.22; n = 430 women; Analysis 1.22).

There was no evidence of a difference between lifestyle and control groups for body mass index (BMI) at the follow-up. Landon 2009 reported data for maternal BMI at long-term follow-up, 4.5 and 10 years after diagnosis of GDM. The trial by Garner 1997 followed up mothers and infants at nine to 11 years. The data in both trials were not in a format suitable for inclusion in a meta-analysis and are summarised in Table 8.

No data were reported for type 1 diabetes, subsequent gestational diabetes, cardiovascular health (blood pressure, hypertension or cardiovascular disease).

Neonatal secondary outcomes

1.23 Stillbirth

Stillbirth was reported in four trials (Bancroft 2000; Crowther 2005; Garner 1997; Landon 2009). There was no evidence of a difference in the risk of stillbirth between the lifestyle and the control groups (RR 0.15, 9% CI 0.01 to 2.86; four trials, n = 2355 infants). There were no events of stillbirth reported in the lifestyle intervention group (0/1172) and three events in the control group (3/1183). All three stillbirths were reported from a single trial (Crowther 2005) (Analysis 1.23).

1.24 Neonatal death

Neonatal death was reported in five trials (Bancroft 2000; Crowther 2005; Garner 1997; Landon 2009; Yang 2014). There was no evidence of a difference in the risk for neonatal death between the lifestyle and the control groups (RR 0.73, 95% CI 0.22 to 2.42; five trials, n = 3055 infants; I^2 = 0%). Event rates were low with 4/1511 (0.3%) neonatal deaths in the lifestyle intervention group and 6/1544 (0.4%) in the control group (Analysis 1.24).

1.25 Macrosomia (greater than 4000 g; or as defined by individual study)

Macrosomia was reported in seven trials (Crowther 2005; Elnour 2008; Ferrara 2011; Garner 1997; Landon 2009; Mendelson 2008; Yang 2014). The Crowther 2005 and Yang 2014 trials defined macrosomia as ≥ 4 kg, the remaining trials used a definition of > 4 kg. Lifestyle interventions were associated with a significant reduction in the risk of macrosomia compared with the control group (average RR 0.64, 95% CI 0.48 to 0.87; seven trials, n = 3422 infants; I^2 = 65%, Tau^2 = 0.10; Analysis 1.25). Sensitivity analyses were used to explore the heterogeneity by looking at those trials that were judged to be low risk of bias for allocation concealment (Crowther 2005; Landon 2009). The direction of the treatment effect was unchanged and heterogeneity was reduced to I^2 = 0% (RR 0.44, 95% CI 0.34 to 0.57; participants = 1961; studies = two).

1.26 Small-for-gestational (SGA) age (as defined by trialists)

There was no evidence of a difference in the risk of the infant being born SGA between the lifestyle or the control group (RR 0.98, 95% CI 0.73 to 1.31; n = 2324 infants; four trials; I^2 = 0%; Analysis 1.26).

1.27 Birth trauma (shoulder dystocia, bone fracture, nerve palsy)

Birth trauma (not specified but including shoulder dystocia, bone fracture and/or nerve palsy) was reported in three trials (Garner 1997; Landon 2009; Yang 2014). Event rates were low with only the Landon 2009 trial reporting 3/964 (0.3%) events in the lifestyle intervention group and 6/966 (0.6%) in the control group (RR 0.48, 95% CI 0.12 to 1.90; three trials, n = 1930 infants) (Analysis 1.27). Event rates for *bone fracture* were very low with only one event being reported in the Crowther 2005 trial in the control group (1/885) compared with no events in the lifestyle intervention group (0/845). No events were reported by Yang 2014 (RR 0.35, 95% CI 0.01 to 8.45, two trials, n = 1730 infants) (Analysis 1.27). *Nerve palsy* was only reported in one trial (Crowther 2005) where there were no events in the lifestyle intervention group (0/506) and 3/524 events in the control group. The difference was not statistically significant. *Shoulder dystocia* was reported in five trials (Bancroft 2000; Crowther 2005; Elnour 2008; Landon 2009; Yang 2014). Lifestyle interventions were associated with a significant decrease in the risk of shoulder dystocia (RR 0.38, 95% CI 0.21 to 0.66; five trials, 2894 infants; Analysis 1.27).

1.28 Gestational age at birth

There was no evidence of a difference for g*estational age at birth* between the lifestyle intervention and control groups reported in five trials (Bancroft 2000; Garner 1997; Gillen 2004; Landon 2009; Yang 2014) (MD 0.04 weeks, 95% CI -0.13 to 0.20; n = 2057 infants; five trials; I^2 = 31%; Analysis 1.28). Two trials (Crowther 2005; Jovanovic-Peterson 1989) reported data in a format that could not be included in a meta-analysis (Table 9); their results concur with the meta-analysis indicating no evidence of a difference in gestational age at birth between infants exposed to the lifestyle intervention and control groups.

1.29 Preterm birth (< 37 weeks' gestation; and < 32 weeks' gestation)

Lifestyle interventions were associated with a reduction in the risk of *preterm birth* (< 37 weeks' gestation) compared with the control group as reported in four trials (Elnour 2008; Landon 2009; Yang 2014) (RR 0.71, 95% CI 0.53 to 0.96; n = 1797 infants; three trials; I^2 = 27%; Analysis 1.29).

1.30 Five-minute Apgar less than seven

There was no evidence of a difference between the lifestyle intervention and control groups for a *five-minute Apgar score less than seven* reported by Crowther 2005 (RR 0.56; 95% CI 0.21 to 1.52; one trial, n = 1030 infants; Analysis 1.30).

1.31 Birthweight and z score

Birthweight was reported in six trials (Bancroft 2000; Crowther 2005; Garner 1997; Jovanovic-Peterson 1989; Landon 2009; Yang 2014). Lifestyle interventions were associated with a significant reduction in birthweight (MD -109.64 g, 95% CI -149.77 to -69.51; six trials, n = 3074 infants; Analysis 1.31) without a consequent increase in the risk of SGA as previously reported (Analysis 1.26). No data were reported for z scores.

1.32 Length and z score

Length at birth - one trial (Yang 2014) reported no evidence of a difference in infant length at birth between infants exposed to a lifestyle intervention or the control group (MD -0.10 cm, 95% CI -0.37 to 0.17; one trial, n = 700 infants; Analysis 1.32). No data were reported for z scores.

1.33 Adiposity (including skinfold thickness measurements (mm); fat mass)

Neonatal fat mass - one trial (Landon 2009) reported that the infants exposed to the lifestyle intervention had a decreased whole-body fat mass (estimated from skinfold thickness) compared with the control group (MD -37.30 g, 95% CI -63.97 to -10.63; one trial, 958 infants; *low-quality evidence;* Analysis 1.33). The evidence was downgraded for risk of bias and imprecision as it was based on a single study. No data were reported for skinfold thickness.

1.34 Neonatal hypoglycaemia

Neonatal hypoglycaemia - six trials (Bancroft 2000; Crowther 2005; Elnour 2008; Garner 1997; Landon 2009; Yang 2014) found no evidence of a difference in the risk of neonatal hypoglycaemia between the infants exposed to a lifestyle intervention and those exposed to the control group (average RR 0.99, 95% CI 0.65 to 1.52; six trials, n = 3000 infants; I^2 = 48%, Tau^2 = 0.12; *moderate-quality evidence;* Analysis 1.34). The evidence was downgraded for risk of bias.

1.35 Respiratory distress syndrome

Respiratory distress syndrome - four trials (Bancroft 2000; Crowther 2005; Elnour 2008; Landon 2009) found no evidence of a difference in the risk of respiratory distress syndrome between exposure to lifestyle intervention or control groups (average RR 0.79, 95% CI 0.34 to 1.85, four trials, n = 2195 infants; I^2 = 64%, Tau^2 = 0.44; Analysis 1.35).

1.36 Neonatal jaundice (hyperbilirubinaemia)

Neonatal jaundice (hyperbilirubinaemia) - four trials (Crowther 2005; Elnour 2008; Garner 1997; Landon 2009) found no evidence of a difference in the risk for hyperbilirubinaemia between infants exposed to the lifestyle intervention or the control group (average RR 0.76, 95% CI 0.50 to 1.16; four trials, n = 2362; I^2 = 47%, Tau^2 = 0.08; Analysis 1.36).

1.37 Hypocalcaemia

Hypocalcaemia was reported in two trials (Elnour 2008; Garner 1997). Lifestyle interventions were associated with an increased risk for hypocalcaemia compared with the control groups (RR 1.38, 95% CI 1.01 to 1.88; two trials, n = 464 infants; Analysis 1.37).

1. 38 Polycythaemia

Polycythaemia was reported in one trial (Elnour 2008). There was no evidence of a difference between lifestyle intervention and control group for the risk for infant polycythaemia (RR 0.22, 95% CI 0.01 to 5.40; one trial, n = 165 infants; Analysis 1.38). Caution is recommended in interpreting the results due to the low event rates (0/99 in the lifestyle group; 1/66 in the control group).

No data were reported for head circumference, z scores for anthropometric measures, ponderal index, skinfold thickness or relevant biochemical markers.

Childhood follow-up

Three trials reported follow-up data into childhood (Crowther 2005; Garner 1997; Landon 2009).

The Landon 2009 trial has reported on follow-up of children at ages five to 10 years. Seventy-four per cent (666/905) of the original trial cohort were contacted and 500 (55%) consented to enrol in the follow-up. Continuous data for BMI z score, cholesterol concentration, triglycerides and impaired glucose tolerance were reported as adjusted means with 95% CIs and we have therefore not included these data in the meta-analysis.

Gillman 2010 reported on the follow-up at four to five years from 199 (20%) children in Australia who were born to 1000 mothers who participated in the ACHOIS trial (Crowther 2005). This cohort of data is likely to be biased as it does not represent the entire trial population. The mean age at follow-up in this cohort was 4.7 ± 0.2 years in the intervention group and 4.7 ± 0.4 years in the control group.

The offspring of the Garner 1997 trial were followed up at seven to 11 years by Keely (2008) (for metabolic markers of insulin resistance).

1.39 Childhood weight and z score

Childhood weight was reported in one trial (Crowther 2005) who found no evidence of a difference between the lifestyle intervention and control group exposed infants (MD -0.30 kg, 95% CI -1.29 to 0.69; one trial, n = 199 children; Analysis 1.39). No data were reported for z scores.

1.40 Childhood height and z score

Childhood height was reported in one trial (Crowther 2005) who found no evidence of a difference between the lifestyle intervention and control group exposed infants (MD -0.60 cm, 95% CI -2.05 to 0.85; one trial, n = 199 children; Analysis 1.39). No data were reported for z scores.

1.41 and 1.42 Adiposity (including BMI, skinfold thickness, fat mass)

Childhood BMI was reported in three trials (Crowther 2005; Garner 1997; Landon 2009).

There was no evidence of a difference between groups for BMI ≥ 85th percentile reported in the three trials (Crowther 2005; Garner 1997; Landon 2009) (RR 0.91, 95% CI 0.75 to 1.11; participants = 767; I^2 = 4%; Analysis 1.41; *moderate-quality evidence*). The evidence was downgraded for risk of bias. Childhood BMI z score was reported in one trial (Crowther 2005) which found no evidence of a difference between groups at four to five years of age (MD 0.08, 95% CI -0.28 to 0.44; one trial, n = 199 children). The Landon 2009 follow-up of children at five to 10 years reported an adjusted mean BMI z score of 0.33 (95% CI 0.15 to 0.51; n = 264) in the treated group and an adjusted mean BMI z score of 0.36 (95% CI 0.17 to 0.55, n = 236) in the untreated group. These data could not be combined in a meta-analysis (Analysis 1.42).

1.43 Impaired glucose tolerance

One study (Garner 1997) reported no evidence of a difference between the treated and untreated groups for fasting blood glucose concentration at seven to 11 years of age (MD 0.10 mg/dL, 95% CI -0.10 to 0.30; one trial, n = 68 children). The follow-up of the Landon 2009 trial reported that 12/264 (5.4%) of children from the treated group and 13/236 (7.2%) of children from the untreated group had impaired fasting glucose concentration ≥ 5.6 mmol/L (100 mg/dL). An adjusted mean fasting blood glucose concentration of 88.41 mg/dL (95% CI 87.33 to 89.50; n = 264) was reported for the treated group and an adjusted mean blood glucose concentration of 88.67 mg/dL (95% CI 87.56 to 89.78, n = 236) was reported for the untreated group. These data could not be combined in a meta-analysis. There was no evidence of a difference between the lifestyle intervention and control groups for child two-hour postprandial glucose concentration (MD 0.00 mg/dL, 95% CI -0.48 to 0.48; one trial, n = 68 children). See Analysis 1.43.

1.44 Dyslipidaemia or metabolic syndrome

Dyslipidaemia or metabolic syndrome - in Garner 1997 there was no evidence of a difference between groups in total cholesterol concentration (MD -0.20 mg/dL, 95% CI -0.55 to 0.15; one trial, n = 68 children); HDL (MD 0.10 mg/dL, 95% CI -0.05 to 0.25; one trial, n = 68 children) or LDL (MD -0.12 mg/dL, 95% CI -0.50 to 0.26; one trial, n = 68 children). The follow-up of the Landon 2009 trial reported low HDL cholesterol (< 40 mg/dL) in 27/264 (13%) of children in the treated group and 22/236 (12%) in the untreated group. The adjusted mean for HDL cholesterol concentration for the treated group was 54.35 mg/dL (95% CI 52.42 to 56.28; n = 264) and for the untreated group the adjusted mean HDL cholesterol concentration was 55.10 mg/dL (95% CI 53.16 to 57.05; n = 236). These data could not be combined in a meta-analysis (Analysis 1.44).

The Landon 2009 follow-up also reported elevated triglyceride concentrations (≥ 100 mg/dL four to nine years, ≥ 130 mg/dL 10 years) in 38/264 (18%) of the treated group and 29/236 (16%) in the untreated group. The adjusted mean triglyceride concentration was 58.91 mg/dL (95% CI 54.82 to 63.30; n = 264) for the treated group and adjusted mean triglyceride concentration for the untreated group was 57.38 mg/dL (95% CI 53.33 to 61.73; n = 236). These data could not be combined in a meta-analysis. Childhood data for triglyceride concentrations were reported by Garner 1997 who found a median value of 0.8 mmol/L (range 0.4 to 2.7) in 43 children followed up whose mothers had been in the lifestyle intervention group and a median (range) of 0.83 mmol/L (0.5 to 5.4) in 25 children whose mothers had been in the control group.

Blood pressure - the follow-up of the Landon 2009 trial reported data for the number of children with hypertension (≥ 95th percentile for age, sex and height) which occurred in 30/264 (11.5%) children from the treated group and 23/236 (10%) in the untreated group. The adjusted mean systolic blood pressure in the treated group was 100 mm/Hg (95% CI 98 to 101, n = 264) and for the untreated group the adjusted mean systolic blood pressure was 100 mm/Hg (95% CI 98 to 101, n = 236). The adjusted mean diastolic blood pressure in the treated group was 60 mm/Hg (95% CI 59 to 61, n = 264) and for the untreated group the adjusted mean systolic blood pressure was 59 mm/Hg (95% CI 58 to 60, n = 236).

No data were reported for the following childhood outcomes: weight or height z scores, head circumference and z scores, educational attainment, type 1 diabetes or type 2 diabetes.

Adult outcomes

No data were reported for any of the pre-specified adult outcomes for this review (weight, height, adiposity, employment, education and social status/achievement, dyslipidaemia or metabolic syndrome, type 1 diabetes, type 2 diabetes, impaired glucose tolerance, cardiovascular health (as defined by trialists including blood pressure, hypertension, cardiovascular disease, metabolic syndrome)).

Health service use

1.45 Number of antenatal visits or admissions

There was no evidence of a difference between the lifestyle intervention and control groups for the *number of antenatal visits or admissions* - (RR 1.06, 95% CI 0.87 to 1.29; one trial, n = 1000 women; Analysis 1.45) reported in one trial (Crowther 2005).

1.46 and 1.47 Number of hospital or health professional visits (including midwife, obstetrician, physician, dietician, diabetic nurse)

Number of hospital or health professional visits (during pregnancy) - the women randomised to the lifestyle intervention groups were more likely to have a visit with a dietitian compared with the control groups (RR 9.24, 95% CI 7.12 to 12.01; one trial, n = 1000 women; Analysis 1.46) or a visit with a diabetes care educator (RR 8.55, 95% CI 6.67 to 10.96; one trial, n = 1000 women; Analysis 1.46) than the control group. This is most likely due to the trial protocol requiring a visit with a dietician (Crowther 2005). One trial (Yang 2014) reported no evidence of a difference in the number of visits to an obstetrician between the lifestyle intervention and control groups (MD 0.20 visits, 95% CI -0.21 to 0.61; one trial, n = 700 women; Analysis 1.47) and Ferrara 2011 reported no evidence of a difference between groups for the number of visits to an antenatal care provider (not specified) - (MD 0.10 visits, 95% CI -1.58 to 1.78; one trial, 197 women; Analysis 1.47). Other data reported as median or mean without standard deviation are summarised in Table 10.

1.48 Admission to neonatal intensive care unit/nursery

There was no evidence of a difference in the *admission to neonatal intensive care unit or special care baby unit* between infants who had been exposed to the lifestyle intervention or the control groups (average RR 0.91, 95% CI 0.59 to 1.40; three trials, n = 2030 infants; I^2 = 70%, Tau^2 = 0.09; Analysis 1.48) reported in three trials (Bancroft 2000; Crowther 2005; Landon 2009).

Costs - only one trial (Crowther 2005) provided data for the economic impact of a lifestyle intervention compared with usual care. Table 11 illustrates the costs of gestational diabetes to the families and the health service for the lifestyle and control groups. Not surprisingly the costs were higher in the intervention group than the control group which is mainly due to increased surveillance and increased contact with health professionals.

No data were reported for duration of stay in neonatal intensive care or special care baby unit, or the duration of the mothers' stay in hospital (antenatal, neonatal, postnatal), extra use of healthcare services or women's view of treatment advice.

Discussion

Summary of main results

Fifteen trials (in 45 reports) are included in this review (4501 women, 3768 infants).

The evidence suggests that for women diagnosed with gestational diabetes mellitus (GDM), a lifestyle intervention (two or more interventions including dietary advice, physical activity, education, self-monitoring of blood glucose), there is no clear difference in risk of developing hypertension in pregnancy or of having a caesarean birth (Summary of findings table 1). There was no evidence of a difference between lifestyle intervention and control groups for the mother developing type 2 diabetes at follow-up ranging from 4.5 to 10 years. A lifestyle intervention was associated with a decrease in weight gain in pregnancy compared with a control group. The lifestyle intervention group did have more visits to health professionals and an increased use of additional pharmacological therapies. One small study (n = 159 women; Ferrara 2011) found that women in the lifestyle intervention group were more likely to meet postpartum weight goals at one year compared with the control group. Few trials reported on long-term maternal outcomes.

There was no evidence of a difference between infants exposed to lifestyle interventions or control for the risk of perinatal death or a composite of serious infant adverse events. Those infants exposed to the lifestyle intervention had a decreased risk of being born large-for-gestational age (LGA) (Summary of findings table 2) compared with the infants whose mothers had been in the control group. None of the included trials reported on childhood neurodisability. Infants who had been exposed to a lifestyle intervention had a decreased risk of or having macrosomia, being born preterm (< 37 weeks') and had a lower birthweight compared with the infants whose mothers had been in the control group. There was also a reduced risk of shoulder dystocia associated with lifestyle interventions. No infant adverse effects or increased likelihood of admission to neonatal intensive care were associated with the interventions reviewed. Follow-up into childhood was poorly reported with only three of the 15 included trials contributing data (Crowther 2005; Garner 1997; Landon 2009). There was no evidence of a difference between groups for body mass index (BMI) greater or equal to the 85th percentile and no evidence of a difference in dyslipidaemia or blood pressure. None of the trials have yet reported data for the infant as an adult (Summary of findings table 2).

Overall completeness and applicability of evidence

This review has focused on lifestyle interventions for the treatment of women with GDM that included a combination of interventions such as nutritional advice, physical activity, education, and self-monitoring of blood glucose concentrations. Lifestyle interventions are used as the primary therapeutic strategy for women diagnosed with GDM. Some women who commence lifestyle interventions will require supplementary pharmacological interventions (insulin or oral anti-diabetic pharmacological therapies), and this is evident from this review with 10 of the included studies reporting an increase in the use of supplementary pharmacological therapy.

The evidence for treatment needs to be taken in context for the needs of the individual woman, and other Cochrane systematic reviews have examined or plan to examine different dietary advice for women with GDM (Han 2013), exercise (Ceysens 2016), insulin (Brown 2016) and oral anti-diabetic pharmacological therapies (Brown 2015b). This review does not include women with impaired glucose tolerance, not meeting criteria for diagnosis of GDM, which is covered by the Han 2012 Cochrane systematic review.

Due to insufficient data we are unable to make any judgements on lifestyle interventions as a sole intervention without any supplementary pharmacological therapy. Nor are we able to make any judgements on the effectiveness of treatment based on duration of treatment as gestational age at trial entry was poorly reported for the included trials. In the description of included studies we have listed all of the interventions described by the included trials. There is a wide variety and diversity of interventions that include exercise, diet, self-monitoring of blood glucose and education for example. We are unable to determine which if any of the interventions are more effective than another but most of the interventions include some dietary component.

Quality of the evidence

Fifteen trials (45 publications) are included in this review (4501 women and their infants). The main reasons for downgrading evidence was inconsistency, imprecision and risk of bias. Overall, the evidence was judged to be of unclear risk of bias due to inadequate reporting of allocation concealment and blinding of outcome assessors and selective outcome reporting. There is variation between the trials with regards to the content of the lifestyle interventions (see Characteristics of included studies). The evidence is dominated by two large trials (Crowther 2005; Landon 2009) that included 1000 women and 958 women, respectively. Both of these trials were judged to be at low risk of bias.

Potential biases in the review process

We believe that we have made every effort to minimise biases in the review process. We have conducted a systematic search of the literature for randomised controlled trial evidence, not restricted by language or date of publication. Where necessary we have attempted to make contact with authors of primary studies to obtain additional methodological and/or outcome data. We have adhered to Cochrane methodology for searching, data extraction and analysis.

Agreements and disagreements with other studies or reviews

A systematic review assessing the effect of treatment of GDM by Poolsup 2014 included 10 studies and reported a decreased risk of macrosomia, LGA, shoulder dystocia and gestational hypertension. Seven of the 10 studies included in the Poolsup 2014 were excluded from our review based on study design and the remaining three trials that they included were also included in our review (Crowther 2005; Garner 1997; Landon 2009). Another systematic review by Hartling 2013 reported on the benefits and harms of treating GDM. The review found increased antenatal visits, reduced pre-eclampsia, shoulder dystocia and macrosomia in the treated group. No clear differences between intervention and control groups were found for neonatal hypoglycaemia, caesarean section, induction of labour or

admission to neonatal intensive care. The evidence was based on five randomised trials (including quasi-randomised trials) and six cohort studies. Three of the studies were included in our review (Crowther 2005; Garner 1997; Landon 2009).

Authors' conclusions

Implications for practice

Lifestyle interventions are resource-intensive and require trained personnel to provide optimal education and management support.

Low-quality evidence suggests that women receiving lifestyle interventions are less likely to have postnatal depression and are more likely to achieve postpartum weight goals than women in usual care or diet-only groups.

For the infant, there is moderate-quality evidence of a reduced risk of being born large-for-gestational age (LGA) and low-quality evidence for reduced adiposity (neonatal fat mass) for infants exposed to lifestyle interventions compared with usual care or diet-alone groups. The limited available moderate-quality evidence for longer-term follow-up suggests there is no clear difference between groups for adiposity in childhood (childhood BMI > 85th centile) and no trials reported data into adulthood for adiposity.

The value of lifestyle interventions in low- and middle-income countries or for different ethnicities remains unclear. The longer-term benefits or harms of lifestyle interventions remains unclear due to limited reporting. Lifestyle interventions are useful as the primary therapeutic strategy and most commonly includes components of healthy eating, physical activity and self-monitoring of blood glucose levels.

Implications for research

Future research should focus on which specific interventions are most useful, which health professionals should give them and the optimal format for providing the information. Evaluation of long-term outcomes for the mother and her child should be a priority when planning future trials. There has been no in-depth exploration of the costs ‘saved’ from reduction in risk of LGA/macrosomia and potential longer-term risks for the infants.

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We acknowledge the contribution of the authors of the other two reviews that were split from this original review in the preparation of the core background sections of the new review protocols:

Oral anti-diabetic pharmacological therapies for the treatment of women with gestational diabetes - Julie Brown, Ruth Martis, Brenda Hughes, Janet Rowan, Caroline Crowther.

Insulin for the treatment of women with gestational diabetes - Julie Brown, Luke Greskowiak, Michelle Downie, Kate Williamson, Caroline Crowther.

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As part of the pre-publication editorial process, this review has been commented on by four peers (an editor and three referees who are external to the editorial team) and the Group's Statistical Adviser.

Contributions of authors

Julie Brown guarantees this review.

Julie Brown wrote the first version of this review and identified studies for inclusion along with Stephen Brown. She also was undertook data extraction and assessed risk of bias. She prepared the GRADE 'Summary of findings' tables.

The remaining authors Nisreen A Alwan; Jane West, Stephen Brown, Christopher JD McKinlay, Diane Farrar and Caroline Crowther have all contributed to interpretation of the data from clinical, paediatric and expert opinions and have provided significant feedback to draft versions, including the GRADE 'Summary of findings' tables.

Declarations of interest

Julie Brown: none known.

Nisreen A Alwan: none known.

Jane West: none known.

Stephen Brown: none known.

Christopher JD McKinlay: none known.

Diane Farrar: none known.

Caroline A Crowther is the lead investigator for the ACHOIS trial that assessed treatment for women with mild gestational diabetes. This will be considered for inclusion in this review. However, Professor Crowther will not be involved in the decisions about inclusion of data or any data extraction from that trial.

Differences between protocol and review

There are some differences between our published protocol (Brown 2015) and the full review, these are listed below.

Background - portions of the background have been amended for clarity following feedback from the authors of this review.

Objectives - this section has been edited to reflect that the intervention is about 'combined' lifestyle interventions which could be with or without pharmacotherapy.

Published notes

The original review (Alwan 2009) has been split into three new reviews due to the complexity of the included interventions. The following new review protocols have been published.

Lifestyle interventions for the treatment of women with gestational diabetes (this review)

Oral anti-diabetic pharmacological therapies for the treatment of women with gestational diabetes

Insulin for the treatment of women with gestational diabetes

There will be similarities in the background, methods and outcomes between these three systematic reviews.

Characteristics of studies

Characteristics of included studies

Bancroft 2000

Methods	Randomised controlled pilot study recruiting from 2 centres.
Participants	68 women. **Inclusion criteria:** impaired glucose tolerance (Table 7). **Exclusion criteria:** none stated. **Setting:** specialist diabetic/antenatal clinics, Leeds, UK. **Timeframe:** not specified.
Interventions	1) Intervention (Monitored) group women were given standard dietary advice about restricting carbohydrate intake to 185 g/day and a diet sheet listing calorific values of common foods. Glucose metabolism was monitored by capillary glucose series 5 days a week (1 to 2 hours postprandial), HbA1c was measured monthly (insulin was introduced if 5 or more capillary measurements > 7.0 mmol/L in 1 week), serial ultrasound for growth and amniotic fluid, Doppler studies, CTG monitoring (n = 32) versus 2) Control (Unmonitored) group women received dietary advice, HbA1c monthly (but data not made available) but no capillary glucose measurements (n = 36). Women cared for in a combined diabetic clinic run jointly by a diabetologist and an obstetrician. Birth was no later than 41 weeks' gestation.
Outcomes	Primary outcome measure was admission to special care baby unit. Secondary outcomes: perinatal morbidity (including birth trauma, metabolic disturbance, gestation at birth, birthweight, stillbirth, neonatal hypoglycaemia, RDS), LGA, measures of maternal inconvenience, number of capillary samples, number of antenatal clinic visits, mode of delivery, IOL, frequency of insulin use, HbA1c.
Notes	2 women in the unmonitored group developed diabetes mellitus, both were diagnosed postnatally and both delivered prematurely. ITT analysis: not stated (but all women remained in their allocated groups). Funding: not stated. Sample size calculation: not stated. Conflicts of interest: no declarations made in manuscript.

Risk of bias table

Bias	Authors' judgement	Support for judgement
Random sequence generation (selection bias)	Low risk	Computer-generated codes, telephone randomisation service used.
Allocation concealment (selection bias)	Low risk	Randomisation was controlled from a trial centre and administered by telephone.
Blinding of participants and personnel (performance bias)	Low risk	"the diabetologist was aware of the group to which each woman was randomised but the obstetrician was blinded."
Blinding of outcome assessment (detection bias)	Unclear risk	Not stated.
Incomplete outcome data (attrition bias)	Unclear risk	No dropouts but 12 failed to attend follow-up postnatal measurements.
Selective reporting (reporting bias)	High risk	The original protocol was not seen. Additional outcomes listed in the methods section were reported in the results.
Other bias	Low risk	No evidence of other bias; groups balanced at baseline.

Bo 2014

Methods	Randomised controlled trial.
Participants	200 women randomised. **Inclusion criteria:** age 18–50 years; 24–26 weeks' gestational age; GDM diagnosis based on a 75 g OGTT (Table 7); singleton pregnancy. **Exclusion criteria:** BMI > 40 kg/m^2; any known diseases, medications or obstetrical contraindications to exercise. **Setting:** Sant'Anna Hospital, Turin, Italy. **Timing:** July 2009 to February 2012.
Interventions	Intervention - Behavioural and exercise (n = 50) - advised to walk briskly at least 20 min/day every day (140 min/week; Borg's scale target rating 12–14) plus individually oral/written recommendations for helping with healthy dietary choices (i.e. lowering carbohydrate intake, strategies for out-of-home eating, healthy cooking and food shopping and related behavioural suggestions) and debunking false myths about diet in pregnancy. Control - Diet (n = 50) - an individually-prescribed diet was given to each woman (carbohydrates 48% to 50%, proteins 18% to 20%, fats 30% to 35%, fibre 20–25 g/day, no alcohol). Exercise (n = 51) - advised to walk briskly at least 20 min/day every day (140 min/week; Borg's scale target rating 12–14). Behavioural (n = 49) - individually oral/written recommendations for helping with healthy dietary choices (i.e. lowering carbohydrate intake, strategies for out-of-home eating, healthy cooking and food shopping and related behavioural suggestions) and debunking false myths about diet in pregnancy. All women self-monitored blood glucose 4 to 6 times daily (preprandial and 2-hours postprandial). For this review we used the diet only group as the control group and the combined behavioural and exercise intervention as the intervention group.
Outcomes	Maternal outcomes - pregnancy-induced hypertension, infectious diseases, caesarean section, cholestasis during pregnancy and peri- and postpartum complications. Metabolic equivalents, triglycerides, insulin, insulin resistance, CRP. Fasting and postprandial blood glucose, and HbA1c. Neonatal outcomes - LGA; birthweight > 90th percentile), pre-term birth (gestational age at delivery < 37 weeks), and any neonatal conditions requiring a specific treatment or a prolonged in-hospital stay.
Notes	Treatment glycaemic targets were not detailed but insulin was started in the presence of fetal abdominal ultrasound > 70th percentile and or maternal hyperglycaemia (no details). Power calculation: yes, based on an expected 10% reduction in fasting glucose by exercise. ITT analysis: yes. Funding: Regione Piemonte 2009. Conflict of interest: the paper specifies that there authors report no conflicts of interest.

Risk of bias table

Bias	Authors' judgement	Support for judgement
Random sequence generation (selection bias)	Unclear risk	Randomisation was conducted through a website and is likely to be low risk of bias but there are insufficient details to be sure.
Allocation concealment (selection bias)	Low risk	'implemented through a website' - third person.
Blinding of participants and personnel (performance bias)	High risk	No details, but due to the different interventions the research staff and participants are unlikely to have been blinded.
Blinding of outcome assessment (detection bias)	Low risk	"The dieticians, the obstetricians who reported maternal/neonatal complications and the laboratory personnel were blinded to the group assignment."
Incomplete outcome data (attrition bias)	Low risk	All women randomised were analysed.
Selective reporting (reporting bias)	High risk	Additional outcomes are reported that are not listed in the methods section. Outcomes listed are very generalised. Primary outcomes are not pre-specified. Birthweight is listed in the trial registration document but is not reported or listed in the manuscript.
Other bias	Low risk	Groups appeared balanced at baseline.

Crowther 2005

Methods	Multi-centre randomised controlled trial (n = 18 centres; 14 in Australia and 4 in UK).
Participants	1000 women. **Inclusion criteria:** singleton or twin pregnancy between 16 and 30 weeks' gestation, 1 or more risk factors on selective screening or impaired glucose tolerance (Table 7), and had an abnormal 75 g OGTT at 24 to 34 weeks' gestation (Table 7). **Exclusion criteria:** women with previously treated GDM or active chronic systemic disease (except essential hypertension), more severe glucose impairment or less than 16 or more than 30 weeks' pregnant. **Setting:** 18 centres in antenatal clinics in Australia and UK. **Timing:** September 1993 to June 2003.
Interventions	1) Intervention group (n = 490): care replicated clinical care in which universal screening and treatment for GDM was available, individualised dietary advice from a qualified dietician, instructions on how to self-monitor glucose levels 4 times a day until fasting glucose levels of at least 3.5 mmol/L [63 mg/dL] and no more than 5.5 mmol/L [99 mg/dL], preprandial levels of no more than 5.5 mmol/L, and levels 2 hours postprandially that were no more than 7.0 mmol/L [126 mg/dL], followed by daily monitoring at rotating times during the day; and insulin therapy, with the dose adjusted based on glucose levels, if there were 2 capillary-blood glucose results during the 2-week period in which the fasting level was at least 5.5 mmol/L or the postprandial level was at least 7.0 mmol/L at 35 weeks' gestation or less, if the postprandial level was at least 8.0 mmol/L (144 mg/dL) at more than 35 weeks' gestation, or if 1 capillary-blood glucose results during the 2-week period was at least 9.0 mmol per L (162 mg per dL). 2) Control group (n = 510): care replicated clinical care in which screening for GDM was not available, women and caregivers were not aware of the diagnosis of glucose intolerance, at the discretion of the attending clinician, if indications arose that were suggestive of diabetes, further assessment for GDM was permitted, with treatment as considered appropriate.

Outcomes	Primary outcomes - infant: composite measures of serious perinatal complications (defined as 1 or more of death, shoulder dystocia, bone fracture, and nerve palsy), admission to neonatal nursery, and jaundice requiring phototherapy. Primary outcomes - women: need for IOL and caesarean section, health status, and psychological outcomes. Secondary outcomes - infant: gestational age at birth, birthweight, Apgar score of less than 7 at 5 mins, hypoglycaemia requiring IV therapy, convulsions, RDS, perinatal death, stillbirth, LGA, macrosomia, SGA. Childhood weight, BMI and height Secondary outcomes - women: number of prenatal visits to a health professional, mode of birth, weight during pregnancy, number of antenatal admissions, presence or absence of pregnancy-induced hypertension (BP ≥ 140/90 mmHg on 2 occasions 4 or more hours apart, perineal trauma, postpartum haemorrhage, postnatal infection, breastfeeding at hospital discharge, use of medication, postnatal depression.
Notes	93% of the women had been found to be at risk of GDM on the basis of OGTT, and the remainder on the basis of risk factors. 5 perinatal deaths (3 stillbirths and 2 neonatal deaths) occurred in the control group: 2 stillbirths were unexplained intrauterine deaths at term of appropriately grown infants, and 1 at 35 weeks' gestation, was associated with pre-eclampsia and intrauterine growth restriction. 1 infant had a lethal congenital anomaly, and 1 infant died after an asphyxial condition during labour with antepartum haemorrhage. After consent had been obtained, a proportion of the women (not fewer than 1 in 5) who had normal OGTT results were assigned to the routine-care group to help maintain blinding. Funding:National Health and Medical Research Council, Australia, Queen Victoria Hospital Research Foundation, Department of Obstetrics and Gynaecology- University of Adelaide. ITT: yes. Sample size calculation: yes based on the risk of serious perinatal outcome. Conflicts of interest: conflicts of interested were not documented in the manuscript. Gillman 2010 reports on 4-5 year follow-up from the ACHOIS trial (subgroup of 199 children from Australia). Pirc 2007 reports on a subgroup of women and infants from the ACHOIS trial from a single centre in Australia (n = 95 women).

Risk of bias table

Bias	Authors' judgement	Support for judgement
Random sequence generation (selection bias)	Low risk	Randomisation method used numbers generated by computer with variable block sizes of 6, 8, and 10.
Allocation concealment (selection bias)	Low risk	Randomisation method was performed centrally.
Blinding of participants and personnel (performance bias)	Low risk	Women and their health providers did not know the blood glucose results until after the birth.
Blinding of outcome assessment (detection bias)	Unclear risk	Not stated.
Incomplete outcome data (attrition bias)	Low risk	Dropouts: no losses to follow-up for primary and secondary clinical outcomes for women at end of treatment. No losses to follow-up for primary and secondary clinical outcomes for infants at end of treatment. For maternal health status outcomes of postnatal depression and quality of life 68% of women provided data for maternal health status. Overall data for clinical outcomes are complete.
Selective reporting (reporting bias)	Low risk	Includes maternal and infant outcomes.
Other bias	Low risk	No evidence of other bias, no differences in baseline.

Elnour 2008

Methods	Randomised controlled trial, stratified by age.
Participants	165 women randomised. **Inclusion criteria:** UAE national, within 20 weeks' gestation, confirmed diagnosis of GDM (Table 7), age 20 to 39 years. **Exclusion criteria:** abnormal renal or hepatic function, haemoglobinopathy. **Setting:** out-patient and primary care clinics, United Arab Emirates. **Timeframe:** not specified.
Interventions	1) Intervention group (structured pharmaceutical care) (n = 108) 10 to 30 mins with a clinical pharmacist. Options of treatment explained and encouraged to participate in self management. Structured education on GDM and management provided (diet and exercise, glycaemic control, self-monitoring, review of treatment if glycaemic control inadequate). Received printed education booklet which contained general information on diabetes, aims of treatment, diet and exercise and action to take if hypo- or hyperglycaemic. Asked to record plasma glucose at least 5 times per day for 3-4 days per week. Intervention took place at baseline and at monthly clinic visits. encourage to telephone pharmacist if any queries/concerns. versus 2) Control group (usual care) (n = 72) - monthly clinic visits and self monitoring but no additional education or counselling or liaison between pharmacist and prescribing doctor. Followed up to 6 months postpartum.
Outcomes	Knowledge, quality of life, maternal (hydramnios, severe hyperglycaemia, pre-eclampsia, gestational hypertension, lactation, postpartum haemorrhage, preterm labour, obstructed delivery, caesarean section, use of insulin, fasting blood glucose, HbA1c) and neonatal (macrosomia > 4 kg, hypoglycaemia, hyperbilirubinaemia, shoulder dystocia, congenital malformation, respiratory difficulties, SGA, LGA, polycythaemia, hypocalcaemia, preterm birth, admission to NICU) complications.
Notes	No details on method of screening or diagnosing GDM. Authors contacted in September 2012. Authors responded immediately with additional information. Power calculation: yes. ITT analysis: yes. Funding: not stated. Conflicts of interest: no evidence of a declaration made in the manuscript.

Risk of bias table

Bias	Authors' judgement	Support for judgement
Random sequence generation (selection bias)	Low risk	Restricted randomisation method 'to ensure that the number of patients allocated to control and intervention were in the same proportion in relation to their subgroup classification' This additional information was obtained through correspondence with the authors.
Allocation concealment (selection bias)	Unclear risk	No details provided.
Blinding of participants and personnel (performance bias)	High risk	Participants and researchers were not blinded.
Blinding of outcome assessment (detection bias)	Low risk	In additional information received from the authors it was stated that "nursing and pharmacy staff who assisted with the questionnaire administration were blinded regarding group to which individual patients had been assigned".
Incomplete outcome data (attrition bias)	Low risk	165 patients completed the trial (99 intervention and 66 control). 9 were lost to follow-up in the intervention group due to abortion (n = 4) and withdrawal (n = 5). 6 were lost in the control group (n = 3 abortion, n = 3 withdrawal). Per-protocol analysis.
Selective reporting (reporting bias)	Low risk	Reported on maternal and infant outcomes of relevance.
Other bias	Low risk	No evidence of other risk of bias.

Ferrara 2011

Methods	Randomised controlled trial - pilot study.
Participants	235 eligible women; 197 randomised. Mean age not provided although 77% were over 30 years. **Inclusion criteria:** women with GDM according to ADA (2000) criteria (Table 7), age 20 to 45 years. **Exclusion criteria:** < 18 years, multiple gestation, diagnosis of diabetic retinopathy, high risk pregnancy, thyroid disease diagnosed within 30 days, non-English speaker, pre-gestational diabetes, known cardiovascular or lung disease, haemoglobin < 9.5 mg/dL, haematocrit < 30%, hypertension within the last month. **Setting:** Northern California, USA. **Timing:** October 2005 to May 2008.
Interventions	Intervention (n = 96) Diet and exercise and breastfeeding intervention (DEBI). Delivered by a dietician using Social Cognitive Theory and Transtheoretical Model. Delivered prenatal, postpartum and maintenance based on 1-to-1 sessions and 2 individual telephone counselling sessions with a lifestyle coach. Advised not to exceed 11.4 kg for obese women and to follow ADA diet and moderate physical activity (150 min/week). Also had lactation consultant and contact maintained for 6 weeks postpartum. versus Control group (usual care) (n = 101) Printed material only in prenatal and postnatal period. The maintenance phase continued for 6 months.
Outcomes	Primary outcome - meeting postpartum weight goal. Secondary outcome - medication use, perinatal clinic visits, birthweight, macrosomia, physical activity, diet, breastfeeding, SGA. Trials registration document also lists plasma glucose levels, plasma insulin levels, markers of insulin resistance and adiponectin as additional outcomes not reported in the published papers.
Notes	Power calculation: not stated. ITT analysis: yes. Funding: National Institute of Diabetes and Digestive Kidney Diseases, Kaiser Garfield Foundation. Conflicts of interest: the authors report no potential conflicts of interest of relevance to the article in the Acknowledgements section of the manuscript. Follow-up of 72 women postpartum is reported by Erlich 2014 for those women in the intervention group who lost weight or did maintained/gained weight postpartum.

Risk of bias table

Bias	Authors' judgement	Support for judgement
Random sequence generation (selection bias)	Low risk	Randomly assigned. Computer-randomisation programme stratified for age, pregravid BMI.
Allocation concealment (selection bias)	Unclear risk	No details.
Blinding of participants and personnel (performance bias)	High risk	No blinding, open-label.
Blinding of outcome assessment (detection bias)	Low risk	Trials registration document indicates that outcome assessors were blinded.
Incomplete outcome data (attrition bias)	Low risk	97% follow-up to postpartum in the usual care group and 95% in the intervention group.
Selective reporting (reporting bias)	High risk	The main outcome was meeting postpartum weight gain. This is a pilot study and the full trial is yet to be reported on. There are very limited neonatal outcomes and additional outcomes are listed in the trial registration document that are not reported in the published papers.
Other bias	Low risk	There was no evidence of other bias.

Garner 1997

Methods	Randomised controlled pilot trial.
Participants	300 women from Canada. **Inclusion criteria:** diagnosis of gestational diabetes between 24 to 32 weeks' using 75 g glucose screen with 1-hour cut-off level of 8 mmol/L (Table 7). **Exclusion criteria:** multiple gestation, maternal foetal blood group incompatibility, known congenital anomaly, prior evidence of planta praevia/abruptio, significant maternal disease (chronic hypertension, connective tissue disease, endocrine disorders, chronic hepatic disease), long-term medical therapy affecting glucose metabolism such as steroids and beta-mimetic tocolytic agents, and imminent delivery. **Setting:** 2 teaching hospitals in Ottawa, Canada. **Timing:** September 1991 to May 1994.
Interventions	Tight versus minimal control. 1) Intervention group - Dietary counselling, calories restricted diet (35 kcal/kg/day), home glucose monitoring, if not controlled by diet alone then insulin supplementation, seen bi-weekly, ultrasound assessment of fetal growth, amniotic fluid volume and cardiac size. Aim to maintain blood glucose within the target range of < 4.4 mmol/L fasting and < 7.8 mmol/L 1-hour post-prandial (n = 149) versus 2) Control group (n = 150) - no dietary counselling but asked to continue unrestricted healthy diet for pregnancy as per Canada Food Guide. They were managed by the primary obstetric provider and were not seen again in the teaching unit. Treatment failures were transferred to the treatment arm of the trial and treated with diet/insulin/monitoring.
Outcomes	None were prespecified but reported on hyperbilirubinaemia, hypoglycaemia, fasting and postprandial blood glucose, hypocalcaemia, macrosomia, mortality, congenital anomaly, birth trauma, birthweight, weight gain in pregnancy and mode of delivery, gestational age at birth. Childhood BMI, cholesterol, blood glucose concentration.
Notes	Sample size calculation: yes. ITT analysis: yes, treatment failures in the control group who were moved to the intervention group were analysed in the control arm. Funding: no details. Conflicts of interest: there were no details on conflicts of interest published in the manuscript. The trial was followed up at 7 to 11 years by Keely (2008) for metabolic markers of insulin resistance in the offspring.

Risk of bias table

Bias	Authors' judgement	Support for judgement
Random sequence generation (selection bias)	Unclear risk	"randomly allocated" no other details.
Allocation concealment (selection bias)	Unclear risk	No details.
Blinding of participants and personnel (performance bias)	Low risk	"Health care workers in the control group were blinded to the blood glucose group."
Blinding of outcome assessment (detection bias)	Unclear risk	No details.
Incomplete outcome data (attrition bias)	Low risk	1 woman in the treatment arm of the trial was lost to follow-up.
Selective reporting (reporting bias)	High risk	Outcomes were not prespecified. An original protocol was not seen.
Other bias	Low risk	There is no evidence of other bias.

Gillen 2004

Methods	Single-centre randomised controlled trial.
Participants	32 women. No data provided on mean maternal age or ethnicity. **Inclusion criteria:** GDM diagnosed at approximately 28 weeks' gestation (Table 7). **Exclusion criteria:** significant other health concerns, poor English language skills. **Setting:** Diabetic clinic. Wollongong, Australia. **Timing:** May to December 2002.
Interventions	1) Intervention group: following a group session on management of GDM from a registered nurse diabetes educator and dietician the group received standard clinical practice plus advice for targeted intakes of foods rich in unsaturated fats based on meeting energy requirements. 2) Control group: following a group session on management of GDM from a registered nurse diabetes educator and dietician the group received standard clinical practice (individualised carbohydrate portion-controlled meal plan, with low-fat and low-glycaemic index dietary strategies and general advice about meeting nutritional requirements of pregnancy).
Outcomes	Outcomes: gestation at birth, mode of birth, changes in dietary intakes, use of insulin.
Notes	ITT analysis was used. not stated. Funding: not stated. Sample size calculation: not stated. Conflicts of interest: no details.

Risk of bias table

Bias	Authors' judgement	Support for judgement
Random sequence generation (selection bias)	Low risk	Open table of random numbers constructed by an independent person and kept confidential from members of the study team. Women were matched consecutively to the next available number in the table and the study team informed of the result.
Allocation concealment (selection bias)	Low risk	Randomisation done centrally.
Blinding of participants and personnel (performance bias)	Unclear risk	Participants unaware of differences in advice between intervention and control groups, research staff were aware.
Blinding of outcome assessment (detection bias)	Unclear risk	Not stated.
Incomplete outcome data (attrition bias)	Low risk	Dropouts: data not available for 1 woman from each group.
Selective reporting (reporting bias)	High risk	An original protocol was not seen. The outcomes were not clearly prespecified. The authors report that there were no differences in pregnancy outcomes or nature of birth but they do not report any of this data in the paper.
Other bias	Low risk	No evidence of other bias, no differences at baseline.

Jovanovic-Peterson 1989

Methods	Randomised controlled trial.
Participants	19 women **Inclusion criteria:** women diagnosed with GDM (Table 7). **Exclusion criteria:** none detailed. **Setting:** USA. **Timing:** not specified.
Interventions	Intervention group - 6 week diet (24 to 30 kcal/kg/24 hours; 20% protein, 40% carbohydrates, 40% fat divided into 3 meals and 3 snacks) plus 20 mins of supervised aerobic exercise 3 times per week for the 6 weeks. An arm ergometer was used to maintain heart rate (220-age in years) x 70% unless this heart rate exceeded 140 bpm and then 140 bpm was the target heart rate. The exercise session never exceeded 50% maximal oxygen consumption. 6 women exercised between 4 pm and 5 pm and 4 women between 10 am and 11 am. Control group - 6 week diet alone (24 to 30 kcal/kg/24 hours; 20% protein, 40% carbohydrates, 40% fat divided into 3 meals and 3 snacks). All women performed glucose self monitoring 4 times per day (before breakfast and 1 hour postprandial). Seen weekly by a physician. Insulin was started if FPG > 5.8 mmol/L or 105 mg/dL) and/or 1 hour postprandial plasma glucose was > 7.8 mmol/L or 140 mg/dL).
Outcomes	HbA1c, fasting and 1-hour postprandial blood glucose, 50 g glucose challenge test, maternal hypoglycaemia, C-peptide, use of insulin, birthweight. No primary outcomes were pre-specified.
Notes	Power calculation - not reported. ITT analysis - not reported. Funding - not reported. Conflicts of interest - not reported in the manuscript.

Risk of bias table

Bias	Authors' judgement	Support for judgement
Random sequence generation (selection bias)	Low risk	'Randomized', by drawing a number.
Allocation concealment (selection bias)	Unclear risk	No details.
Blinding of participants and personnel (performance bias)	High risk	No details but unlikely to have been blinded.
Blinding of outcome assessment (detection bias)	Unclear risk	No details.
Incomplete outcome data (attrition bias)	Low risk	Data reported for all 19 women randomised.
Selective reporting (reporting bias)	High risk	Additional outcomes are reported that were not listed a priori in the methods section including gestational age at delivery and birthweight.
Other bias	High risk	The women randomised to the exercise and diet intervention had a significantly higher 1 hour plasma glucose in the diagnostic test at baseline.

Kaviani 2014

Methods	Randomised controlled trial.
Participants	58 pregnant women with gestational diabetes (69 eligible). **Inclusion criteria:** having gestational diabetes (Table 7), being in the pre-diabetic stage, not using insulin and psychiatric medications, no previous history of relaxation therapy, age 18 to 40 years, gestational age 24 to 30 weeks'. **Exclusion criteria:** overt diabetes, unwillingness to co-operate at any stage of the study, being absent for more than 1 session of the training classes, not doing the relaxation exercises at home for more than 5 days, changing diet or physical activity during study, having pregnancy complications during study. **Setting:** Shiraz, Iran. **Timing:** February to April 2013.
Interventions	Intervention group (Relaxation training) (n = 29) over 10 weeks, five 45 min sessions - Session 1 Training on the nature and mechanism of diabetes, nature of stress and effect on body; Session 2 Different breathing techniques and body positions during relaxation; Session 3 How to relax muscles in various parts of the body after stress; Session 4 Relaxation through conditioning; Session 5 Training of differential relaxation and relaxation along with positive mental imagery (Based on the principles of Herbert Benson). Encouraged to practice relaxation at home for a month. Provided with a chart for recording relaxation exercises to evaluate their performance, CD with soft music explaining how do perform the relaxation. Contacted by telephone by the researcher 3 times per week. Control group (n = 29) routine prenatal care (no details).
Outcomes	BP, fasting blood sugar, 2-hour postprandial blood sugar, use of insulin.
Notes	Sample size calculation: yes but unclear on what outcome the calculation was based. ITT analysis: yes. Funding: University funding. Conflicts of interest: not reported in the manuscript.

Risk of bias table

Bias	Authors' judgement	Support for judgement
Random sequence generation (selection bias)	Low risk	'random numbers' and 'permutation blocks.'
Allocation concealment (selection bias)	Unclear risk	No details.
Blinding of participants and personnel (performance bias)	High risk	No details but unlikely to be blinded due to nature of intervention.
Blinding of outcome assessment (detection bias)	Unclear risk	No details.
Incomplete outcome data (attrition bias)	Low risk	58 women randomised and analysed.
Selective reporting (reporting bias)	High risk	Very limited outcomes reported for mother, no neonatal outcomes reported.
Other bias	Low risk	No differences at baseline.

Landon 2009

Methods	Randomised controlled trial.
Participants	958 women. **Inclusion criteria:** abnormal result on glucose loading test (Table 7). Between 24 and 30 weeks' gestation. **Exclusion criteria:** pregestational diabetes, abnormal glucose screen before 24 weeks', previous GDM, history of stillbirth, multiple gestation, asthma, chronic hypertension, taking corticosteroids, known fetal anomaly or imminent preterm delivery. Fasting glucose > 5.3 mmol/L (95 mg/dL). **Setting:** Obstetric research centre, Washington, USA. **Timing:** October 2002 to November 2007.
Interventions	1) Intervention group - Formal nutrition counselling and diet therapy +/- insulin and daily self monitoring (fasting and 2 hour post-prandial) (n = 485) versus 2) Control group - usual prenatal care +/- insulin and self monitoring (n = 473). Insulin was commenced if fasting glucose levels were predominantly at 5.3 mmol/L or greater or postprandial glucose was 6.7 mmol/L or greater.
Outcomes	Primary outcome was a composite score (perinatal mortality, hyperglycaemia, hypoglycaemia, hyperbilirubinaemia, neonatal hyperinsulinaemia, birth trauma). Secondary outcomes included individual components of the composite score, C-peptide, birthweight, preterm birth, macrosomia, LGA, SGA, neonatal glucose levels, neonatal hypoglycaemia, hyperbilirubinaemia, birth trauma, gestational age at birth, NICU admission, RDS, neonatal fat mass, adiposity, gestational weight gain, hypertension, pre-eclampsia, caesarean section, IOL, shoulder dystocia, maternal diabetes, use of insulin, metabolic syndrome. Childhood BMI.
Notes	Sample size calculation: yes based on composite score. ITT analysis: yes. Funding: Eunice Kennedy Shriver National Institute of Child Health and Human Development. Conflicts of interest: the authors reported that there were no potential conflicts of interest relevant to this manuscript. Casey 2015 reported on long-term maternal outcomes from this trial on 457 (50%) of the eligible 905 women. 243 women were treated in the original trial and 214 untreated. 430 women had blood drawn for analysis. Bahado-Singh 2012 reported on gender differences in fetal outcomes. Durnwald 2011 reported on glycaemic characteristics and neonatal outcomes but do not report on differences between treatment and intervention groups. Sutton 2014 reported on timing of delivery and caesarean section. Landon 2015 reported on long-term follow-up of children at 5 to 10 years of age.

Risk of bias table

Bias	Authors' judgement	Support for judgement
Random sequence generation (selection bias)	Low risk	'simple urn method.'
Allocation concealment (selection bias)	Low risk	'by the coordinating centre.'
Blinding of participants and personnel (performance bias)	High risk	Not possible to blind participants; staff were not blinded to allocation.
Blinding of outcome assessment (detection bias)	Low risk	Outcome assessors were blinded for some outcomes.
Incomplete outcome data (attrition bias)	Low risk	25 women in the intervention group were lost to follow-up (8 had no delivery data and 17 had missing laboratory data). 33 women in the control group were lost to follow-up (18 had no delivery data and 15 had missing laboratory data). ITT analysis was conducted.
Selective reporting (reporting bias)	Low risk	The original protocol was not seen. All outcomes appear to be reported and include maternal and neonatal outcomes. Long-term maternal outcomes are reported in abstract form by Casey 2015.
Other bias	Low risk	No evidence of other bias, no differences in baseline measurements between groups.

Mendelson 2008

Methods	Randomised trial.
Participants	100 Mexican-American women. Mean age in Parish nurse group was 30.6 ± 5.6 years and in the usual care group was 31.5 ± 5.2 years. **Inclusion criteria**: diagnosed and referred for treatment for gestational diabetes (Table 7), self-reported Mexican descent, able to speak, read and write in English or Spanish, 18 to 40 years of age, between 12 and 32 weeks' gestation, singleton pregnancy. **Exclusion criteria**: not specified. **Setting:** outpatient clinic for women with gestational diabetes in a hospital in California, USA. **Timing:** no details.
Interventions	Intervention group (Parish nurse intervention program) (n = 49) Enhanced education and support provided by parish nurses fluent in Spanish. A supplemental 1-hour Parish nurse led discussion regarding medical recommendations for control of gestational diabetes to clarify areas of concern or misunderstanding. Also included spiritual principles such as encouragement of prayer and spiritual connection within the belief system of the women. Education included what is diabetes, types and risk factors; diabetes control with nutrition, activity, and medical treatment; and nutrition therapy (food groups and measurements). Control group (Usual care) (n = 51) Education on diet, exercise, blood glucose testing and insulin administration if required in individual 1-hour sessions provided through handouts, demonstration and discussion.
Outcomes	Health promotions behaviour questionnaire, macrosomia, fasting blood glucose, random blood glucose, HbA1c, duration of maternal and neonatal hospitalisation, caesarean section, use of insulin. Primary outcomes were not specified.
Notes	Power calculation: no data provided. ITT analysis: not stated Funding: Eugene Cota Robles Fellowship. Conflicts of interest: not provided in the manuscript.

Risk of bias table

Bias	Authors' judgement	Support for judgement
Random sequence generation (selection bias)	Low risk	'random number tables.'
Allocation concealment (selection bias)	Unclear risk	No clear details provided despite contacting author.
Blinding of participants and personnel (performance bias)	Unclear risk	Women were not blinded (information obtained from email), diabetes educators were blinded to allocation.
Blinding of outcome assessment (detection bias)	Unclear risk	No details provided.
Incomplete outcome data (attrition bias)	High risk	Not all women had an HbA1c at birth (27 out of 100).
Selective reporting (reporting bias)	Unclear risk	The outcomes listed in the methods section were all reported in the results. Caesarean section which was not prespecified was reported as an outcome in the results.
Other bias	Low risk	No differences between groups at baseline. No evidence of other bias.

Rahimikian 2014

Methods	Randomised controlled trial.
Participants	126 women with gestational diabetes (diagnostic criteria unclear). **Inclusion criteria:** gestational age 28 to 36 weeks', hospitalised due to high blood sugar or gestational diabetes (Table 7), not attending education sessions before, literate. **Exclusion criteria:** absent for 1 of the training sessions, not wanting to continue with the study. **Setting:** Iran. **Timing:** 2013.
Interventions	Intervention group (Face-to-face education) (n = 42) 2 sessions of 40 mins as individuals or in groups. Session 1 definition of GDM, causes, symptoms, those at risk, management of GDM including training on glycaemic control; session 2 nutrition, physical activity and exercise, insulin, pregnancy follow-up. or (Instructional booklet education) (n = 42) a booklet provided that includes all the information given in the face-to-face sessions (not used in this review). Control (n = 42) routine hospital services (no details).
Outcomes	Maternal hospitalisation due to gestational diabetes and duration, type of delivery, use of insulin, birthweight, gestational age at birth, Apgar 1 and 5 mins, stillbirth.
Notes	Sample size calculation - no. ITT analysis - no. Funding - no details. Conflicts of interest - no details.

Risk of bias table

Bias	Authors' judgement	Support for judgement
Random sequence generation (selection bias)	Unclear risk	'randomly assigned.'
Allocation concealment (selection bias)	Unclear risk	No details.
Blinding of participants and personnel (performance bias)	High risk	No details but unlikely that participants or researchers were blinded.
Blinding of outcome assessment (detection bias)	Unclear risk	No details.
Incomplete outcome data (attrition bias)	Unclear risk	Data appear to be missing for 1 of the intervention groups but no details provided.
Selective reporting (reporting bias)	High risk	Raw data not reported for all outcomes.
Other bias	Low risk	No differences at baseline.

Yang 2003

Methods	Randomised controlled trial.
Participants	150 women randomised. **No details of inclusion or exclusion**. Women were diagnosed with GDM after a 50 g, 1-hour screen at 26 to 30 weeks (Table 7). **Setting:** Tianjin, China. **Timing:** no details of time that trial was conducted.
Interventions	1) Intervention group (Intensive care) (n = 95) Intensive Diabetes Management Plan - diet and exercise advice, self home blood glucose monitoring ± insulin if required. Fortnightly specialist review. Low calorie intake prescribed according to pre-gravid BMI. Goal: to achieve fasting capillary blood glucose < 5.5 mmol/L and 1 hour post prandial < 7.0 mmol/L versus 2) Control group (usual obstetric care) no details (n = 55).
Outcomes	Not prespecified but reported premature rupture of membranes, preterm birth, perinatal morbidity, caesarean section, birthweight, perinatal mortality, congenital anomaly, birth trauma, dystocia, use of insulin.
Notes	Power calculation: power analysis was performed but the variable was not reported. The sample size was estimated at 200 whereas only 100 were randomised. ITT analysis: state that used ITT for pregnancy outcomes. Funding: not stated.

Risk of bias table

Bias	Authors' judgement	Support for judgement
Random sequence generation (selection bias)	Unclear risk	'randomized' no other details.
Allocation concealment (selection bias)	Unclear risk	No details.
Blinding of participants and personnel (performance bias)	High risk	No details but unlikely that participants or researchers were blinded.
Blinding of outcome assessment (detection bias)	Unclear risk	Not reported.
Incomplete outcome data (attrition bias)	High risk	Of 95 women in the intervention group, only 48 completed the management plan compared with 55/55 in the usual care group.
Selective reporting (reporting bias)	High risk	The study is reported as a letter only. There was no evidence of a full paper and only the data on caesarean section and PROM is reported.
Other bias	High risk	Unable to establish if there are other biases due to lack of information. Power analysis was performed but the variable was not reported. The sample size was estimated at 200 whereas only 100 were randomised.

Yang 2014

Methods	Randomised trial.
Participants	948 women randomised. Mean age of usual care group was 29.7 ± 3.2 years and 29.9 ± 3.5 years in the shared care group. 97% were Han Chinese. GDM diagnosed by 50 g 1-hour glucose challenge test between 24 and 28 weeks' gestation (Table 7). **Exclusion criteria:** OGTT meeting criteria for diabetes (FPG ≥ 7.0 mmol/L, 2 hour ≥ 11.1 mmol/L or HbA1c ≥ 6.5%/48 mmol/mol); < 18 years of age, multiple pregnancy, maternal fetal ABO blood type incompatibility, maternal diseases such as chronic hypertension, thyrotoxicosis, pre-pregnancy diabetes and long-term use of medications that might affect glucose metabolism. **Setting:** Tianjin, China. **Timing:** December 2010 to October 2012.
Interventions	Intervention group (Shared care) (n = 344) - adapted from ACHOIS protocol. Intervention delivered by trained nurses and doctors. All women were offered individualised dietary advice and physical activity counselling. Different energy intakes were recommended based on prepregnancy BMI. All women were asked to engage in at least 30 mins of light to moderate physical activity daily. All women were offered a free glucose meter with memory function and free test strips. Asked to perform self-monitoring 4 times daily for first 2 weeks and then daily at different times in rotation. Glycaemic target was ≥ 3.5 to ≤ 5.1 mmol/L for fasting capillary glucose and ≤ 7.0 mmol/L for 2-hour post-prandial capillary glucose up to 36 weeks' gestation and ≤ 8.0 mmol/L from 36 weeks' onwards. If target levels were exceeded 2 or more times during a 2-week interval or the 2-hour postprandial level exceed 9.0 mmol/L once during a 1 week period then insulin was recommended. At 30 and 34 weeks' gestation the group was offered 2 additional individualised counselling sessions to reinforce diet, physical activity and self monitoring. They were also offered group education sessions lasting 2 hours at 27, 29 and 33 weeks' gestation. Control group (Usual care) (n = 362) - offered group education class lasting 30-40 mins delivered by a diabetes educator. Received advice on diet and physical activity but not specifically taught to self monitor blood glucose. Insulin treatment recommended if HbA1c ≥ 6.5% (48 mol/mol).
Outcomes	Primary - macrosomia (≥ 4000 g), LGA. Secondary - pregnancy-induced hypertension. Other outcomes included depression, caesarean section, use of insulin, weight gain in pregnancy, IOL, neonatal death, birth trauma, gestational age at birth, preterm birth, birthweight, birth length, neonatal hypoglycaemia, visits to health professional. Physical activity, food recall. Other outcomes were not reported a priori.
Notes	During Nov 2010 to July 2011 separate areas for intervention and follow-up in the 2 groups were unavailable due to building renovation and data collection for the usual care women was performed by the intervention staff members. The 242 women entering the trials during this period also received unintentional intervention. The authors excluded these women from the analysis. Power calculation: yes based on a reduction in risk for pregnancy-induced hypertension. ITT analysis: no, women wrongly allocated during a specific period were excluded from the analysis. Funding: funding from BRIDGES an educational grant from Lilly Diabetes. Conflicts of interest: a conflict was reported by 1 of the 12 authors.

Risk of bias table

Bias	Authors' judgement	Support for judgement
Random sequence generation (selection bias)	Low risk	'simple randomization procedure without replacement' 'computer generated random assignment'
Allocation concealment (selection bias)	Unclear risk	No details provided.
Blinding of participants and personnel (performance bias)	Low risk	'The women with GDM in the trial but not the research team members were masked to the random assignment.'
Blinding of outcome assessment (detection bias)	Low risk	Outcome assessors for pregnancy-induced hypertension were blinded to allocation.
Incomplete outcome data (attrition bias)	High risk	During Nov 2010 to July 2011 separate areas for intervention and follow-up in the 2 groups were unavailable due to building renovation and data collection for the usual care women was performed by the intervention staff members. The 242 women entering the trials during this period also received unintentional intervention. The authors excluded these women from the analysis. In addition to this 1 woman in the usual care group and 5 women in the shared care group gave birth outside Tianjin. 339 women in the usual care and 361 women in the shared care group were analysed.
Selective reporting (reporting bias)	High risk	Additional outcomes are reported that were not pre-specified in the methods section.
Other bias	Low risk	Groups were similar at baseline. No other risk of bias was identified.

Youngwanichsetha 2014

Methods	Randomised controlled trial.
Participants	180 women randomised. Mean maternal age in the intervention group was 32.58 ± 5.01 years and for the control group was 31.24 ± 4.54 years. No ethnicity is reported but women were Thai. **Inclusion criteria:** diagnosed with GDM A1 at 24 to 30 weeks' gestation (Table 7); not receiving insulin therapy for glycaemic control; having no serious complications such as gestational hypertension, pre-eclampsia, preterm labour or other serious health problems. **Exclusion criteria:** none detailed. **Setting:** tertiary hospital in Thailand. **Timing:** not specified.
Interventions	Intervention (n = 90) Trained to perform mindfulness eating and yoga exercise in 2 50-min sessions. Videos were used in classes and practicing manuals were offered for all the women to follow. Afterwards they were encouraged to continue with mindfulness eating and yoga at home 5 times a week for 8 weeks. Mindfulness eating involved setting a goal for blood glucose control, integrating medical nutrition therapy including carbohydrate choices and low glycaemic index food, considering portion size, being aware while consuming diabetic food, and eating slowly for 30 to 40 mins. The yoga that was used was yoga pranayama (deep breathing techniques) and asanas (posture and movements). it was designed for 15 to 20 mins daily practice for 5 days a week. The group were encouraged weekly by research staff. Control group (n = 90) Standard diabetes care (no details).
Outcomes	Primary: capillary fasting glucose and postprandial blood glucose and HbA1c, use of insulin.
Notes	Power calculation: yes based on an expected difference in glycaemic control. ITT analysis: no, they did not analyse the women who did not complete the study or who had been lost to follow-up. Funding: no details provided in manuscript. Conflicts of interest: no details provided in manuscript.

Risk of bias table

Bias	Authors' judgement	Support for judgement
Random sequence generation (selection bias)	Unclear risk	No details on method of randomisation.
Allocation concealment (selection bias)	Unclear risk	'randomization was performed by a research assistant using opaque envelopes technique'. Not clear if this was sequential or not.
Blinding of participants and personnel (performance bias)	High risk	No details provided but staff and participants are unlikely to have been blinded.
Blinding of outcome assessment (detection bias)	Low risk	HbA1c testing was conducted by a laboratory and the personnel are likely to have been blinded to allocation.
Incomplete outcome data (attrition bias)	Low risk	3 women in the intervention group moved to another town for work and did not complete the study and another 2 were lost to follow-up for the same reason, therefore 85 women were analysed. 5 women in the control group were lost to follow-up as they had moved to another town for work, therefore 85 were analysed.
Selective reporting (reporting bias)	High risk	The trial only reports on the effects on glycaemic control and no neonatal or other maternal outcomes are reported.
Other bias	Low risk	Groups appear balanced at baseline, no evidence of other bias.

Footnotes

BMI: body mass index
BP: blood pressure
BPM: beats per minute
CRP: C-reactive protein
CTG: cardiotocography
dL: decilitre
FPG: fasting plasma glucose
GDM: gestational diabetes mellitus
IOL: induction of labour
ITT: intention-to-treat
IV: intravenous
L: litre
LGA: large-for-gestational age
min: minute
NICU: neonatal intensive care unit
OGTT: oral glucose tolerance test
RDS: respiratory distress syndrome
SGA: small-for-gestational age

Characteristics of excluded studies

Abirami 2014

Reason for exclusion	After contacting the authors it was clarified that this was not a randomised controlled trial.

Adam 2014

Reason for exclusion	This is a randomised controlled trial of exercise interventions and belongs in the exercise for pregnant diabetic women review.

Bastani 2015

Reason for exclusion	This is a randomised trial using acupressure to treat anxiety in women with GDM and not being used for glycaemic control.

Berry 2013

Reason for exclusion	Although women are recruited in pregnancy with GDM, the main intervention starts at 6 weeks postpartum.

Bevier 1999

Reason for exclusion	Women did not meet the criteria for GDM as they only had an elevated glucose challenge test and a normal glucose tolerance test.

Bonomo 2005

Reason for exclusion	Women did not meet the criteria for GDM as they only had an elevated glucose challenge test and a normal glucose tolerance test.

Branch 2010

Reason for exclusion	This trial was registered but never started due to insufficient funding for enrolling participants.

Fadl 2015

Reason for exclusion	Wrong comparison for this review - insulin versus no additional treatment.

Ford 1997

Reason for exclusion	Trial compared diet alone versus usual care. Does not meet the review criteria for a lifestyle intervention.

Grant 2011

Reason for exclusion	Wrong comparison for this review. This trial is included in the Cochrane systematic review on different types of dietary advice for women with GDM.

Holmes 2012

Reason for exclusion	This is not a treatment trial for women with GDM.

Homko 2002

Reason for exclusion	Trial compared different types of monitoring of blood glucose - Wrong comparison.

Kitzmiller 1990

Reason for exclusion	Informed by author that trial never started (March 2006).

Langer 1989

Reason for exclusion	Trial compared diet alone versus usual care. Not meeting the review criteria for a lifestyle intervention.

Li 1987

Reason for exclusion	This trial randomised women to 2 different screening/diagnosis strategies. Wrong comparison.

Mirzamoradi 2015

Reason for exclusion	This trial randomised women to 2 different screening/diagnosis strategies. Wrong comparison.

O'Sullivan 1971

Reason for exclusion	Quasi-randomised, alternate allocation.

O'Sullivan 1974

Reason for exclusion	Primary outcome death. Allocation used an alternate method, no intention-to-treat analysis. Endpoints unclear.

O'Sullivan 1980

Reason for exclusion	Not randomised.

Osmundson 2015

Reason for exclusion	Randomised trial of treating women with prediabetes, screened at < 14 weeks' gestation. GDM was confirmed by screening at 26 to 28 weeks' if not on insulin. Therefore not a trial treating women with GDM.

Perichart-Perera 2009

Reason for exclusion	Quasi-experimental design with an historical control group.

Reader 2006

Reason for exclusion	This was an implementation trial for clinical practice guidelines rather than an intervention trial.

Rey 1997

Reason for exclusion	Study compared home monitoring of blood glucose with clinic follow-up. Wrong comparison.

Footnotes

GDM: gestational diabetes mellitus

Characteristics of studies awaiting classification

Cao 2012

Methods	States that groups were randomly allocated and also that the control group was age-matched to the intensive treatment group. Unclear if true randomisation took place.
Participants	Pregnant women with GDM.
Interventions	A comprehensive intensive individualised therapy including education, dietary and exercise advice and instructions on self monitoring. Monitored by physician every 2 weeks. Standard group received group education on diet and exercise, self-monitoring information given but not required at the same frequency as intensive group.
Outcomes	Caesarean section, pre-eclampsia, postpartum complications, birthweight, stillbirth, jaundice, neonatal death, admission to NICU, preterm delivery, congenital malformation, neonatal hypoglycaemia. Later follow-up maternal diabetes and metabolic syndrome.
Notes	Contact author emailed 20/01/2015.

Kaveh 2012

Methods	Randomised controlled trial.
Participants	Women with GDM.
Interventions	Educational intervention with nutrition and exercise versus control.
Outcomes	Fasting and postprandial glucose levels; knowledge.
Notes	The article is in Persian and we are awaiting a translation to confirm inclusion/exclusion in this review.

Zhang 2012

Methods	'randomly divided.'
Participants	Women with gestational diabetes.
Interventions	Health education intervention with nutrition, exercise and foot care versus standard care.
Outcomes	Self-efficacy, blood glucose levels.
Notes	Translation required to determine if true randomisation and if intervention and control groups meet inclusion criteria for this review.

Footnotes

GDM: gestational diabetes mellitus
NICU: neonatal intensive care unit

Characteristics of ongoing studies

Durnwald NCT01858233

Study name	The IBEP Study: an intervention for lifestyle modification in women with gestational diabetes.
Methods	Randomised open-label trial.
Participants	120 women with GDM between 20 and 34 weeks' gestation.
Interventions	Intensive behavioural modification, dietary counselling, lactation counselling versus routine care and standard dietary counselling.
Outcomes	Primary outcomes: OGTT at 6 weeks postpartum. Secondary outcomes: weight loss and lipid profiles.
Starting date	November 2012.
Contact information	Valerie.armendariz@uphs.upenn.edu
Notes	

Ferrara NCT01489163

Study name	Lifestyle intervention program for women with gestational diabetes or gestational impaired glucose tolerance (APPLES).
Methods	Randomised double-blind trial.
Participants	350 women with pregnancy complicated by high glucose levels.
Interventions	Lifestyle counselling versus no intervention.
Outcomes	Primary - postpartum body weight. Secondary - proportion of women reaching body weight goals, percent of calories from fat, time spent in physical activity, postpartum glycaemia.
Starting date	December 2011.
Contact information	Assiamira Ferrara - Kaiser Permanente. USA.
Notes	

Hoseinzadeh IRCT2014080418682N1

Study name	The effects of an educational intervention based on the theory of planned behavior on self-care behavior and blood glucose levels in pregnant women with gestational diabetes treated with insulin.
Methods	Randomised open-label trial ongoing in Iran.
Participants	60 pregnant women with gestational diabetes treated with insulin gestational age 20 to 24 weeks'.
Interventions	Education based on the theory of planned behaviour performed with 4 sessions of 60 minutes duration versus routine prenatal care.
Outcomes	Primary outcome - self-care behaviour. Secondary outcomes - fasting and 2-hour post-prandial blood glucose level.
Starting date	2014.
Contact information	HoseinzadehM911@mums.ac.ir
Notes	

Mirfeizi IRCT201406022892N3

Study name	The effect of self-care education on quality of life in women with gestational diabetes.
Methods	Randomised controlled trial.
Participants	240 women with gestational diabetes from Iran. Singleton pregnancy, 20 to 24 weeks' gestation.
Interventions	4, 45-minute group education sessions for a month in self-care versus routine prenatal care.
Outcomes	Primary outcome - quality of life.
Starting date	2014.
Contact information	latibari@kiau.ac.ir; mani@kiau.ac.ir
Notes	

Roeder NCT01926457

Study name	Treating prediabetes in the first trimester.
Methods	Randomised controlled trial, single-blind.
Participants	240 women from USA diagnosed with pre-diabetes < 15 weeks' gestational age.
Interventions	First trimester diabetes education, blood glucose monitoring, medication if required, growth ultrasounds, antenatal testing versus third trimester diabetes education, blood glucose monitoring, medication if required, growth ultrasounds, antenatal testing.
Outcomes	Primary outcomes - cord C-peptide. Secondary outcomes - neonatal fat mass, gestational weight gain, return to pre-pregnancy weight, maternal adiponectin, birthweight, LGA, ponderal index, admission to NICU, infant weight-for-length, need for maternal pharmacotherapy, birth trauma, mode of birth, gestational weight gain, postpartum weight retention, pre-eclampsia.
Starting date	2013.
Contact information	haroeder@ucsd.edu
Notes	

Sahnaz IRCT2014042017346N1

Study name	Effectiveness of stress management with cognitive behavioural method on blood sugar levels and stress among patient with gestational diabetes.
Methods	Randomised controlled trial.
Participants	Iranian trial in progress. Pregnant women with gestational diabetes, age 18 to 45 years, gestational age 24 to 32 weeks'.
Interventions	Stress management training with 6, 2-hour sessions of cognitive-behavioural group-based treatment versus routine prenatal care.
Outcomes	Primary outcomes - fasting blood sugar, stress. Secondary outcomes - anxiety, depression.
Starting date	2014.
Contact information	najarshanaz@yahoo.com
Notes	

Ziegler DRKS00000465

Study name	MuKiS - Mother-child sports - a study to evaluate the impact of exercise on maternal metabolism and fetal development in women with gestational diabetes.
Methods	Randomised controlled trial, open-label.
Participants	60 women with gestational diabetes in Munich, Germany. Age > 18 years, 24 to 30 weeks' gestation.
Interventions	Supervised physical activity twice a week for 45 minutes including walking and bicycle ergometry plus diet as recommended by the German Diabetes Society versus diet therapy alone.
Outcomes	Participation rates, mood, cardiovascular measurements, maternal biomarkers, fetal abdominal circumference, polyhydramnios, caesarean section rate, birthweight, macrosomia, cardiac hypertrophy.
Starting date	2009.
Contact information	anziegler@lrz.uni-muenchen.de; Lydia.Henneberger@lrz.uni-muenchen.de
Notes	

Footnotes

LGA: large-for-gestational age
OGTT: oral glucose tolerance test
GDM: gestational diabetes mellitus
NICU: neonatal intensive care unit

Summary of findings tables

1 Lifestyle interventions versus control - Maternal outcomes

Lifestyle interventions versus usual care or diet alone for the treatment of women with gestational diabetes

Patient or population: Women with gestational diabetes

Settings: UK, Italy, Australia, Canada, United Arab Emirates, China
Intervention: Lifestyle intervention
Comparison: Usual care or diet alone

Outcomes	Anticipated absolute effects* (95% CI)		Relative effect (95% CI)	№ of participants (studies)	Quality of the evidence (GRADE)	Comments
	Risk with usual care/control	Risk with lifestyle intervention				
Hypertensive disorders of pregnancy (pre-eclampsia)	129 per 1000	90 per 1000 (51 to 157)	RR 0.70 (0.40 to 1.22)	2796 (4 RCTs)	⊕⊕⊖⊖ LOW [1]	
Perineal trauma/tear	498 per 1000	518 per 1000 (463 to 588)	RR 1.04 (0.93 to 1.18)	1000 (1 RCT)	⊕⊕⊕⊖ MODERATE [5]	
Caesarean section	380 per 1000	342 per 1000 (296 to 399)	RR 0.90 (0.78 to 1.05)	3545 (10 RCTs)	⊕⊕⊖⊖ LOW [2] [3]	
Induction of labour	211 per 1000	252 per 1000 (220 to 285)	RR 1.20 (0.99 to 1.46)	2699 (4 RCTs)	⊕⊕⊕⊕ HIGH	
Postnatal depression	169 per 1000	83 per 1000 (53 to 132)	RR 0.49 (0.31 to 0.78)	573 (1 RCT)	⊕⊕⊖⊖ LOW [5] [7]	
Postnatal weight retention or return to pre-pregnancy weight	214 per 1000	375 per 1000 (225 to 621)	RR 1.75 (1.05 to 2.90)	156 (1 RCT)	⊕⊕⊖⊖ LOW [5] [6]	These data refer to women meeting postpartum weight goals at 12 months postpartum
Development of type 2 diabetes (follow-up)	83 per 1000	81 per 1000 (45 to 146)	RR 0.98 (0.54 to 1.76)	486 (2 RCTs)	⊕⊕⊖⊖ LOW [4] [7]	

***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; **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

Footnotes

[1] Evidence of inconsistency with I^2 > 70%, downgraded 2 levels

[2] Evidence of selective reporting in more than half of the trials reporting this outcome - downgraded 1 level

[3] Evidence of inconsistency with I^2 = > 50% but < 70%, downgraded 1 level

[4] Evidence of risk of bias with one of the two studies not blinding participants/researchers, downgraded 1 level

[5] Imprecision - Evidence is based on a single trial, downgraded 1 level

[6] Evidence of risk of bias - Allocation concealment unclear and no blinding of participants/researchers, downgraded 1 level

[7] Evidence of risk of bias - attrition bias, downgraded 1 level

2 Lifestyle versus control - Neonatal and later outcomes

Lifestyle interventions versus usual care or diet alone for the treatment of women with gestational diabetes

Patient or population: Women with gestational diabetes

Settings: UK, Italy, Australia, United Arab Emirates, Canada, China, USA
Intervention: Lifestyle intervention
Comparison: Usual care or diet alone

Outcomes	Anticipated absolute effects* (95% CI)		Relative effect (95% CI)	№ of participants (studies)	Quality of the evidence (GRADE)	Comments
	Risk with usual care/control	Risk with lifestyle intervention				
Large-for-gestational age	189 per 1000	113 per 1000 (95 to 134)	RR 0.60 (0.50 to 0.71)	2994 (6 RCTs)	⊕⊕⊕⊖ MODERATE[9]	
Perinatal (fetal and neonatal death) and later infant mortality	5 per 1000	0 per 1000 (0 to 9)	RR 0.09 (0.01 to 1.70)	1988 (2 RCTs)	⊕⊕⊖⊖ LOW [2 4]	Analysis refers to perinatal death only. No data were reported for later infant mortality
Composite outcome in infant (death, shoulder dystocia, nerve palsy, bone fracture)	193 per 1000	110 per 1000 (41 to 299)	RR 0.57 (0.21 to 1.55)	1930 (2 RCTs)	⊕⊖⊖⊖ VERY LOW [1 2 5]	
Neonatal hypoglycaemia	75 per 1000	74 per 1000 (49 to 114)	RR 0.99 (0.65 to 1.52)	3000 (6 RCTs)	⊕⊕⊕⊖ MODERATE [6]	
Adiposity (neonatal) - Neonatal fat mass (g)	The mean neonatal fat mass was 427 g	The mean neonatal fat mass in the intervention group was 37.30 g fewer (63.97 fewer to 10.63 fewer)	-	958 (1 RCT)	⊕⊕⊖⊖ LOW [3 7]	
Adiposity (child) - Childhood BMI > 85th percentile	350 per 1000	318 per 1000 (262 to 388)	RR 0.91 (0.75 to 1.11)	767 (3 RCTs)	⊕⊕⊕⊖ MODERATE [8]	
Adiposity (adult) not measured	see comment	see comment	not estimable			None of the included trials pre-specified adult adiposity as an outcome
Diabetes (type 2) (child) - not measured	see comment	see comment	not estimable			None of the included trials pre-specified childhood diabetes (type 2) as an outcome
Diabetes (type 2) (adult) - not measured	see comment	see comment	not estimable			None of the included trials pre-specified adulthood diabetes (type 2) as an outcome
Neurosensory disability (child) - not measured	see comment	see comment	not estimable			None of the included trials pre-specified childhood neurosensory disability as an outcome
Neurosensory disability (adult) - not measured	see comment	see comment	not estimable			None of the included trials pre-specified adulthood neurosensory disability as an outcome

*__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; **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

Footnotes

[1] Evidence of inconsistency with I^2 > 70%, downgraded 2 levels

[2] 1 of the 2 studies did not blind participants/researchers, downgraded 1 level

[3] Imprecision. Evidence is based on a single trial, downgraded 1 level

[4] There is evidence of imprecision with wide confidence intervals and low event rates, downgraded 1 level

[5] Evidence of imprecision with wide confidence intervals crossing the line of no effect, downgraded 1 level

[6] Allocation concealment was unclear in 2/6 trials and blinding was not undertaken in 2/6 trials, downgraded 1 level

[7] There was no blinding of researchers/participants in this single trial, downgraded 1 level

[8] Allocation concealment and randomisation was unclear in 1/3 trials and 1/3 trials did not blind participants/researchers, downgraded 1 level

[9] Several of the included studies had high risk of bias for lack of blinding, incomplete outcome data and selective reporting, allocation concealment was unclear in 2 of the 6 studies. Downgraded 1 level.

Additional tables

1 Examples of diagnostic criteria for gestational diabetes mellitus

Organisation/professional body	Screening criteria	Diagnostic criteria				
	1-hour oral glucose challenge test	Oral glucose tolerance test	Fasting	One hour	Two hour	Three hour
ADA 2015b*, IADPSG 2010 *, ADIPS 2014* (Nankervis 2014);	-	75 g	≥ 5.1 mmol/L (≥ 92 mg/dL)	≥ 10 mmol/L (≥ 180 mg/dL)	≥ 8.5 mmol/L (≥ 153 mg/dL)	-
WHO 2013*		75 g	5.1-6.9 mmol/L (92 -125 mg/dl)	≥ 10 mmol/L (≥ 180 mg/dL)	8.5-11.0 mmol/L (153 -199 mg/dl)	
ADA 2015b	50 g (≥ 7.8 mmol/L; ≥ 140 mg/dL)	75 g	≥ 5.1 mmol/L (≥ 92 mg/dL)	≥ 10 mmol/L (≥ 180 mg/dL)	≥ 8.5 mmol/L (≥ 153 mg/dL)	-
ACOG 2013 Carpenter and Coustan^ National Diabetes Data Group^	50 g (> 7.2 mmol/L; > 130 mg/dL)	100 g	≥ 5.3 mmol/L (95 mg/dL)	≥ 10 mmol/L (180 mg/dL)	≥ 8.6 mmol/L (155 mg/dL)	≥ 7.8 mmol/L (140 mg/dL)
	50 g (> 7.8 mmol/L; > 140 mg/dL)	100 g	≥ 5.8 mmol/L (105 mg/dL)	≥ 10.6 mmol/L (190 mg/dL)	≥ 9.2 mmol/L (165 mg/dL)	≥ 8.0 mmol/L (145 mg/dL)
ADIPS 1998 (Hoffman 1998)		75 g	≥ 5.5 mmol/L (≥ 99 mg/dL)		≥ 8.0 mmol/L (≥ 144 mg/dL)	
WHO 1999*		75 g	≥ 7.0 mmol/L (≥ 126 mg/dL)	-	≥ 7.8 mmol/L (140 mg/dL)	
NICE 2015	-	75 g	≥ 5.6 mmol/L (≥ 101 mg/dL)	-	≥ 7.8 mmol/L (140 mg/dL)	-
New Zealand Ministry of Health 2014*	50 g if HbA1c < 41 mmol/mol (≥ 7.8 mmol/L; ≥ 140 mg/dL)	75 g	≥ 5.5 mmol/L (≥ 99 mg/dL)	-	≥ 9.0 mmol/L (≥ 162 mg/dL)	-

Footnotes

ADA American Diabetes Association (recommends either the one step or two step strategy)

IADPSG International Association of the Diabetes and Pregnancy Study Groups

ADIPS Australasian Diabetes in Pregnancy Society

ACOG American College of Obstetrics and Gynecology

NICE National Institute for Health and Care Excellence

*1 abnormal result required for diagnosis

^2 or more abnormal results required for diagnosis

2 Maternal age (years)

Study ID	Lifestyle intervention	Usual care or diet alone
Bancroft 2000	Not stated	Not stated
Bo 2014	Exercise 35.9 ± 4.8 (n = 51) Behaviour 35.1 ± 4.4 (n = 49) Behaviour/exercise/diet 35.5 ± 4.4 (n = 50)	Diet 33.9 ± 5.3 (n = 50)
Crowther 2005	30.9 ± 5.4 (n = 490)	30.1 ± 5.5 (n = 510)
Elnour 2008	31.1 (95% CI 30.2 to 32.1)	30.7 (95% CI 29.4 to 32)
Ferrara 2011	Not stated	Not stated
Garner 1997	30.7 ± 4.8 (n = 149)	30.7 ± 4.6 (n = 150)
Gillen 2004	Not stated	Not stated
Jovanovic-Peterson 1989	31.1 ± 2.8 (n = 9)	29.5 ± 2.5 (n = 10)
Kaviani 2014	Not stated	Not stated
Landon 2009	29.2 ± 5.7 (n = 485)	28.9 ± 5.6 (n = 473)
Mendelson 2008	30.6 ± 5.6 (n = 49)	31.5 ± 5.2 (n = 51)
Rahimikian 2014	30.9 ± 5.7 (n = 42) face to face 30.4 ± 5.5 (n = 42) booklet	30.1 ± 5.8 (n = 42)
Yang 2003	Not stated	Not stated
Yang 2014	29.9 ± 3.5 (n = 339)	29.73 ± 3.2 (n = 361)
Youngwanichsetha 2014	32.58 ± 5.01 (n = 85)	31.24 ± 4.54 (n = 85)

Footnotes

Bo 2014; Jovanovic-Peterson 1989 compared a lifestyle intervention with diet alone

3 Maternal BMI at trial entry (kg/m2)

Study ID	Lifestyle intervention	Usual care or diet alone
Bancroft 2000	31.2 ± 6.7 (n = 32)	27.5 ± 6.1 (n = 36)
Bo 2014	Exercise 27.7 ± 4.3 (n = 49) Behaviour 27.5 ± 4.4 (n = 49) Behaviour/exercise/diet 27.5 ± 3.9 (n = 50)	Diet 27.5 ± 4.5 (n = 50)
Crowther 2005	Median 26 (IQR 23.3 to 31.2) (n = 490)	Median 26 (IQR 22.9 to 30.9) (n = 510)
Elnour 2008	Not stated	Not stated
Ferrara 2011	Not stated but 57% had BMI > 30	Not stated but 53% had BMI > 30
Garner 1997	Not stated	Not stated
Gillen 2004	Not stated	Not stated
Jovanovic-Peterson 1989	Not stated	Not stated
Kaviani 2014	Not stated	Not stated
Landon 2009	30.1 ± 5.0 (n = 485)	30.2 ± 5.1 (n = 473)
Mendelson 2008	Not stated	Not stated
Rahimikian 2014	Not stated	Not stated
Yang 2003	Not stated	Not stated
Yang 2014	22.9 ± 3.6 (n = 339)	23.4 ± 3.9 (n = 361)
Youngwanichsetha 2014	27.09 ± 3.56 (n = 85)	27.05 ± 4.06 (n = 85)

Footnotes

Bo 2014; Jovanovic-Peterson 1989 compared a lifestyle intervention with diet alone

4 Ethnicity/Race

Study ID	Ethnicity/Race
Bancroft 2000	31% of women were Asian and 69% were Caucasian
Bo 2014	Not stated
Crowther 2005	76% were Caucasian and 17% were Asian
Elnour 2008	UAE national
Ferrara 2011	52% were Asian or Pacific Islander; 19% were non-Hispanic Caucasian and 19% were Hispanic
Garner 1997	Not stated
Gillen 2004	Not stated
Jovanovic-Peterson 1989	Not stated
Kaviani 2014	Persian
Landon 2009	11.5% Black, 25% Caucasian, 5% Asian, 56.5% Hispanic
Mendelson 2008	Mexican
Rahimikian 2014	Not stated
Yang 2003	Not stated - probably Chinese
Yang 2014	97% Chinese Han
Youngwanichsetha 2014	Thai

Footnotes

Bo 2014; Jovanovic-Peterson 1989 compared a lifestyle intervention with diet alone

5 Gestation at trial entry (weeks)

Study ID	Lifestyle intervention	Usual care or diet alone
Bancroft 2000	31 (range 24 to 38) (n = 32)	32 (range 15 to 37) (n = 36)
Bo 2014	Not stated	Not stated
Crowther 2005	Median 29.1 (IQR 28.2 to 30.0) (n = 490)	Median 29.2 (IQR 28.2 to 30.0) (n = 510)
Elnour 2008	< 20 weeks'	< 20 weeks'
Ferrara 2011	31 ± 5.6 (n = 96)	31.0 ± (n = 6.1)
Garner 1997	Not stated	Not stated
Gillen 2004	Not stated	Not stated
Jovanovic-Peterson 1989	Not stated	Not stated
Kaviani 2014	Not stated	Not stated
Landon 2009	28.8 ± 1.6 (n = 485)	28.9 ± 1.5 (n = 473)
Mendelson 2008	Not stated	Not stated
Rahimikian 2014	Not stated	Not stated
Yang 2003	Not stated	Not stated
Yang 2014	Not stated	Not stated
Youngwanichsetha 2014	Not stated	Not stated

Footnotes

Bo 2014; Jovanovic-Peterson 1989 compared a lifestyle intervention with diet alone

6 Treatment target

Study ID	Treatment target
Bancroft 2000	Insulin was introduced if 5 or more capillary measurements > 7.0 mmol/L (126 mg/dL) in 1 week
Bo 2014	Treatment glycaemic targets were not detailed but insulin was started in the presence of fetal abdominal ultrasound > 70th percentile and or maternal hyperglycaemia (no details)
Crowther 2005	Fasting glucose levels 3.5 mmol/L (63 mg/dL) to 5.5 mmol/L (99 mg/dL), pre-prandial levels of no more than 5.5 mmol/L (99 mg/dL), and levels 2 hours post-prandially that were no more than 7.0 mmol/L (126 mg/dL)
Elnour 2008	Not stated
Ferrara 2011	Not stated
Garner 1997	Fasting glucose levels < 4.4 mmol/L (80 mg/dL); 1-hour post-prandial < 7.8 mmol/L (140 mg/dL)
Gillen 2004	Not stated
Jovanovic-Peterson 1989	Fasting plasma glucose ≤ 5.8 mmol/L or 105 mg/dL and/or 1-hour post-prandial plasma glucose was ≤ 7.8 mmol/L (140 mg/dL)
Kaviani 2014	Not stated
Landon 2009	Fasting glucose levels < 5.3 mmol/L, 2-hour post-prandial glucose < 6.7 mmol/L
Mendelson 2008	Not stated
Rahimikian 2014	Not stated
Yang 2003	< 5.5 mmol/L (99 mg/dL) fasting; < 7.0 mmol/L (126 mg/dL) 1.5 hours postprandial
Yang 2014	≥ 3.5 to ≤ 5.1 mmol/L for fasting capillary glucose and ≤ 7.0 mmol/L (126 mg/dL) for 2-hour post-prandial capillary glucose up to 36 weeks' gestation and ≤ 8.0 mmol/L from 36 weeks' onwards
Youngwanichsetha 2014	Not stated

Footnotes

Bo 2014; Jovanovic-Peterson 1989 compared a lifestyle intervention with diet alone

7 Details of diagnosis

Study ID	Timing	Screening	Diagnosis	
Bancroft 2000	Not stated	Not stated	75 g OGTT: fasting ≥ 7.0 mmol/L; 2 hour 7.8 to 11.0 mmol/L	WHO 1999
Bo 2014	24 to 26 weeks'	Not stated	75 g OGTT no further details	No details
Crowther 2005	24 to 34 weeks'	50 g 1-hour glucose challenge at least 7.8 mmol/L (140 mg/dL)	75 g OGTT plasma glucose level was less than 7.8 mmol/L and 2-hour value was 7.8 to 11.0 mmol/L (198 mg/dL)	WHO 1999
Elnour 2008	24 and 28 weeks'	50 g 1-hour glucose challenge, serum value > 7.2 mmol/L or plasma value > 7.8 mmol/L or risk factors present	100 g OGTT diagnosis if 2 or more values are abnormal from fasting ≥ 5.3 mmol/L, 1-hour value ≥ 10.0 mmol/L, 2-hour value ≥ 8.7 mmol/L, 3-hour value ≥ 7.8 mmol/L	Carpenter and Coustan criteria
Ferrara 2011	Not stated	50 g 1-hour glucose challenge	100 g OGTT; 3-hour	ADA (2000) criteria
Garner 1997	24 to 32 weeks'	75 g 1-hour > 8 mmol/L	75 g OGTT > 7.5 mmol/L (2nd trimester) and > 9.6 mmol/L (3rd trimester)	Hatem 1988
Gillen 2004	28 weeks'	50 g 1-hour venous plasma glucose level ≥ 7.8 mmol/L or 75 g hour venous plasma glucose level ≥ 8.0 mmol/L	75 g OGTT plasma glucose level at fasting of ≥ 5.5 mmol/L and/or at 2 hours of ≥ 8.0 mmol/L	ADIPS 1998
Jovanovic-Peterson 1989	Not stated	50 g 1-hour glucose challenge	Fasting and 1-hour tolerance test but no other details provided	No details
Kaviani 2014	Not stated	Not stated	Not stated	Not stated
Landon 2009	24 to 30 weeks'	50 g 1-hour glucose challenge 5.3 to 11 mmol/L	100 g OGTT; 2 or more of results was abnormal in addition to the abnormal challenge test (fasting < 5.3 mmol/L, 1-hour > 10.0 mmol/L, 2-hours > 8.6 mmol/L, 3-hours > 7.8 mmol/L	Carpenter and Coustan criteria
Mendelson 2008	Not stated	Not stated	Not stated	Not stated
Rahimikian 2014	Not stated	Not stated	Not stated	Not stated
Yang 2003	26 to 30 weeks'	50 g, 1-hour glucose challenge, ≥ 7.8 mmol/L	75 g, 2-hour OGTT; fasting ≥ 7.0 mmol/L, or 2-hour glucose ≥ 7.8 and ≤ 11.1 mmol/L	WHO criteria
Yang 2014	24 to 28 weeks'	50 g, 1-hour glucose challenge, ≥ 7.8 mmol/L	75 g, 2-hour OGTT; fasting ≥ 5.1 mmol/L, or 1-hour glucose ≥ 10.0 mmol/L or 2-hour glucose ≥ 8.5 mmol/L	IADPSG criteria
Youngwanichsetha 2014	24 to 30 weeks'	Not stated	Not stated	Not stated

Footnotes

OGTT: oral glucose tolerance test

8 Other maternal outcomes

Study ID	Outcome	Lifestyle	Usual care
Bancroft 2000	Postnatal fasting glucose (mmol/L)	Median 4.5 (range 2.7 - 5.9) n = 28	Median 4.4 (range 2.4 -8.8) n = 28
	Postnatal post prandial 2-hour (mmol/L)	Median 5.1 (range 2.1-8.5) n = 28	Median 5.5 (range 3.0-13.7) n = 28
Landon 2009	Maternal BMI (kg/m2) at follow-up	Mean 29.4 (95% CI 28.6 to 30.3) n = 243	Mean 29.1 (95% CI 28.2 to 30.0) n = 214
Garner 1997	Maternal BMI (kg/m2) at follow-up	BMI median 27.3 (range 19.4 to 50.5)	BMI median 29.6 (21.3 to 49.1)
Garner 1997	Maternal fasting glucose at follow-up	Fasting glucose median 5.4 (range 4.4 to 7.8) mmol/L	Fasting glucose median 5.5 (range 4.8 to 17.6) mmol/L

Footnotes

9 Neonatal outcomes

Study ID	Outcome	Lifestyle intervention	Ususal care or diet alone
Crowther 2005	Gestational age at birth	Median 39 weeks (IQR 38.1 -40.0) (n = 490)	Median 39.3 (IQR 38.3 -40.4) (n = 510)
Jovanovic-Peterson 1989	Gestational age at birth	Range 39.5 to 40.5 weeks	Range 39.4 to 40.0 weeks

Footnotes

Jovanovic-Peterson 1989 compared a lifestyle intervention with diet alone

10 Health service use

Study ID	Type of health service use	Lifestyle intervention	Usual care
Bancroft 2000	Number of capillary blood tests	Median 118 (range 0-520); n = 32	Median 0 (range 0); n = 36
Bancroft 2000	Number of antenatal visits	Median 17 (range 2-28); n = 32	Median 14 (range 6-33); n = 36
Crowther 2005	Number of antenatal clinic visits after enrolment	Median 5.0 (IQR 1-7) n = 490	Median 5.2 (IQR 3-7) n = 510
Crowther 2005	Number of physician clinic visits after enrolment	Median 3 (IQR 1-7) n = 490	Median 0 (IQR 0-2) n = 510
Bancroft 2000	Number of hospital admissions	Median 1 (range 0-6); n = 32	Median 0 (range 0-8); n = 36
Crowther 2005	Duration of stay in neonatal nursery	Median 1 day (IQR 1-2) n = 506	Median 1 day (IQR 1-3) n = 524
Crowther 2005	Length of postnatal stay (mother)	Median 4 days (IQR 3-5) n = 490	Median 4 days (IQR 3-5) n = 510
Mendelson 2008	Hospitalisation (days) (mother)	Mean 3.3, no SD provided	3.3, no SD provided
Mendelson 2008	Hospitalisation (days) (infant)	Mean 3.4, no SD provided	3.3, no SD provided

Footnotes

IQR: interquartile range

11 Cost

Crowther 2005	Lifestyle intervention	Usual care
Package of treatment for mild GDM versus usual care		
Direct costs per 100 women with a singleton pregnancy - including antenatal clinic visits, specialist clinics, dietician, diabetes educator, insulin therapy	AUD67,432	AUD33,681
In-patient costs - hospital costs	AUD545,125	AUD524,891
Total direct health service costs	AUD612,557	AUD558,572
Patient/family costs	AUD36,749	AUD30,229

Footnotes

These data are in the publication by Moss (2007)

References to studies

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Bancroft 2000

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Elnour 2008

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Classification pending references

Data and analyses

1 Lifestyle intervention versus usual care/control

Outcome or Subgroup	Studies	Participants	Statistical Method	Effect Estimate
1.1 Hypertensive disorders of pregnancy (including pre-eclampsia, pregnancy-induced hypertension, eclampsia)	4	2796	Risk Ratio(M-H, Random, 95% CI)	0.70 [0.40, 1.22]
1.1.1 World Health Organization 1999	1	1000	Risk Ratio(M-H, Random, 95% CI)	0.65 [0.48, 0.88]
1.1.2 ADA 2013	2	1096	Risk Ratio(M-H, Random, 95% CI)	0.44 [0.18, 1.06]
1.1.3 IADPSG 2010	1	700	Risk Ratio(M-H, Random, 95% CI)	2.40 [1.06, 5.44]
1.2 Caesarean section	10	3545	Risk Ratio(M-H, Random, 95% CI)	0.90 [0.78, 1.05]
1.2.1 WHO 1999/ADIPS 1998	4	1250	Risk Ratio(M-H, Random, 95% CI)	0.89 [0.78, 1.02]
1.2.2 ADA 2013	2	1096	Risk Ratio(M-H, Random, 95% CI)	0.63 [0.33, 1.22]
1.2.3 IADPSG 2010	1	700	Risk Ratio(M-H, Random, 95% CI)	1.09 [0.99, 1.21]
1.2.4 Other/not specified	3	499	Risk Ratio(M-H, Random, 95% CI)	0.94 [0.65, 1.38]
1.3 Development of type 2 diabetes	2	486	Risk Ratio(M-H, Fixed, 95% CI)	0.98 [0.54, 1.76]
1.4 Perinatal (fetal and neonatal death) and later infant mortality	2	1988	Risk Ratio(M-H, Fixed, 95% CI)	0.09 [0.01, 1.70]
1.5 Large-for-gestational age	6	2994	Risk Ratio(M-H, Fixed, 95% CI)	0.60 [0.50, 0.71]
1.6 Death or serious morbidity composite (variously defined by trials, e.g. perinatal or infant death, shoulder dystocia, bone fracture or nerve palsy)	2	1930	Risk Ratio(M-H, Random, 95% CI)	0.57 [0.21, 1.55]
1.7 Use of additional pharmacotherapy	9		Risk Ratio(M-H, Random, 95% CI)	Subtotals only
1.7.1 Use of anti-diabetic oral medication	1	197	Risk Ratio(M-H, Random, 95% CI)	0.79 [0.52, 1.19]
1.7.2 Use of insulin treatment	9	3254	Risk Ratio(M-H, Random, 95% CI)	2.54 [1.19, 5.42]
1.8 Maternal hypoglycaemia	1	19	Risk Ratio(M-H, Fixed, 95% CI)	Not estimable
1.9 Glycaemic control during/end treatment	7		Mean Difference(IV, Random, 95% CI)	Subtotals only
1.9.1 Fasting blood glucose concentration mg/dL	6	853	Mean Difference(IV, Random, 95% CI)	-3.10 [-7.01, 0.81]
1.9.2 Postprandial blood glucose concentration mg/dL	4	588	Mean Difference(IV, Random, 95% CI)	-27.11 [-44.62, -9.61]
1.9.3 HbA1c mmol/mol	6	532	Mean Difference(IV, Random, 95% CI)	-0.33 [-0.47, -0.19]
1.10 Weight gain in pregnancy (kg)	4	2930	Mean Difference(IV, Random, 95% CI)	-1.30 [-2.26, -0.35]
1.11 Induction of labour	4	2699	Risk Ratio(M-H, Random, 95% CI)	1.20 [0.99, 1.46]
1.12 Postpartum haemorrhage	2	1165	Risk Ratio(M-H, Random, 95% CI)	0.61 [0.20, 1.89]
1.13 Postnatal infection/pyrexia	1	1000	Risk Ratio(M-H, Fixed, 95% CI)	0.61 [0.34, 1.10]
1.14 Perineal trauma/tear	1	1000	Risk Ratio(M-H, Fixed, 95% CI)	1.04 [0.93, 1.18]

1.15 Breastfeeding at discharge, six weeks postpartum, six months or longer	2		Risk Ratio(M-H, Fixed, 95% CI)	Subtotals only
1.15.1 At discharge	1	1000	Risk Ratio(M-H, Fixed, 95% CI)	1.04 [0.99, 1.10]
1.15.2 At six months postpartum	1	188	Risk Ratio(M-H, Fixed, 95% CI)	0.97 [0.87, 1.07]
1.15.3 Six months postpartum or longer	1	161	Risk Ratio(M-H, Fixed, 95% CI)	1.31 [0.99, 1.74]
1.16 Sense of well-being and quality of lifeduring treatment	2		Mean Difference(IV, Fixed, 95% CI)	Subtotals only
1.16.1 Physical functioning	2	847	Mean Difference(IV, Fixed, 95% CI)	3.09 [0.63, 5.54]
1.16.2 Role physical	2	847	Mean Difference(IV, Fixed, 95% CI)	7.94 [3.29, 12.59]
1.16.3 Bodily pain	2	847	Mean Difference(IV, Fixed, 95% CI)	3.69 [1.33, 6.05]
1.16.4 General health	2	847	Mean Difference(IV, Fixed, 95% CI)	1.76 [0.30, 3.21]
1.16.5 Vitality	2	847	Mean Difference(IV, Fixed, 95% CI)	2.71 [0.88, 4.54]
1.16.6 Social functioning	2	847	Mean Difference(IV, Fixed, 95% CI)	3.27 [0.81, 5.74]
1.16.7 Role emotional	2	847	Mean Difference(IV, Fixed, 95% CI)	9.08 [4.49, 13.67]
1.16.8 Mental health	2	847	Mean Difference(IV, Fixed, 95% CI)	0.90 [-0.96, 2.77]
1.16.9 Health state utility	1	682	Mean Difference(IV, Fixed, 95% CI)	0.02 [0.00, 0.04]
1.16.10 Overall physical component	1	682	Mean Difference(IV, Fixed, 95% CI)	1.50 [0.12, 2.88]
1.16.11 Overall mental component	1	682	Mean Difference(IV, Fixed, 95% CI)	1.30 [-0.17, 2.77]
1.16.12 Anxiety	1	682	Mean Difference(IV, Fixed, 95% CI)	-0.30 [-0.88, 0.28]
1.17 Sense of well-being and quality of life three months postpartum	2		Mean Difference(IV, Random, 95% CI)	Subtotals only
1.17.1 Physical functioning 3 months postpartum	2	738	Mean Difference(IV, Random, 95% CI)	5.05 [-0.91, 11.02]
1.17.2 Physical role 3 months postpartum	2	738	Mean Difference(IV, Random, 95% CI)	8.45 [-3.21, 20.12]
1.17.3 Bodily pain 3 months postpartum	2	738	Mean Difference(IV, Random, 95% CI)	2.37 [-1.03, 5.77]
1.17.4 General health 3 months postpartum	2	738	Mean Difference(IV, Random, 95% CI)	3.98 [-0.46, 8.43]
1.17.5 Vitality 3 months postpartum	2	738	Mean Difference(IV, Random, 95% CI)	4.47 [-1.67, 10.62]
1.17.6 Social functioning 3 months	2	738	Mean Difference(IV, Random, 95% CI)	9.73 [5.17, 14.28]
1.17.7 Role emotional 3 months postpartum	2	738	Mean Difference(IV, Random, 95% CI)	6.92 [-8.24, 22.08]
1.17.8 Mental health 3 months postpartum	2	738	Mean Difference(IV, Random, 95% CI)	-0.09 [-2.58, 2.40]
1.17.9 Health state utility 3 months postpartum	1	573	Mean Difference(IV, Random, 95% CI)	0.01 [-0.01, 0.03]
1.17.10 Overall physical component 3 months postpartum	1	573	Mean Difference(IV, Random, 95% CI)	1.20 [-0.19, 2.59]
1.17.11 Overall mental component 3 months postpartum	1	573	Mean Difference(IV, Random, 95% CI)	0.20 [-1.51, 1.91]
1.17.12 Anxiety scores 3 months postpartum	1	573	Mean Difference(IV, Random, 95% CI)	-0.20 [-0.83, 0.43]
1.18 Postnatal depression	1	573	Risk Ratio(M-H, Fixed, 95% CI)	0.49 [0.31, 0.78]
1.19 Postnatal weight retention or return to pre-pregnancy weight	1		Risk Ratio(M-H, Fixed, 95% CI)	Subtotals only
1.19.1 Six weeks postpartum	1	189	Risk Ratio(M-H, Fixed, 95% CI)	1.20 [0.67, 2.17]
1.19.2 Seven months postpartum	1	159	Risk Ratio(M-H, Fixed, 95% CI)	1.59 [0.99, 2.57]
1.19.3 12 months postpartum	1	156	Risk Ratio(M-H, Fixed, 95% CI)	1.75 [1.05, 2.90]
1.20 Fasting plasma glucose 3 months postpartum mmol/L	1		Mean Difference(IV, Fixed, 95% CI)	Subtotals only
1.20.1 Three months postpartum	1	165	Mean Difference(IV, Fixed, 95% CI)	-0.08 [-0.16, 0.00]
1.20.2 Six months postpartum	1	165	Mean Difference(IV, Fixed, 95% CI)	-0.14 [-0.22, -0.06]
1.21 Maternal postnatal impaired glucose tolerance	1	56	Risk Ratio(M-H, Fixed, 95% CI)	0.67 [0.12, 3.69]

1.22 Maternal metabolic syndrome (follow-up)	1	430	Risk Ratio(M-H, Fixed, 95% CI)	0.93 [0.71, 1.22]
1.23 Stillbirth	4	2355	Risk Ratio(M-H, Fixed, 95% CI)	0.15 [0.01, 2.86]
1.24 Neonatal death	5	3055	Risk Ratio(M-H, Fixed, 95% CI)	0.73 [0.22, 2.42]
1.25 Macrosomia	7	3422	Risk Ratio(M-H, Random, 95% CI)	0.64 [0.48, 0.87]
1.26 Small-for-gestational age	4	2324	Risk Ratio(M-H, Fixed, 95% CI)	0.98 [0.73, 1.31]
1.27 Birth trauma (shoulder dystocia, bone fracture, nerve palsy)	6		Risk Ratio(M-H, Fixed, 95% CI)	Subtotals only
1.27.1 Birth trauma not specified	3	1930	Risk Ratio(M-H, Fixed, 95% CI)	0.48 [0.12, 1.90]
1.27.2 Bone fracture	2	1730	Risk Ratio(M-H, Fixed, 95% CI)	0.35 [0.01, 8.45]
1.27.3 Nerve palsy	1	1030	Risk Ratio(M-H, Fixed, 95% CI)	0.15 [0.01, 2.86]
1.27.4 Shoulder dystocia	5	2894	Risk Ratio(M-H, Fixed, 95% CI)	0.38 [0.21, 0.66]
1.28 Gestational age at birth (weeks)	5	2057	Mean Difference(IV, Fixed, 95% CI)	0.04 [-0.13, 0.20]
1.29 Preterm birth (< 37 weeks' gestation; and < 32 weeks' gestation)	3	1797	Risk Ratio(M-H, Fixed, 95% CI)	0.71 [0.53, 0.96]
1.30 Five-minute Apgar less than seven	1	1030	Risk Ratio(M-H, Fixed, 95% CI)	0.56 [0.21, 1.52]
1.31 Birthweight (grams)	6	3074	Mean Difference(IV, Fixed, 95% CI)	-109.64 [-149.77, -69.51]
1.32 Length (cm)	1	700	Mean Difference(IV, Fixed, 95% CI)	-0.10 [-0.37, 0.17]
1.33 Adiposity (Neonatal fat mass (g))	1	958	Mean Difference(IV, Fixed, 95% CI)	-37.30 [-63.97, -10.63]
1.34 Neonatal hypoglycaemia	6	3000	Risk Ratio(M-H, Random, 95% CI)	0.99 [0.65, 1.52]
1.35 Respiratory distress syndrome	4	2195	Risk Ratio(M-H, Random, 95% CI)	0.79 [0.34, 1.85]
1.36 Neonatal jaundice (hyperbilirubinaemia)	4	2362	Risk Ratio(M-H, Random, 95% CI)	0.76 [0.50, 1.16]
1.37 Hypocalcaemia	2	464	Risk Ratio(M-H, Fixed, 95% CI)	1.38 [1.01, 1.88]
1.38 Polycythemia	1	165	Risk Ratio(M-H, Fixed, 95% CI)	0.22 [0.01, 5.40]
1.39 Childhood weight (kg)	1	199	Mean Difference(IV, Fixed, 95% CI)	-0.30 [-1.29, 0.69]
1.40 Childhood height (cm)	1	199	Mean Difference(IV, Fixed, 95% CI)	-0.60 [-2.05, 0.85]
1.41 Adiposity (Childhood BMI > 85th percentile)	3	767	Risk Ratio(M-H, Fixed, 95% CI)	0.91 [0.75, 1.11]
1.42 Adiposity (BMI Z score childhood)	1	199	Mean Difference(IV, Fixed, 95% CI)	0.08 [-0.28, 0.44]
1.43 Childhood glycaemic control (mmol/L)	1		Mean Difference(IV, Fixed, 95% CI)	Subtotals only
1.43.1 Fasting blood glucose	1	68	Mean Difference(IV, Fixed, 95% CI)	0.10 [-0.10, 0.30]
1.43.2 Two-hour postprandial blood glucose	1	68	Mean Difference(IV, Fixed, 95% CI)	0.00 [-0.48, 0.48]
1.44 Dyslipidaemia or metabolic syndrome (Childhood cholesterol (mg/dL))	1		Mean Difference(IV, Fixed, 95% CI)	Subtotals only
1.44.1 Total cholesterol	1	68	Mean Difference(IV, Fixed, 95% CI)	-0.20 [-0.55, 0.15]
1.44.2 LDL cholesterol	1	68	Mean Difference(IV, Fixed, 95% CI)	-0.12 [-0.50, 0.26]
1.44.3 HDL cholesterol	1	68	Mean Difference(IV, Fixed, 95% CI)	0.10 [-0.05, 0.25]
1.45 Number of antenatal visits or admissions	1	1000	Risk Ratio(M-H, Fixed, 95% CI)	1.06 [0.87, 1.29]
1.46 Number of hospital or health professional visits (including midwife, obstetrician, physician, dietician, diabetic nurse)	1		Risk Ratio(M-H, Fixed, 95% CI)	Subtotals only
1.46.1 Dietitian	1	1000	Risk Ratio(M-H, Fixed, 95% CI)	9.24 [7.12, 12.01]
1.46.2 Diabetes educator	1	1000	Risk Ratio(M-H, Fixed, 95% CI)	8.55 [6.67, 10.96]

1.47 Number of hospital or health professional visits (including midwife, obstetrician, physician, dietician, diabetic nurse)l	2		Mean Difference(IV, Fixed, 95% CI)	Subtotals only
1.47.1 Obstetrician	1	700	Mean Difference(IV, Fixed, 95% CI)	0.20 [-0.21, 0.61]
1.47.2 Healthcare provider (not specified)	1	197	Mean Difference(IV, Fixed, 95% CI)	0.10 [-1.58, 1.78]
1.48 Admission to neonatal intensive care unit/nursery	3	2030	Risk Ratio(M-H, Random, 95% CI)	0.91 [0.59, 1.40]

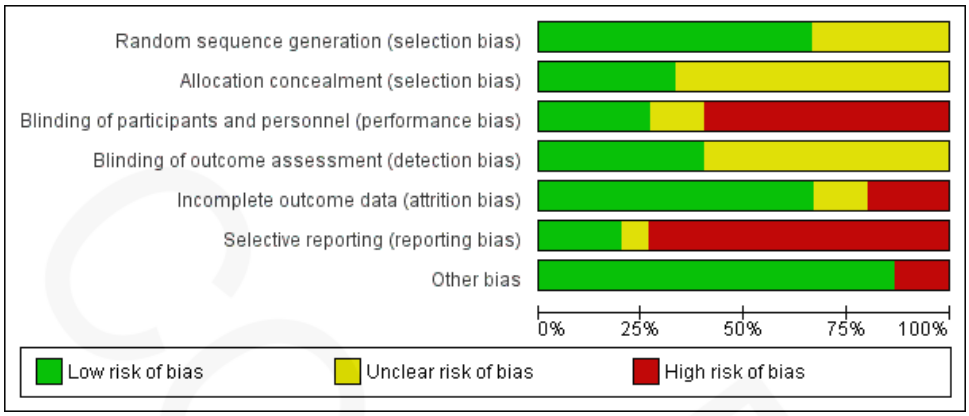
Figures

Figure 1

Caption

Study flow diagram.

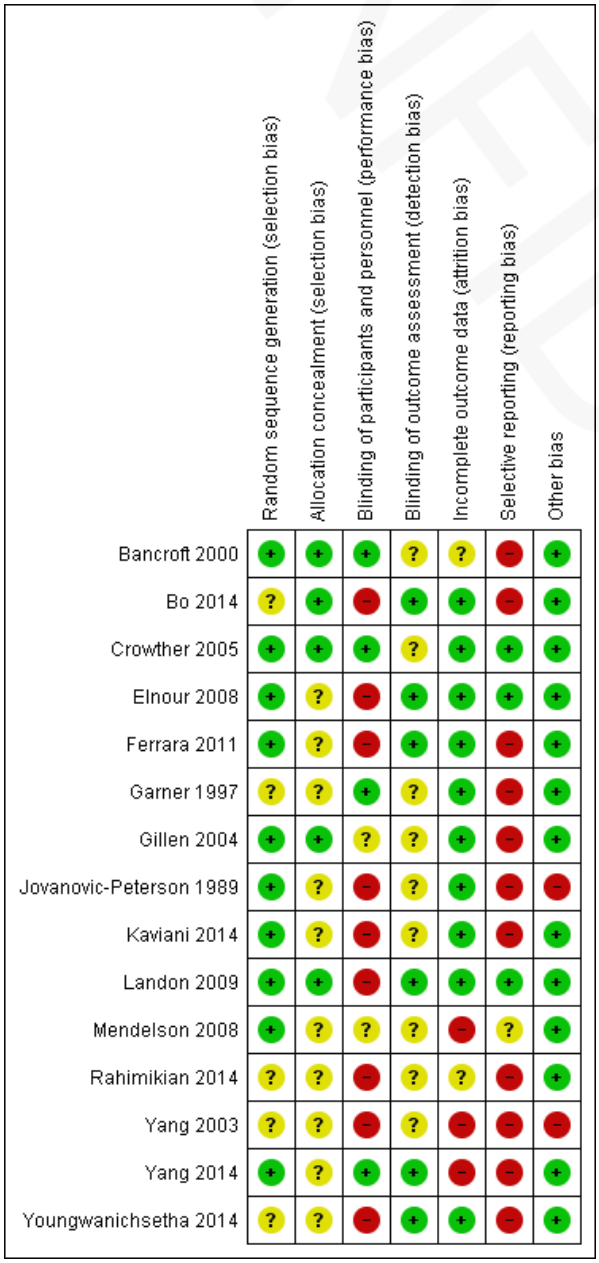
Figure 2



Caption

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

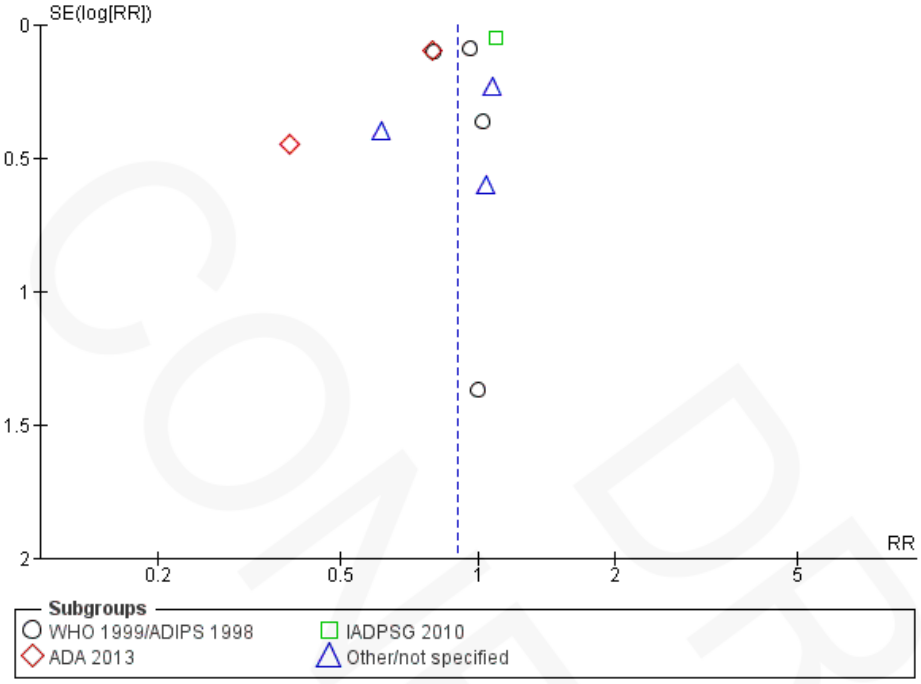
Figure 3



Caption

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

Figure 4 (Analysis 1.2)



Caption

Funnel plot of comparison: 1 Lifestyle intervention versus usual care/control, outcome: 1.2 Caesarean section.

Sources of support

Internal sources

- An internal University department grant, New Zealand
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Feedback

Appendices

1 Clinical trial registry search strategy

gestational diabetes OR GDM

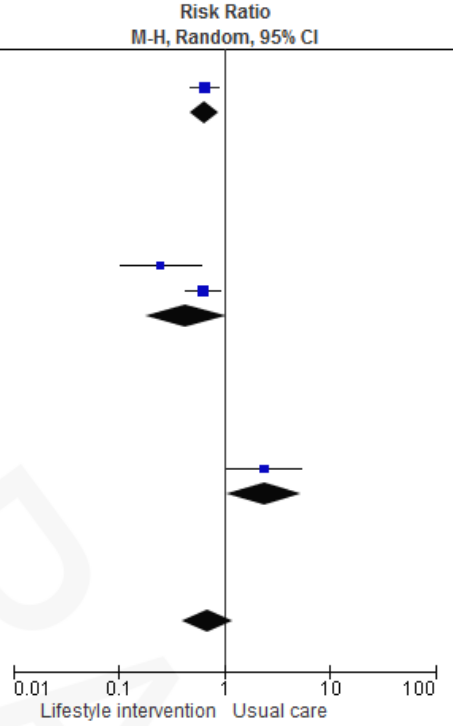
diabetes AND pregnancy

Graphs

1 - Lifestyle intervention versus usual care/control

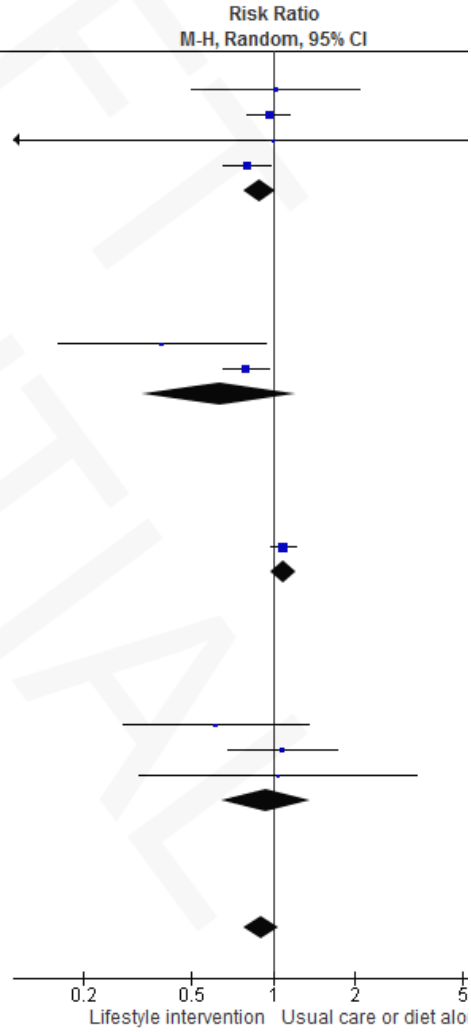
1.1 Hypertensive disorders of pregnancy (including pre-eclampsia, pregnancy-induced hypertension, eclampsia)

Study or Subgroup	Lifestyle intervention Events	Total	Usual care Events	Total	Weight	Risk Ratio M-H, Random, 95% CI
1.1.1 World Health Organization 1999						
Crowther 2005	58	490	93	510	31.5%	0.65 [0.48, 0.88]
Subtotal (95% CI)		**490**		**510**	**31.5%**	**0.65 [0.48, 0.88]**
Total events	58		93			
Heterogeneity: Not applicable						
Test for overall effect: Z = 2.79 (P = 0.005)						
1.1.2 ADA 2013						
Elnour 2008	6	99	16	66	18.6%	0.25 [0.10, 0.61]
Landon 2009	41	476	62	455	30.1%	0.63 [0.44, 0.92]
Subtotal (95% CI)		**575**		**521**	**48.6%**	**0.44 [0.18, 1.06]**
Total events	47		78			
Heterogeneity: Tau² = 0.31; Chi² = 3.59, df = 1 (P = 0.06); I² = 72%						
Test for overall effect: Z = 1.83 (P = 0.07)						
1.1.3 IADPSG 2010						
Yang 2014	18	339	8	361	19.9%	2.40 [1.06, 5.44]
Subtotal (95% CI)		**339**		**361**	**19.9%**	**2.40 [1.06, 5.44]**
Total events	18		8			
Heterogeneity: Not applicable						
Test for overall effect: Z = 2.09 (P = 0.04)						
Total (95% CI)		**1404**		**1392**	**100.0%**	**0.70 [0.40, 1.22]**
Total events	123		179			
Heterogeneity: Tau² = 0.23; Chi² = 14.20, df = 3 (P = 0.003); I² = 79%						
Test for overall effect: Z = 1.26 (P = 0.21)						
Test for subgroup differences: Chi² = 9.94, df = 2 (P = 0.007), I² = 79.9%						



1.2 Caesarean section

Study or Subgroup	Lifestyle intervention Events	Total	Usual care or diet alone Events	Total	Weight	Risk Ratio M-H, Random, 95% CI
1.2.1 WHO 1999/ADIPS 1998						
Bancroft 2000 (1)	10	32	11	36	3.7%	1.02 [0.50, 2.08]
Crowther 2005	152	490	164	510	19.6%	0.96 [0.80, 1.16]
Gillen 2004	1	16	1	16	0.3%	1.00 [0.07, 14.64]
Yang 2003	61	95	44	55	18.5%	0.80 [0.66, 0.98]
Subtotal (95% CI)		**633**		**617**	**42.1%**	**0.89 [0.78, 1.02]**
Total events	224		220			
Heterogeneity: Tau² = 0.00; Chi² = 2.27, df = 3 (P = 0.52); I² = 0%						
Test for overall effect: Z = 1.69 (P = 0.09)						
1.2.2 ADA 2013						
Elnour 2008	7	99	12	66	2.6%	0.39 [0.16, 0.94]
Landon 2009	128	476	154	455	18.7%	0.79 [0.65, 0.97]
Subtotal (95% CI)		**575**		**521**	**21.3%**	**0.63 [0.33, 1.22]**
Total events	135		166			
Heterogeneity: Tau² = 0.15; Chi² = 2.43, df = 1 (P = 0.12); I² = 59%						
Test for overall effect: Z = 1.37 (P = 0.17)						
1.2.3 IADPSG 2010						
Yang 2014	239	339	233	361	24.6%	1.09 [0.99, 1.21]
Subtotal (95% CI)		**339**		**361**	**24.6%**	**1.09 [0.99, 1.21]**
Total events	239		233			
Heterogeneity: Not applicable						
Test for overall effect: Z = 1.68 (P = 0.09)						
1.2.4 Other/not specified						
Bo 2014 (2)	8	50	13	50	3.1%	0.62 [0.28, 1.35]
Garner 1997	30	149	28	150	7.5%	1.08 [0.68, 1.71]
Mendelson 2008	5	49	5	51	1.5%	1.04 [0.32, 3.37]
Subtotal (95% CI)		**248**		**251**	**12.1%**	**0.94 [0.65, 1.38]**
Total events	43		46			
Heterogeneity: Tau² = 0.00; Chi² = 1.48, df = 2 (P = 0.48); I² = 0%						
Test for overall effect: Z = 0.29 (P = 0.77)						
Total (95% CI)		**1795**		**1750**	**100.0%**	**0.90 [0.78, 1.05]**
Total events	641		665			
Heterogeneity: Tau² = 0.02; Chi² = 18.83, df = 9 (P = 0.03); I² = 52%						
Test for overall effect: Z = 1.34 (P = 0.18)						
Test for subgroup differences: Chi² = 7.65, df = 3 (P = 0.05), I² = 60.8%						



Footnotes

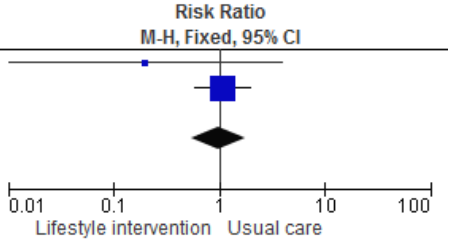
(1) LSCS

(2) Control group was diet alone

1.3 Development of type 2 diabetes

Study or Subgroup	Lifestyle intervention Events	Total	Usual care Events	Total	Weight	Risk Ratio M-H, Fixed, 95% CI
Bancroft 2000 (1)	0	28	2	28	12.1%	0.20 [0.01, 3.99]
Landon 2009 (2)	21	229	17	201	87.9%	1.08 [0.59, 2.00]
Total (95% CI)		**257**		**229**	**100.0%**	**0.98 [0.54, 1.76]**
Total events	21		19			

Heterogeneity: Chi² = 1.19, df = 1 (P = 0.28); I² = 16%
Test for overall effect: Z = 0.08 (P = 0.94)

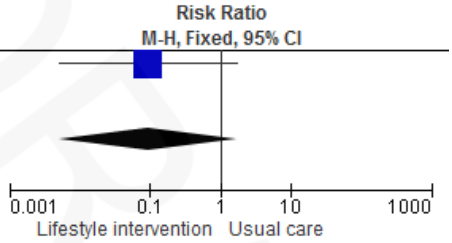


Footnotes
(1) No details
(2) 4.5 to 10 years follow-up

1.4 Perinatal (fetal and neonatal death) and later infant mortality

Study or Subgroup	Lifestyle Events	Total	Usual care Events	Total	Weight	Risk Ratio M-H, Fixed, 95% CI
Crowther 2005 (1)	0	506	5	524	100.0%	0.09 [0.01, 1.70]
Landon 2009 (2)	0	485	0	473		Not estimable
Total (95% CI)		**991**		**997**	**100.0%**	**0.09 [0.01, 1.70]**
Total events	0		5			

Heterogeneity: Not applicable
Test for overall effect: Z = 1.60 (P = 0.11)

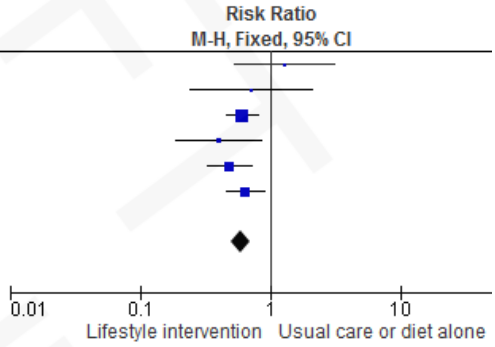


Footnotes
(1) Perinatal death
(2) Perinatal death

1.5 Large-for-gestational age

Study or Subgroup	Lifestyle intervention Events	Total	Usual care or diet alone Events	Total	Weight	Risk Ratio M-H, Fixed, 95% CI
Bancroft 2000	8	32	7	36	2.3%	1.29 [0.53, 3.15]
Bo 2014 (1)	5	50	7	50	2.5%	0.71 [0.24, 2.10]
Crowther 2005	68	506	115	524	40.1%	0.61 [0.47, 0.81]
Elnour 2008	9	99	15	66	6.4%	0.40 [0.19, 0.86]
Landon 2009	34	477	66	454	24.0%	0.49 [0.33, 0.73]
Yang 2014	44	339	72	361	24.7%	0.65 [0.46, 0.92]
Total (95% CI)		**1503**		**1491**	**100.0%**	**0.60 [0.50, 0.71]**
Total events	168		282			

Heterogeneity: Chi² = 5.21, df = 5 (P = 0.39); I² = 4%
Test for overall effect: Z = 5.74 (P < 0.00001)

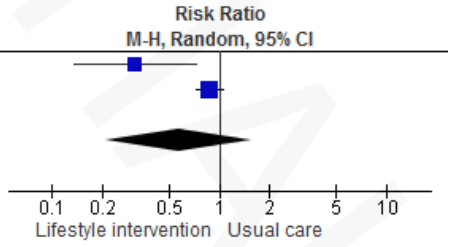


Footnotes
(1) Control group was diet alone

1.6 Death or serious morbidity composite (variously defined by trials, e.g. perinatal or infant death, shoulder dystocia, bone fracture or nerve pals

Study or Subgroup	Lifestyle intervention Events	Total	Usual care Events	Total	Weight	Risk Ratio M-H, Random, 95% CI
Crowther 2005 (1)	7	506	23	524	41.9%	0.32 [0.14, 0.73]
Landon 2009 (2)	149	460	163	440	58.1%	0.87 [0.73, 1.05]
Total (95% CI)		**966**		**964**	**100.0%**	**0.57 [0.21, 1.55]**
Total events	156		186			

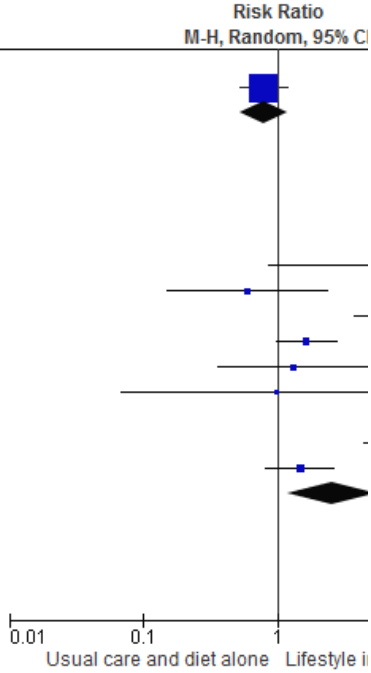
Heterogeneity: Tau² = 0.44; Chi² = 5.60, df = 1 (P = 0.02); I² = 82%
Test for overall effect: Z = 1.10 (P = 0.27)



Footnotes
(1) Composite included one or more of: death, shoulder dystocia, bone fracture and nerve palsy
(2) Composite included: stillbirth, neonatal death, hypoglycaemia, hyperbilirubinaemia, elevated cord-blood C-peptide level and birth trauma

1.7 Use of additional pharmacotherapy

Study or Subgroup	Lifestyle intervention Events	Total	Usual care and diet alone Events	Total	Weight	Risk Ratio M-H, Random, 95% CI
1.7.1 Use of anti-diabetic oral medication						
Ferrara 2011	27	96	36	101	100.0%	0.79 [0.52, 1.19]
Subtotal (95% CI)		**96**		**101**	**100.0%**	**0.79 [0.52, 1.19]**
Total events	27		36			
Heterogeneity: Not applicable						
Test for overall effect: Z = 1.12 (P = 0.26)						
1.7.2 Use of insulin treatment						
Bancroft 2000	6	32	0	36	5.2%	14.58 [0.85, 248.95]
Bo 2014 (1)	3	50	5	50	11.9%	0.60 [0.15, 2.38]
Crowther 2005	100	490	17	510	17.9%	6.12 [3.72, 10.08]
Elnour 2008	37	108	15	72	17.8%	1.64 [0.98, 2.77]
Ferrara 2011	5	96	4	101	12.5%	1.32 [0.36, 4.75]
Gillen 2004	1	16	1	16	5.7%	1.00 [0.07, 14.64]
Jovanovic-Peterson 1989 (2)	0	10	0	9		Not estimable
Landon 2009	37	485	2	473	11.6%	18.04 [4.37, 74.44]
Yang 2014	25	339	18	361	17.4%	1.48 [0.82, 2.66]
Subtotal (95% CI)		**1626**		**1628**	**100.0%**	**2.54 [1.19, 5.42]**
Total events	214		62			
Heterogeneity: Tau² = 0.77; Chi² = 34.82, df = 7 (P < 0.0001); I² = 80%						
Test for overall effect: Z = 2.40 (P = 0.02)						



Test for subgroup differences: Chi² = 7.00, df = 1 (P = 0.008), I² = 85.7%

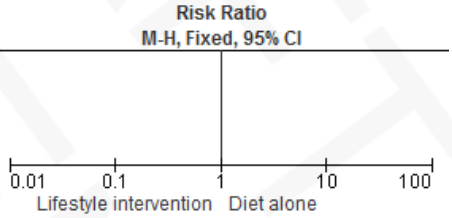
Footnotes

(1) Control group was diet alone

(2) Control group was diet alone

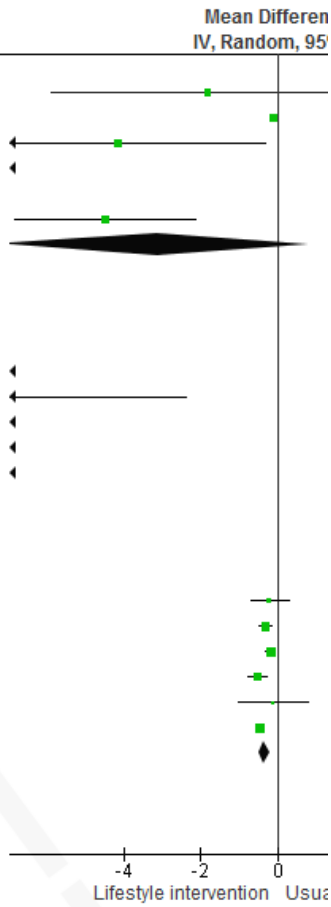
1.8 Maternal hypoglycaemia

Study or Subgroup	Lifestyle Events	Total	Diet alone Events	Total	Weight	Risk Ratio M-H, Fixed, 95% CI
Jovanovic-Peterson 1989	0	10	0	9		Not estimable
Total (95% CI)		**10**		**9**		**Not estimable**
Total events	0		0			
Heterogeneity: Not applicable						
Test for overall effect: Not applicable						



1.9 Glycaemic control during/end treatment

Study or Subgroup	Lifestyle intervention Mean	SD	Total	Usual care and diet alone Mean	SD	Total	Weight	Mean Difference IV, Random, 95% CI
1.9.1 Fasting blood glucose concentration mg/dL								
Bo 2014 (1)	72.3	10.6	50	74.1	10.1	50	16.4%	-1.80 [-5.86, 2.26]
Elnour 2008	5.06	0.18	99	5.14	0.26	66	19.9%	-0.08 [-0.15, -0.01]
Garner 1997	80.46	14.76	149	84.6	18.8	150	16.7%	-4.14 [-7.97, -0.31]
Jovanovic-Peterson 1989 (2)	70.1	6.6	10	87.6	6.2	9	13.9%	-17.50 [-23.26, -11.74]
Mendelson 2008 (3)	101	9.26	49	92.9	17.3	51	14.4%	8.10 [2.69, 13.51]
Youngwanichsetha 2014	83.39	7.69	85	87.85	7.94	85	18.6%	-4.46 [-6.81, -2.11]
Subtotal (95% CI)			**442**			**411**	**100.0%**	**-3.10 [-7.01, 0.81]**
Heterogeneity: Tau² = 19.94; Chi² = 62.27, df = 5 (P < 0.00001); I² = 92%								
Test for overall effect: Z = 1.55 (P = 0.12)								
1.9.2 Postprandial blood glucose concentration mg/dL								
Bo 2014 (4)	107.8	18.3	50	121.8	16.1	50	25.6%	-14.00 [-20.76, -7.24]
Garner 1997 (5)	126.18	25.2	149	135.36	34.14	150	25.6%	-9.18 [-15.98, -2.38]
Jovanovic-Peterson 1989 (6)	105.9	18.9	10	187.5	12.9	9	22.5%	-81.60 [-96.03, -67.17]
Youngwanichsetha 2014 (7)	103.67	9.93	85	114.36	10.15	85	26.4%	-10.69 [-13.71, -7.67]
Subtotal (95% CI)			**294**			**294**	**100.0%**	**-27.11 [-44.62, -9.61]**
Heterogeneity: Tau² = 300.13; Chi² = 90.35, df = 3 (P < 0.00001); I² = 97%								
Test for overall effect: Z = 3.04 (P = 0.002)								
1.9.3 HbA1c mmol/mol								
Bancroft 2000 (8)	5.3	0.9	24	5.5	0.9	27	6.3%	-0.20 [-0.69, 0.29]
Bo 2014 (9)	4.7	0.4	50	5	0.4	50	22.5%	-0.30 [-0.46, -0.14]
Elnour 2008	6.38	0.2	99	6.55	0.5	66	25.0%	-0.17 [-0.30, -0.04]
Jovanovic-Peterson 1989 (10)	4.16	0.28	10	4.67	0.25	9	16.4%	-0.51 [-0.75, -0.27]
Mendelson 2008	5.62	1.03	15	5.73	1.29	12	2.2%	-0.11 [-1.01, 0.79]
Youngwanichsetha 2014	5.23	0.22	85	5.68	0.38	85	27.6%	-0.45 [-0.54, -0.36]
Subtotal (95% CI)			**283**			**249**	**100.0%**	**-0.33 [-0.47, -0.19]**
Heterogeneity: Tau² = 0.02; Chi² = 14.89, df = 5 (P = 0.01); I² = 66%								
Test for overall effect: Z = 4.69 (P < 0.00001)								



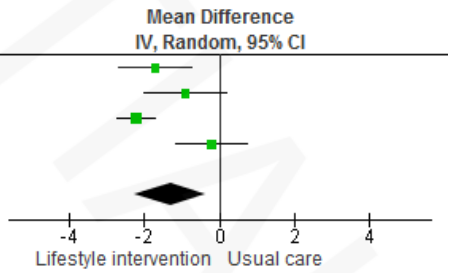
Test for subgroup differences: Chi² = 10.91, df = 2 (P = 0.004), I² = 81.7%

Footnotes

(1) End of trial; control group was diet alone
(2) Control group was diet alone
(3) Mendelson 2008 data was end of treatment at birth
(4) Unknown timing; control group was diet alone
(5) one hour postprandial
(6) One hour postprandial; control group was diet alone
(7) two hour postprandial
(8) 38 weeks' gestation
(9) Control group was diet alone
(10) Control group was diet alone

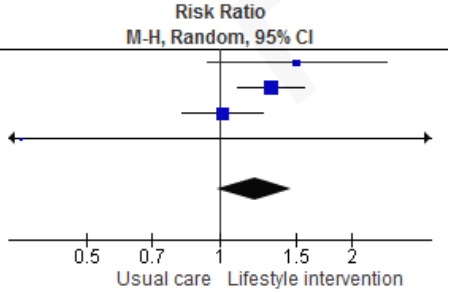
1.10 Weight gain in pregnancy (kg)

Study or Subgroup	Lifestyle intervention Mean	SD	Total	Usual care Mean	SD	Total	Weight	Mean Difference IV, Random, 95% CI
Crowther 2005	8.1	6.6	490	9.8	9	510	24.0%	-1.70 [-2.68, -0.72]
Garner 1997	12.5	4.8	149	13.4	4.8	150	22.6%	-0.90 [-1.99, 0.19]
Landon 2009	2.8	4.5	476	5	3.3	455	29.3%	-2.20 [-2.71, -1.69]
Yang 2014	15.5	6.5	339	15.7	6.4	361	24.2%	-0.20 [-1.16, 0.76]
Total (95% CI)			**1454**			**1476**	**100.0%**	**-1.30 [-2.26, -0.35]**
Heterogeneity: Tau² = 0.75; Chi² = 15.22, df = 3 (P = 0.002); I² = 80%								
Test for overall effect: Z = 2.67 (P = 0.008)								



1.11 Induction of labour

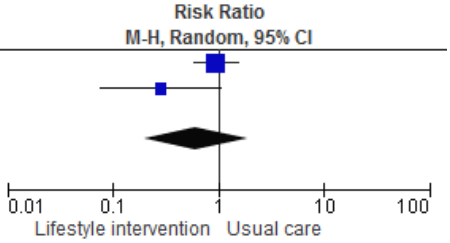
Study or Subgroup	Lifestyle intervention Events	Total	Usual care Events	Total	Weight	Risk Ratio M-H, Random, 95% CI
Bancroft 2000	20	32	15	36	14.0%	1.50 [0.94, 2.40]
Crowther 2005	189	490	150	510	46.1%	1.31 [1.10, 1.56]
Landon 2009	130	476	122	455	39.5%	1.02 [0.82, 1.26]
Yang 2014	0	339	1	361	0.4%	0.35 [0.01, 8.68]
Total (95% CI)		**1337**		**1362**	**100.0%**	**1.20 [0.99, 1.46]**
Total events	339		288			
Heterogeneity: Tau² = 0.01; Chi² = 4.76, df = 3 (P = 0.19); I² = 37%						
Test for overall effect: Z = 1.85 (P = 0.06)						



1.12 Postpartum haemorrhage

Study or Subgroup	Specific treatment Events	Total	Usual care Events	Total	Weight	Risk Ratio M-H, Random, 95% CI
Crowther 2005	29	490	32	510	63.6%	0.94 [0.58, 1.54]
Elnour 2008	3	99	7	66	36.4%	0.29 [0.08, 1.07]
Total (95% CI)		**589**		**576**	**100.0%**	**0.61 [0.20, 1.89]**
Total events	32		39			

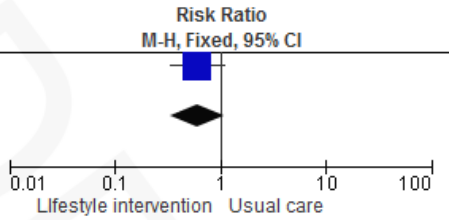
Heterogeneity: Tau² = 0.46; Chi² = 2.79, df = 1 (P = 0.10); I² = 64%
Test for overall effect: Z = 0.86 (P = 0.39)



1.13 Postnatal infection/pyrexia

Study or Subgroup	Lifestyle Events	Total	Usual care Events	Total	Weight	Risk Ratio M-H, Fixed, 95% CI
Crowther 2005	17	490	29	510	100.0%	0.61 [0.34, 1.10]
Total (95% CI)		**490**		**510**	**100.0%**	**0.61 [0.34, 1.10]**
Total events	17		29			

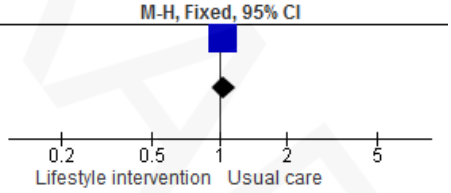
Heterogeneity: Not applicable
Test for overall effect: Z = 1.65 (P = 0.10)



1.14 Perineal trauma/tear

Study or Subgroup	Experimental Events	Total	Usual care Events	Total	Weight	Risk Ratio M-H, Fixed, 95% CI
Crowther 2005	255	490	254	510	100.0%	1.04 [0.93, 1.18]
Total (95% CI)		**490**		**510**	**100.0%**	**1.04 [0.93, 1.18]**
Total events	255		254			

Heterogeneity: Not applicable
Test for overall effect: Z = 0.71 (P = 0.48)



1.15 Breastfeeding at discharge, six weeks postpartum, six months or longer

Study or Subgroup	Lifestyle intervention Events	Total	Usual care Events	Total	Weight	Risk Ratio M-H, Fixed, 95% CI
1.15.1 At discharge						
Crowther 2005	413	490	412	510	100.0%	1.04 [0.99, 1.10]
Subtotal (95% CI)		**490**		**510**	**100.0%**	**1.04 [0.99, 1.10]**
Total events	413		412			

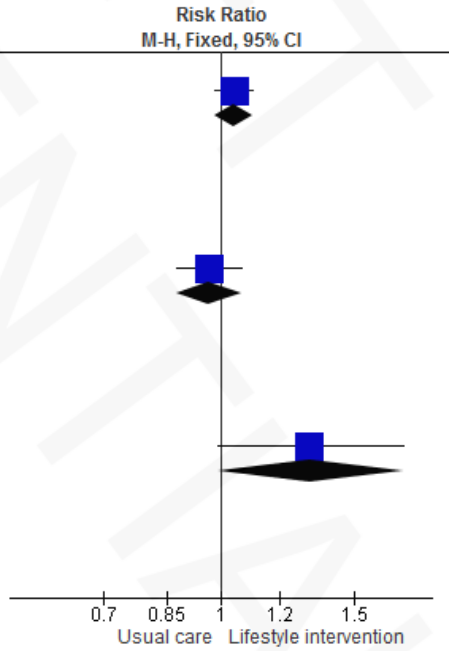
Heterogeneity: Not applicable
Test for overall effect: Z = 1.46 (P = 0.14)

1.15.2 At six months postpartum						
Ferrara 2011	79	90	89	98	100.0%	0.97 [0.87, 1.07]
Subtotal (95% CI)		**90**		**98**	**100.0%**	**0.97 [0.87, 1.07]**
Total events	79		89			

Heterogeneity: Not applicable
Test for overall effect: Z = 0.67 (P = 0.50)

1.15.3 Six months postpartum or longer						
Ferrara 2011	47	75	41	86	100.0%	1.31 [0.99, 1.74]
Subtotal (95% CI)		**75**		**86**	**100.0%**	**1.31 [0.99, 1.74]**
Total events	47		41			

Heterogeneity: Not applicable
Test for overall effect: Z = 1.90 (P = 0.06)



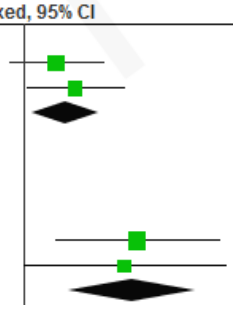
Test for subgroup differences: Chi² = 4.63, df = 2 (P = 0.10), I² = 56.8%

1.16 Sense of well-being and quality of lifeduring treatment

Study or Subgroup	Lifestyle Mean	SD	Total	Usual care Mean	SD	Total	Weight	Mean Difference IV, Fixed, 95% CI
1.16.1 Physical functioning								
Crowther 2005	56.4	23.1	332	54	22.7	350	51.0%	2.40 [-1.04, 5.84]
Elnour 2008	67.2	8.9	99	63.4	12.6	66	49.0%	3.80 [0.29, 7.31]
Subtotal (95% CI)			**431**			**416**	**100.0%**	**3.09 [0.63, 5.54]**

Heterogeneity: Chi² = 0.31, df = 1 (P = 0.58); I² = 0%
Test for overall effect: Z = 2.46 (P = 0.01)

1.16.2 Role physical								
Crowther 2005	40.7	41.4	332	32.4	38.1	350	60.4%	8.30 [2.32, 14.28]
Elnour 2008	51	20.8	99	43.6	25.5	66	39.6%	7.40 [0.01, 14.79]
Subtotal (95% CI)			**431**			**416**	**100.0%**	**7.94 [3.29, 12.59]**



Heterogeneity: Chi² = 0.03, df = 1 (P = 0.85); I² = 0%
Test for overall effect: Z = 3.35 (P = 0.0008)

1.16.3 Bodily pain

Crowther 2005	63.1	24.6	332	59	24.1	350	41.6%	4.10 [0.44, 7.76]
Elnour 2008	72.8	12.7	99	69.4	7.5	66	58.4%	3.40 [0.31, 6.49]
Subtotal (95% CI)			**431**			**416**	**100.0%**	**3.69 [1.33, 6.05]**

Heterogeneity: Chi² = 0.08, df = 1 (P = 0.77); I² = 0%
Test for overall effect: Z = 3.07 (P = 0.002)

1.16.4 General health

Crowther 2005	73.4	17.4	332	72.5	18.9	350	28.6%	0.90 [-1.82, 3.62]
Elnour 2008	68.4	6.9	99	66.3	4.4	66	71.4%	2.10 [0.38, 3.82]
Subtotal (95% CI)			**431**			**416**	**100.0%**	**1.76 [0.30, 3.21]**

Heterogeneity: Chi² = 0.53, df = 1 (P = 0.47); I² = 0%
Test for overall effect: Z = 2.36 (P = 0.02)

1.16.5 Vitality

Crowther 2005	50	21	332	46.7	20.3	350	34.8%	3.30 [0.20, 6.40]
Elnour 2008	58.8	6.6	99	56.4	7.7	66	65.2%	2.40 [0.13, 4.67]
Subtotal (95% CI)			**431**			**416**	**100.0%**	**2.71 [0.88, 4.54]**

Heterogeneity: Chi² = 0.21, df = 1 (P = 0.65); I² = 0%
Test for overall effect: Z = 2.91 (P = 0.004)

1.16.6 Social functioning

Crowther 2005	73.5	24	332	70.9	23.2	350	48.3%	2.60 [-0.95, 6.15]
Elnour 2008	63.5	9.3	99	59.6	12	66	51.7%	3.90 [0.47, 7.33]
Subtotal (95% CI)			**431**			**416**	**100.0%**	**3.27 [0.81, 5.74]**

Heterogeneity: Chi² = 0.27, df = 1 (P = 0.61); I² = 0%
Test for overall effect: Z = 2.60 (P = 0.009)

1.16.7 Role emotional

Crowther 2005	77.5	35.3	332	69.1	40.9	350	64.2%	8.40 [2.67, 14.13]
Elnour 2008	68.4	20.7	99	58.1	26.9	66	35.8%	10.30 [2.64, 17.96]
Subtotal (95% CI)			**431**			**416**	**100.0%**	**9.08 [4.49, 13.67]**

Heterogeneity: Chi² = 0.15, df = 1 (P = 0.70); I² = 0%
Test for overall effect: Z = 3.88 (P = 0.0001)

1.16.8 Mental health

Crowther 2005	75.1	15.4	332	73.8	16.6	350	60.4%	1.30 [-1.10, 3.70]
Elnour 2008	60.7	7.6	99	60.4	10.6	66	39.6%	0.30 [-2.66, 3.26]
Subtotal (95% CI)			**431**			**416**	**100.0%**	**0.90 [-0.96, 2.77]**

Heterogeneity: Chi² = 0.26, df = 1 (P = 0.61); I² = 0%
Test for overall effect: Z = 0.95 (P = 0.34)

1.16.9 Health state utility

Crowther 2005	0.72	0.11	332	0.7	0.11	350	100.0%	0.02 [0.00, 0.04]
Subtotal (95% CI)			**332**			**350**	**100.0%**	**0.02 [0.00, 0.04]**

Heterogeneity: Not applicable
Test for overall effect: Z = 2.37 (P = 0.02)

1.16.10 Overall physical component

Crowther 2005	38.8	9.4	332	37.3	9	350	100.0%	1.50 [0.12, 2.88]
Subtotal (95% CI)			**332**			**350**	**100.0%**	**1.50 [0.12, 2.88]**

Heterogeneity: Not applicable
Test for overall effect: Z = 2.13 (P = 0.03)

1.16.11 Overall mental component

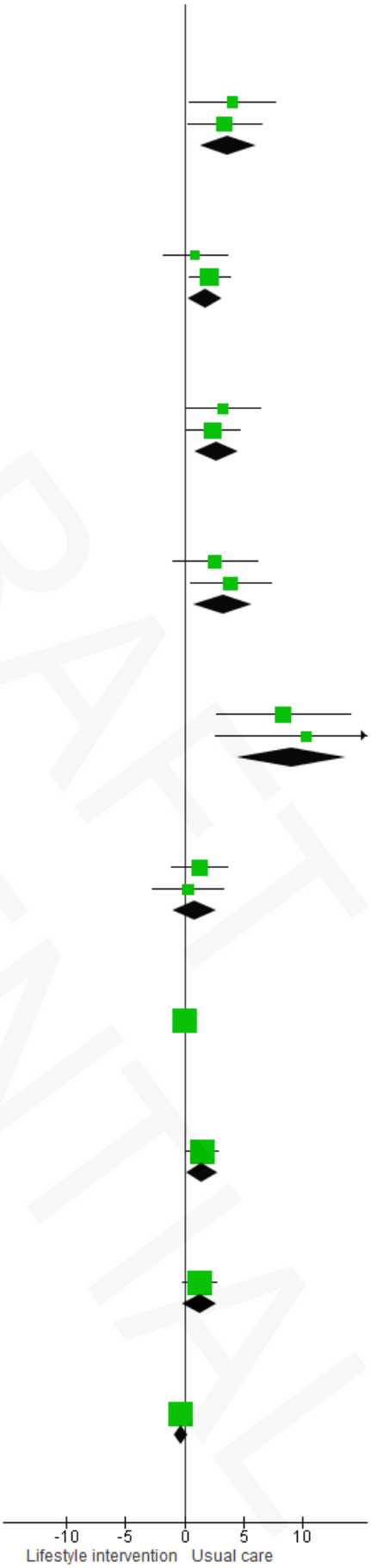
Crowther 2005	50.9	9.2	332	49.6	10.4	350	100.0%	1.30 [-0.17, 2.77]
Subtotal (95% CI)			**332**			**350**	**100.0%**	**1.30 [-0.17, 2.77]**

Heterogeneity: Not applicable
Test for overall effect: Z = 1.73 (P = 0.08)

1.16.12 Anxiety

Crowther 2005	11.2	3.7	332	11.5	4	350	100.0%	-0.30 [-0.88, 0.28]
Subtotal (95% CI)			**332**			**350**	**100.0%**	**-0.30 [-0.88, 0.28]**

Heterogeneity: Not applicable
Test for overall effect: Z = 1.02 (P = 0.31)



Test for subgroup differences: Chi² = 71.22, df = 11 (P < 0.00001), I² = 84.6%

1.17 Sense of well-being and quality of life three months postpartum

	Lifestyle			Usual care				Mean Difference	Mean Difference
Study or Subgroup	**Mean**	**SD**	**Total**	**Mean**	**SD**	**Total**	**Weight**	**IV, Random, 95% CI**	**IV, Random, 95% CI**
1.17.1 Physical functioning 3 months postpartum									
Crowther 2005	85.8	19.5	278	83.6	19.6	295	53.3%	2.20 [-1.00, 5.40]	
Elnour 2008	86.6	18.5	99	78.2	19.4	66	46.7%	8.40 [2.88, 13.92]	

Elnour 2008	86.6	16.5	99	78.3	10.4	66	46.7%	8.30 [3.88, 12.72]
Subtotal (95% CI)			**377**			**361**	**100.0%**	**5.05 [-0.91, 11.02]**

Heterogeneity: Tau² = 14.72; Chi² = 4.79, df = 1 (P = 0.03); I² = 79%
Test for overall effect: Z = 1.66 (P = 0.10)

1.17.2 Physical role 3 months postpartum

Crowther 2005	79.9	33.7	278	75.9	36.3	295	64.1%	4.00 [-1.73, 9.73]
Elnour 2008	81.6	39.3	99	65.2	48.9	66	35.9%	16.40 [2.29, 30.51]
Subtotal (95% CI)			**377**			**361**	**100.0%**	**8.45 [-3.21, 20.12]**

Heterogeneity: Tau² = 46.69; Chi² = 2.55, df = 1 (P = 0.11); I² = 61%
Test for overall effect: Z = 1.42 (P = 0.16)

1.17.3 Bodily pain 3 months postpartum

Crowther 2005	77.7	23	278	77.3	21.6	295	43.7%	0.40 [-3.26, 4.06]
Elnour 2008	94.1	10.9	99	90.2	7	66	56.3%	3.90 [1.17, 6.63]
Subtotal (95% CI)			**377**			**361**	**100.0%**	**2.37 [-1.03, 5.77]**

Heterogeneity: Tau² = 3.41; Chi² = 2.26, df = 1 (P = 0.13); I² = 56%
Test for overall effect: Z = 1.37 (P = 0.17)

1.17.4 General health 3 months postpartum

Crowther 2005	76.8	17.5	278	74.2	18.2	295	72.8%	2.60 [-0.32, 5.52]
Elnour 2008	78	26.4	99	70.3	21.6	66	27.2%	7.70 [0.34, 15.06]
Subtotal (95% CI)			**377**			**361**	**100.0%**	**3.98 [-0.46, 8.43]**

Heterogeneity: Tau² = 4.84; Chi² = 1.59, df = 1 (P = 0.21); I² = 37%
Test for overall effect: Z = 1.76 (P = 0.08)

1.17.5 Vitality 3 months postpartum

Crowther 2005	60	19.3	278	57.7	19.7	295	67.6%	2.30 [-0.89, 5.49]
Elnour 2008	84.6	22.6	99	75.6	29.6	66	32.4%	9.00 [0.58, 17.42]
Subtotal (95% CI)			**377**			**361**	**100.0%**	**4.47 [-1.67, 10.62]**

Heterogeneity: Tau² = 11.90; Chi² = 2.13, df = 1 (P = 0.14); I² = 53%
Test for overall effect: Z = 1.43 (P = 0.15)

1.17.6 Social functioning 3 months

Crowther 2005	81.4	21.3	278	70	23.3	295	65.8%	11.40 [7.75, 15.05]
Elnour 2008	87.3	17.8	99	80.8	22.8	66	34.2%	6.50 [-0.02, 13.02]
Subtotal (95% CI)			**377**			**361**	**100.0%**	**9.73 [5.17, 14.28]**

Heterogeneity: Tau² = 4.73; Chi² = 1.65, df = 1 (P = 0.20); I² = 39%
Test for overall effect: Z = 4.19 (P < 0.0001)

1.17.7 Role emotional 3 months postpartum

Crowther 2005	78.9	35	278	78.5	35.7	295	58.5%	0.40 [-5.39, 6.19]
Elnour 2008	82.8	38.3	99	66.7	48.5	66	41.5%	16.10 [2.18, 30.02]
Subtotal (95% CI)			**377**			**361**	**100.0%**	**6.92 [-8.24, 22.08]**

Heterogeneity: Tau² = 93.65; Chi² = 4.16, df = 1 (P = 0.04); I² = 76%
Test for overall effect: Z = 0.89 (P = 0.37)

1.17.8 Mental health 3 months postpartum

Crowther 2005	77	15.4	278	77.4	16.7	295	90.0%	-0.40 [-3.03, 2.23]
Elnour 2008	86.3	22.6	99	83.6	27	66	10.0%	2.70 [-5.19, 10.59]
Subtotal (95% CI)			**377**			**361**	**100.0%**	**-0.09 [-2.58, 2.40]**

Heterogeneity: Tau² = 0.00; Chi² = 0.53, df = 1 (P = 0.47); I² = 0%
Test for overall effect: Z = 0.07 (P = 0.94)

1.17.9 Health state utility 3 months postpartum

Crowther 2005	0.79	0.1	278	0.78	0.11	295	100.0%	0.01 [-0.01, 0.03]
Subtotal (95% CI)			**278**			**295**	**100.0%**	**0.01 [-0.01, 0.03]**

Heterogeneity: Not applicable
Test for overall effect: Z = 1.14 (P = 0.25)

1.17.10 Overall physical component 3 months postpartum

Crowther 2005	51.2	8.5	278	50	8.5	295	100.0%	1.20 [-0.19, 2.59]
Subtotal (95% CI)			**278**			**295**	**100.0%**	**1.20 [-0.19, 2.59]**

Heterogeneity: Not applicable
Test for overall effect: Z = 1.69 (P = 0.09)

1.17.11 Overall mental component 3 months postpartum

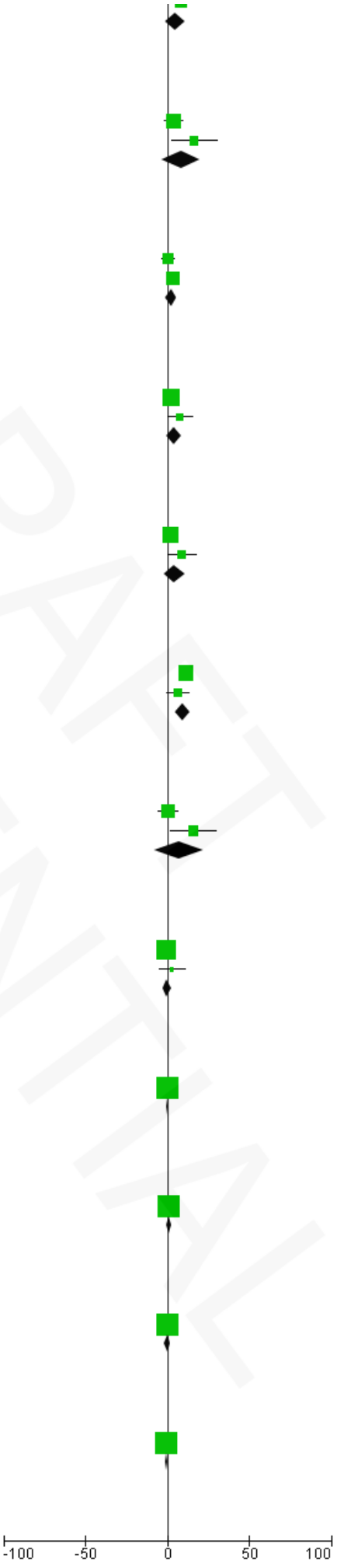
Crowther 2005	48.6	10	278	48.4	10.9	295	100.0%	0.20 [-1.51, 1.91]
Subtotal (95% CI)			**278**			**295**	**100.0%**	**0.20 [-1.51, 1.91]**

Heterogeneity: Not applicable
Test for overall effect: Z = 0.23 (P = 0.82)

1.17.12 Anxiety scores 3 months postpartum

Crowther 2005	10.6	3.9	278	10.8	3.8	295	100.0%	-0.20 [-0.83, 0.43]
Subtotal (95% CI)			**278**			**295**	**100.0%**	**-0.20 [-0.83, 0.43]**

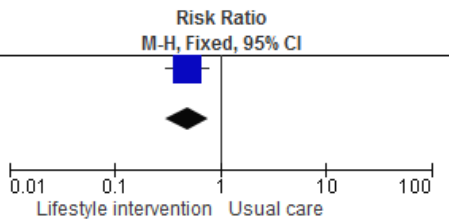
Heterogeneity: Not applicable
Test for overall effect: Z = 0.62 (P = 0.53)



Test for subgroup differences: Chi² = 33.26, df = 11 (P = 0.0005), I² = 66.9%

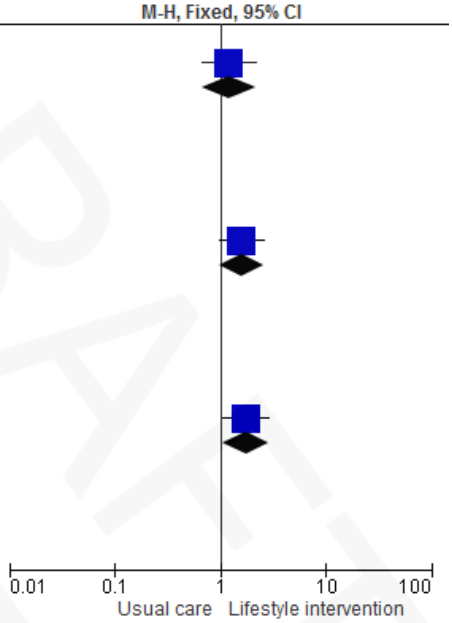
1.18 Postnatal depression

Study or Subgroup	Lifestyle intervention Events	Total	Usual care Events	Total	Weight	Risk Ratio M-H, Fixed, 95% CI
Crowther 2005	23	278	50	295	100.0%	0.49 [0.31, 0.78]
Total (95% CI)		**278**		**295**	**100.0%**	**0.49 [0.31, 0.78]**
Total events	23		50			
Heterogeneity: Not applicable						
Test for overall effect: Z = 3.02 (P = 0.003)						



1.19 Postnatal weight retention or return to pre-pregnancy weight

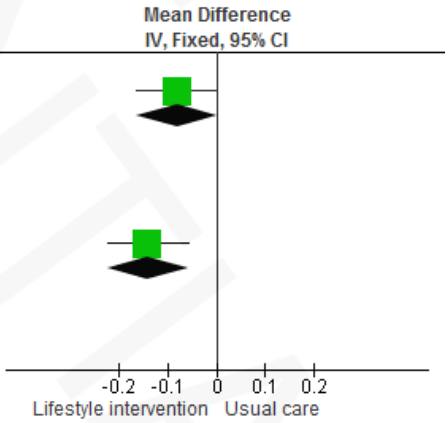
Study or Subgroup	Lifestyle intervention Events	Total	Usual care Events	Total	Weight	Risk Ratio M-H, Fixed, 95% CI
1.19.1 Six weeks postpartum						
Ferrara 2011	19	91	17	98	100.0%	1.20 [0.67, 2.17]
Subtotal (95% CI)		**91**		**98**	**100.0%**	**1.20 [0.67, 2.17]**
Total events	19		17			
Heterogeneity: Not applicable						
Test for overall effect: Z = 0.62 (P = 0.54)						
1.19.2 Seven months postpartum						
Ferrara 2011	27	71	21	88	100.0%	1.59 [0.99, 2.57]
Subtotal (95% CI)		**71**		**88**	**100.0%**	**1.59 [0.99, 2.57]**
Total events	27		21			
Heterogeneity: Not applicable						
Test for overall effect: Z = 1.92 (P = 0.06)						
1.19.3 12 months postpartum						
Ferrara 2011	27	72	18	84	100.0%	1.75 [1.05, 2.90]
Subtotal (95% CI)		**72**		**84**	**100.0%**	**1.75 [1.05, 2.90]**
Total events	27		18			
Heterogeneity: Not applicable						
Test for overall effect: Z = 2.17 (P = 0.03)						



Test for subgroup differences: Chi² = 0.94, df = 2 (P = 0.63), I² = 0%

1.20 Fasting plasma glucose 3 months postpartum mmol/L

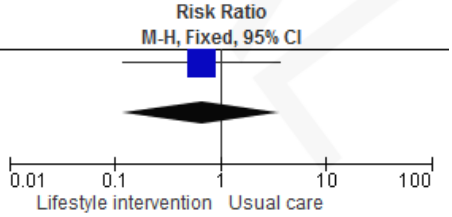
Study or Subgroup	Lifestyle intervention Mean	SD	Total	Usual care Mean	SD	Total	Weight	Mean Difference IV, Fixed, 95% CI
1.20.1 Three months postpartum								
Elnour 2008	5.07	0.2	99	5.15	0.3	66	100.0%	-0.08 [-0.16, 0.00]
Subtotal (95% CI)			**99**			**66**	**100.0%**	**-0.08 [-0.16, 0.00]**
Heterogeneity: Not applicable								
Test for overall effect: Z = 1.90 (P = 0.06)								
1.20.2 Six months postpartum								
Elnour 2008	5.33	0.2	99	5.47	0.3	66	100.0%	-0.14 [-0.22, -0.06]
Subtotal (95% CI)			**99**			**66**	**100.0%**	**-0.14 [-0.22, -0.06]**
Heterogeneity: Not applicable								
Test for overall effect: Z = 3.33 (P = 0.0009)								



Test for subgroup differences: Chi² = 1.02, df = 1 (P = 0.31), I² = 1.8%

1.21 Maternal postnatal impaired glucose tolerance

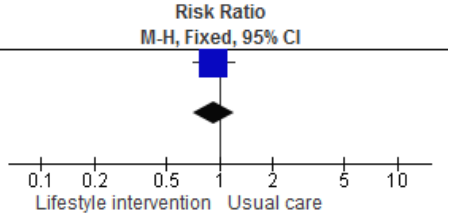
Study or Subgroup	Lifestyle intervention Events	Total	Usual care Events	Total	Weight	Risk Ratio M-H, Fixed, 95% CI
Bancroft 2000	2	28	3	28	100.0%	0.67 [0.12, 3.69]
Total (95% CI)		**28**		**28**	**100.0%**	**0.67 [0.12, 3.69]**
Total events	2		3			
Heterogeneity: Not applicable						
Test for overall effect: Z = 0.46 (P = 0.64)						



1.22 Maternal metabolic syndrome (follow-up)

Study or Subgroup	Lifestyle intervention Events	Total	Usual care Events	Total	Weight	Risk Ratio M-H, Fixed, 95% CI
Landon 2009	73	229	69	201	100.0%	0.93 [0.71, 1.22]
Total (95% CI)		**229**		**201**	**100.0%**	**0.93 [0.71, 1.22]**
Total events	73		69			

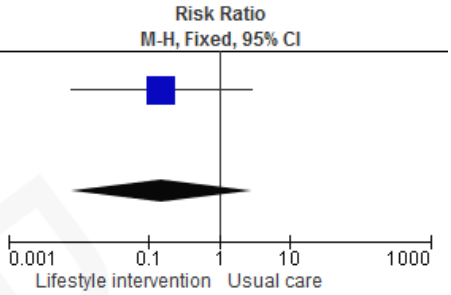
Heterogeneity: Not applicable
Test for overall effect: Z = 0.54 (P = 0.59)



1.23 Stillbirth

Study or Subgroup	Lifestyle intervention Events	Total	Usual care Events	Total	Weight	Risk Ratio M-H, Fixed, 95% CI
Bancroft 2000	0	32	0	36		Not estimable
Crowther 2005	0	506	3	524	100.0%	0.15 [0.01, 2.86]
Garner 1997	0	149	0	150		Not estimable
Landon 2009	0	485	0	473		Not estimable
Total (95% CI)		**1172**		**1183**	**100.0%**	**0.15 [0.01, 2.86]**
Total events	0		3			

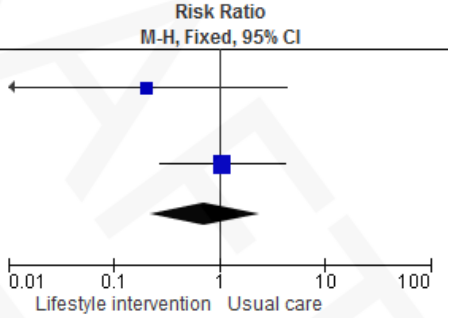
Heterogeneity: Not applicable
Test for overall effect: Z = 1.27 (P = 0.21)



1.24 Neonatal death

Study or Subgroup	Lifestyle intervention Events	Total	Usual care Events	Total	Weight	Risk Ratio M-H, Fixed, 95% CI
Bancroft 2000	0	32	0	36		Not estimable
Crowther 2005	0	506	2	524	38.8%	0.21 [0.01, 4.30]
Garner 1997	0	149	0	150		Not estimable
Landon 2009	0	485	0	473		Not estimable
Yang 2014	4	339	4	361	61.2%	1.06 [0.27, 4.22]
Total (95% CI)		**1511**		**1544**	**100.0%**	**0.73 [0.22, 2.42]**
Total events	4		6			

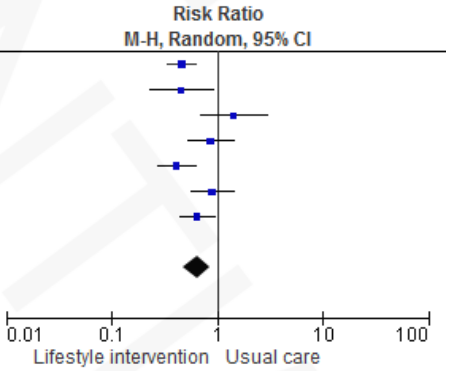
Heterogeneity: Chi² = 0.95, df = 1 (P = 0.33); I² = 0%
Test for overall effect: Z = 0.51 (P = 0.61)



1.25 Macrosomia

Study or Subgroup	Lifestyle intervention Events	Total	Usual care Events	Total	Weight	Risk Ratio M-H, Random, 95% CI
Crowther 2005 (1)	49	506	110	524	18.5%	0.46 [0.34, 0.63]
Elnour 2008 (2)	11	99	16	66	10.2%	0.46 [0.23, 0.92]
Ferrara 2011 (3)	15	96	11	101	9.8%	1.43 [0.69, 2.97]
Garner 1997 (4)	24	149	28	150	14.1%	0.86 [0.53, 1.42]
Landon 2009 (5)	28	477	65	454	15.8%	0.41 [0.27, 0.63]
Mendelson 2008 (6)	19	49	22	51	14.7%	0.90 [0.56, 1.44]
Yang 2014 (7)	38	339	63	361	17.0%	0.64 [0.44, 0.93]
Total (95% CI)		**1715**		**1707**	**100.0%**	**0.64 [0.48, 0.87]**
Total events	184		315			

Heterogeneity: Tau² = 0.10; Chi² = 17.02, df = 6 (P = 0.009); I² = 65%
Test for overall effect: Z = 2.89 (P = 0.004)

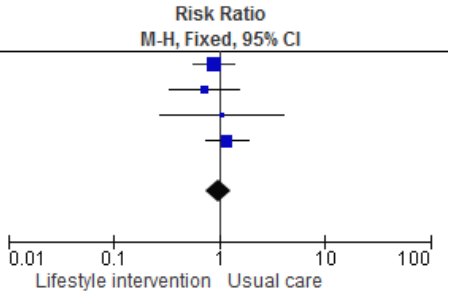


Footnotes
(1) ≥ 4 kg
(2) > 4 kg
(3) > 4 kg
(4) > 4 kg
(5) > 4 kg
(6) > 4 kg
(7) ≥ 4 kg

1.26 Small-for-gestational age

Study or Subgroup	Lifestyle intervention Events	Total	Usual care Events	Total	Weight	Risk Ratio M-H, Fixed, 95% CI
Crowther 2005	33	506	38	524	44.4%	0.90 [0.57, 1.41]
Elnour 2008	12	99	11	66	15.7%	0.73 [0.34, 1.55]
Ferrara 2011	4	96	4	101	4.6%	1.05 [0.27, 4.09]
Landon 2009	36	477	29	455	35.3%	1.18 [0.74, 1.90]
Total (95% CI)		**1178**		**1146**	**100.0%**	**0.98 [0.73, 1.31]**
Total events	85		82			

Heterogeneity: Chi² = 1.37, df = 3 (P = 0.71); I² = 0%
Test for overall effect: Z = 0.14 (P = 0.89)

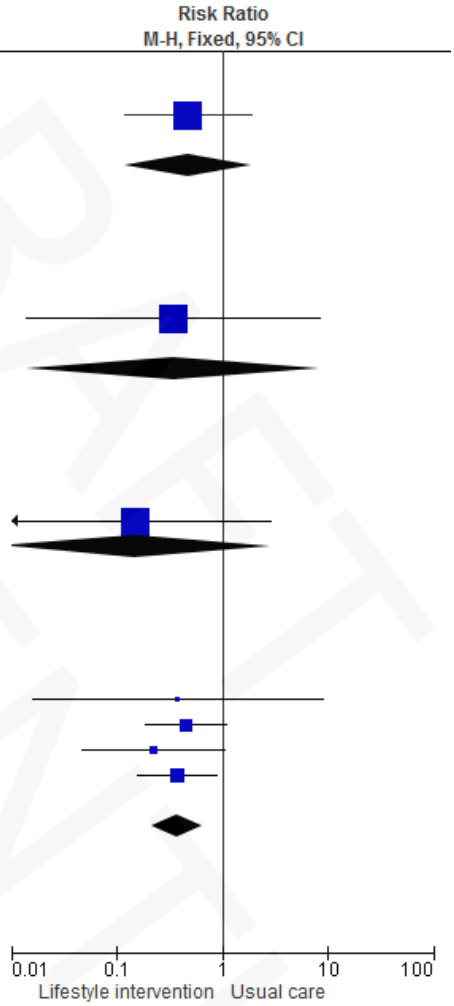


1.27 Birth trauma (shoulder dystocia, bone fracture, nerve palsy)

Study or Subgroup	Lifestyle intervention Events	Total	Usual care Events	Total	Weight	Risk Ratio M-H, Fixed, 95% CI
1.27.1 Birth trauma not specified						
Garner 1997	0	149	0	150		Not estimable
Landon 2009	3	476	6	455	100.0%	0.48 [0.12, 1.90]
Yang 2014	0	339	0	361		Not estimable
Subtotal (95% CI)		**964**		**966**	**100.0%**	**0.48 [0.12, 1.90]**
Total events	3		6			
Heterogeneity: Not applicable						
Test for overall effect: Z = 1.05 (P = 0.29)						
1.27.2 Bone fracture						
Crowther 2005	0	506	1	524	100.0%	0.35 [0.01, 8.45]
Yang 2014	0	339	0	361		Not estimable
Subtotal (95% CI)		**845**		**885**	**100.0%**	**0.35 [0.01, 8.45]**
Total events	0		1			
Heterogeneity: Not applicable						
Test for overall effect: Z = 0.65 (P = 0.51)						
1.27.3 Nerve palsy						
Crowther 2005	0	506	3	524	100.0%	0.15 [0.01, 2.86]
Subtotal (95% CI)		**506**		**524**	**100.0%**	**0.15 [0.01, 2.86]**
Total events	0		3			
Heterogeneity: Not applicable						
Test for overall effect: Z = 1.27 (P = 0.21)						
1.27.4 Shoulder dystocia						
Bancroft 2000	0	32	1	36	3.3%	0.37 [0.02, 8.86]
Crowther 2005	7	506	16	524	36.8%	0.45 [0.19, 1.09]
Elnour 2008	2	99	6	66	16.8%	0.22 [0.05, 1.07]
Landon 2009	7	476	18	455	43.1%	0.37 [0.16, 0.88]
Yang 2014	0	339	0	361		Not estimable
Subtotal (95% CI)		**1452**		**1442**	**100.0%**	**0.38 [0.21, 0.66]**
Total events	16		41			

Heterogeneity: Chi² = 0.60, df = 3 (P = 0.90); I² = 0%
Test for overall effect: Z = 3.41 (P = 0.0006)

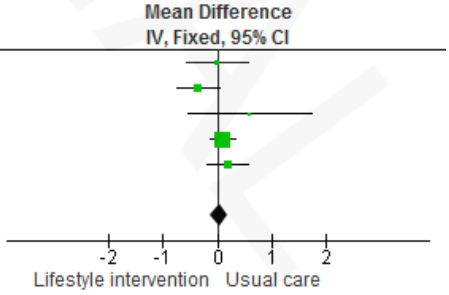
Test for subgroup differences: Chi² = 0.50, df = 3 (P = 0.92), I² = 0%



1.28 Gestational age at birth (weeks)

Study or Subgroup	Lifestyle intervention Mean	SD	Total	Usual care Mean	SD	Total	Weight	Mean Difference IV, Fixed, 95% CI
Bancroft 2000	39	1.25	32	39	1.15	36	8.3%	0.00 [-0.57, 0.57]
Garner 1997	38.79	1.77	149	39.14	1.64	150	18.2%	-0.35 [-0.74, 0.04]
Gillen 2004	39.5	2	16	38.9	1.2	16	2.1%	0.60 [-0.54, 1.74]
Landon 2009	39	1.8	485	38.9	1.8	473	52.3%	0.10 [-0.13, 0.33]
Yang 2014	39.4	2.9	339	39.2	2.1	361	19.1%	0.20 [-0.18, 0.58]
Total (95% CI)			**1021**			**1036**	**100.0%**	**0.04 [-0.13, 0.20]**

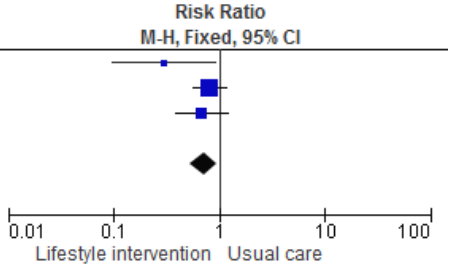
Heterogeneity: Chi² = 5.80, df = 4 (P = 0.21); I² = 31%
Test for overall effect: Z = 0.47 (P = 0.64)



1.29 Preterm birth (< 37 weeks' gestation; and < 32 weeks' gestation)

Study or Subgroup	Lifestyle intervention Events	Total	Usual care Events	Total	Weight	Risk Ratio M-H, Fixed, 95% CI
Elnour 2008	4	99	9	66	11.7%	0.30 [0.10, 0.92]
Landon 2009	45	477	53	455	58.9%	0.81 [0.56, 1.18]
Yang 2014	18	339	28	361	29.4%	0.68 [0.39, 1.21]
Total (95% CI)		**915**		**882**	**100.0%**	**0.71 [0.53, 0.96]**
Total events	67		90			

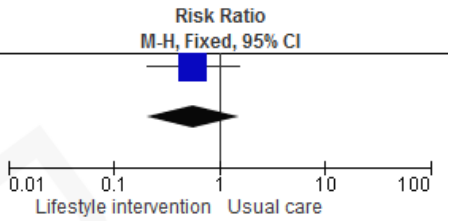
Heterogeneity: Chi² = 2.76, df = 2 (P = 0.25); I² = 27%
Test for overall effect: Z = 2.21 (P = 0.03)



1.30 Five-minute Apgar less than seven

Study or Subgroup	Lifestyle intervention Events	Total	Usual care Events	Total	Weight	Risk Ratio M-H, Fixed, 95% CI
Crowther 2005	6	506	11	524	100.0%	0.56 [0.21, 1.52]
Total (95% CI)		**506**		**524**	**100.0%**	**0.56 [0.21, 1.52]**
Total events	6		11			

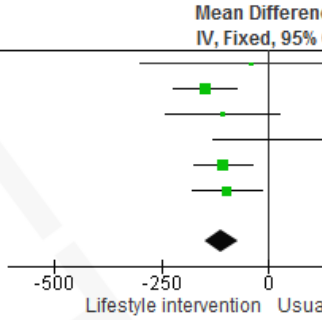
Heterogeneity: Not applicable
Test for overall effect: Z = 1.13 (P = 0.26)



1.31 Birthweight (grams)

Study or Subgroup	Lifestyle intervention Mean	SD	Total	Usual care and diet only Mean	SD	Total	Weight	Mean Difference IV, Fixed, 95% CI
Bancroft 2000	3,580	550	32	3,620	550	36	2.3%	-40.00 [-301.90, 221.90]
Crowther 2005	3,335	551	506	3,482	660	524	29.3%	-147.00 [-221.15, -72.85]
Garner 1997	3,437	575	149	3,544	601	150	9.1%	-107.00 [-240.32, 26.32]
Jovanovic-Peterson 1989 (1)	3,634	317	10	3,465	343	9	1.8%	169.00 [-129.02, 467.02]
Landon 2009	3,302	502.4	485	3,408	589.4	473	33.4%	-106.00 [-175.43, -36.57]
Yang 2014	3,371	530	339	3,469	574	361	24.1%	-98.00 [-179.79, -16.21]
Total (95% CI)			**1521**			**1553**	**100.0%**	**-109.64 [-149.77, -69.51]**

Heterogeneity: Chi² = 4.69, df = 5 (P = 0.45); I² = 0%
Test for overall effect: Z = 5.35 (P < 0.00001)

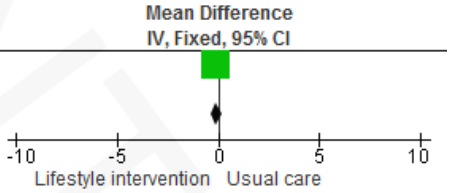


Footnotes
(1) Control group was diet only

1.32 Length (cm)

Study or Subgroup	Lifestyle intervention Mean	SD	Total	Usual care Mean	SD	Total	Weight	Mean Difference IV, Fixed, 95% CI
Yang 2014	50.1	1.8	339	50.2	1.9	361	100.0%	-0.10 [-0.37, 0.17]
Total (95% CI)			**339**			**361**	**100.0%**	**-0.10 [-0.37, 0.17]**

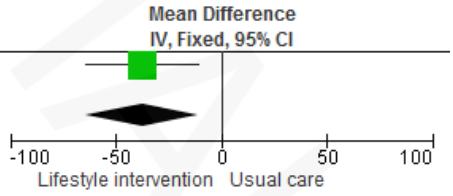
Heterogeneity: Not applicable
Test for overall effect: Z = 0.72 (P = 0.47)



1.33 Adiposity (Neonatal fat mass (g))

Study or Subgroup	Lifestyle intervention Mean	SD	Total	Usual care Mean	SD	Total	Weight	Mean Difference IV, Fixed, 95% CI
Landon 2009	427	197.9	485	464.3	222.3	473	100.0%	-37.30 [-63.97, -10.63]
Total (95% CI)			**485**			**473**	**100.0%**	**-37.30 [-63.97, -10.63]**

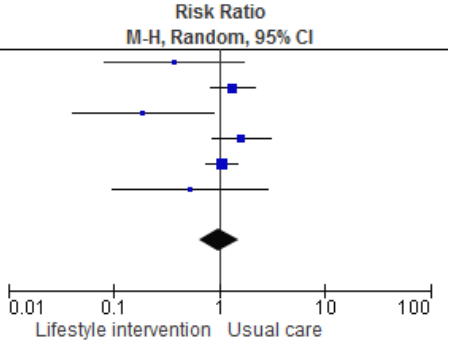
Heterogeneity: Not applicable
Test for overall effect: Z = 2.74 (P = 0.006)



1.34 Neonatal hypoglycaemia

Study or Subgroup	Lifestyle intervention Events	Total	Usual care Events	Total	Weight	Risk Ratio M-H, Random, 95% CI
Bancroft 2000	2	32	6	36	6.6%	0.38 [0.08, 1.73]
Crowther 2005	35	506	27	524	27.0%	1.34 [0.82, 2.18]
Elnour 2008	2	99	7	66	6.5%	0.19 [0.04, 0.89]
Garner 1997	21	149	13	150	21.1%	1.63 [0.85, 3.13]
Landon 2009	62	381	55	357	33.1%	1.06 [0.76, 1.47]
Yang 2014	2	339	4	361	5.6%	0.53 [0.10, 2.89]
Total (95% CI)		**1506**		**1494**	**100.0%**	**0.99 [0.65, 1.52]**
Total events	124		112			

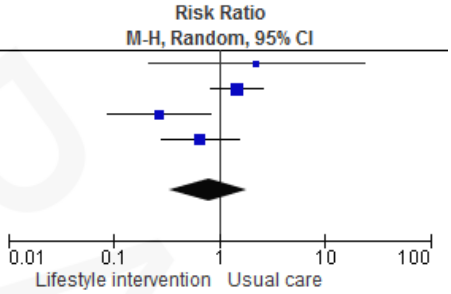
Heterogeneity: Tau² = 0.12; Chi² = 9.70, df = 5 (P = 0.08); I² = 48%
Test for overall effect: Z = 0.04 (P = 0.97)



1.35 Respiratory distress syndrome

Study or Subgroup	Lifestyle intervention Events	Total	Usual care Events	Total	Weight	Risk Ratio M-H, Random, 95% CI
Bancroft 2000	2	32	1	36	9.9%	2.25 [0.21, 23.66]
Crowther 2005	27	506	19	524	35.6%	1.47 [0.83, 2.61]
Elnour 2008	4	99	10	66	24.4%	0.27 [0.09, 0.81]
Landon 2009	9	477	13	455	30.0%	0.66 [0.29, 1.53]
Total (95% CI)		**1114**		**1081**	**100.0%**	**0.79 [0.34, 1.85]**
Total events	42		43			

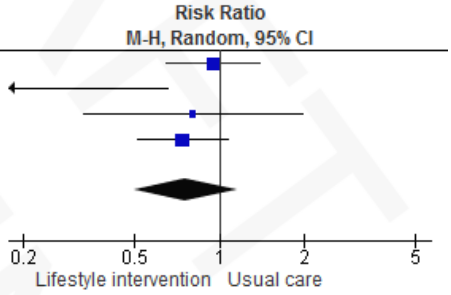
Heterogeneity: Tau² = 0.44; Chi² = 8.44, df = 3 (P = 0.04); I² = 64%
Test for overall effect: Z = 0.53 (P = 0.59)



1.36 Neonatal jaundice (hyperbilirubinaemia)

Study or Subgroup	Lifestyle intervention Events	Total	Usual care Events	Total	Weight	Risk Ratio M-H, Random, 95% CI
Crowther 2005 (1)	44	506	48	524	39.6%	0.95 [0.64, 1.40]
Elnour 2008	1	99	8	66	3.9%	0.08 [0.01, 0.65]
Garner 1997	8	149	10	150	16.1%	0.81 [0.33, 1.98]
Landon 2009	43	450	54	418	40.4%	0.74 [0.51, 1.08]
Total (95% CI)		**1204**		**1158**	**100.0%**	**0.76 [0.50, 1.16]**
Total events	96		120			

Heterogeneity: Tau² = 0.08; Chi² = 5.63, df = 3 (P = 0.13); I² = 47%
Test for overall effect: Z = 1.28 (P = 0.20)

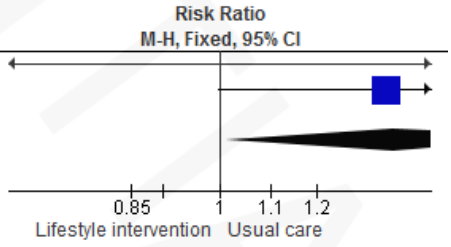


Footnotes
(1) Requiring phototherapy

1.37 Hypocalcaemia

Study or Subgroup	Lifestyle intervention Events	Total	Usual care Events	Total	Weight	Risk Ratio M-H, Fixed, 95% CI
Elnour 2008	3	99	1	66	2.6%	2.00 [0.21, 18.82]
Garner 1997	61	149	45	150	97.4%	1.36 [1.00, 1.86]
Total (95% CI)		**248**		**216**	**100.0%**	**1.38 [1.01, 1.88]**
Total events	64		46			

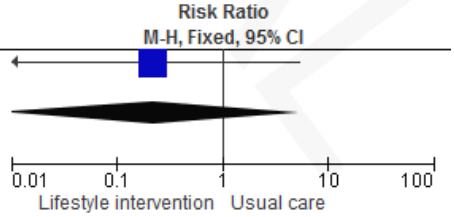
Heterogeneity: Chi² = 0.11, df = 1 (P = 0.74); I² = 0%
Test for overall effect: Z = 2.05 (P = 0.04)



1.38 Polycythemia

Study or Subgroup	Lifestyle intervention Events	Total	Usual care Events	Total	Weight	Risk Ratio M-H, Fixed, 95% CI
Elnour 2008	0	99	1	66	100.0%	0.22 [0.01, 5.40]
Total (95% CI)		**99**		**66**	**100.0%**	**0.22 [0.01, 5.40]**
Total events	0		1			

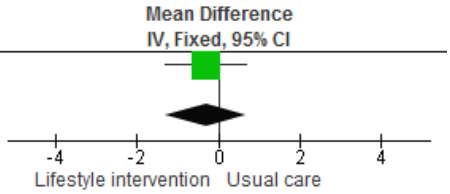
Heterogeneity: Not applicable
Test for overall effect: Z = 0.92 (P = 0.36)



1.39 Childhood weight (kg)

Study or Subgroup	Lifestyle intervention Mean	SD	Total	Usual care Mean	SD	Total	Weight	Mean Difference IV, Fixed, 95% CI
Crowther 2005	19.1	2.9	94	19.4	4.2	105	100.0%	-0.30 [-1.29, 0.69]
Total (95% CI)			**94**			**105**	**100.0%**	**-0.30 [-1.29, 0.69]**

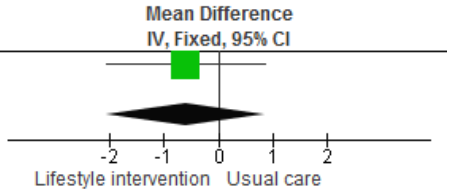
Heterogeneity: Not applicable
Test for overall effect: Z = 0.59 (P = 0.55)



1.40 Childhood height (cm)

Study or Subgroup	Lifestyle intervention Mean	SD	Total	Usual care Mean	SD	Total	Weight	Mean Difference IV, Fixed, 95% CI
Crowther 2005	107.9	4.6	94	108.5	5.8	105	100.0%	-0.60 [-2.05, 0.85]
Total (95% CI)			**94**			**105**	**100.0%**	**-0.60 [-2.05, 0.85]**

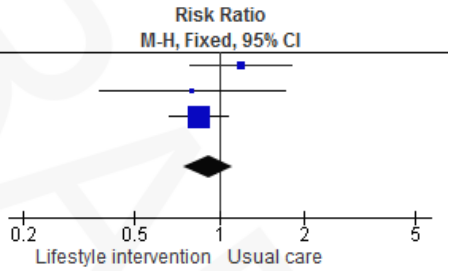
Heterogeneity: Not applicable
Test for overall effect: Z = 0.81 (P = 0.42)



1.41 Adiposity (Childhood BMI > 85th percentile)

Study or Subgroup	Lifestyle intervention Events	Total	Usual care Events	Total	Weight	Risk Ratio M-H, Fixed, 95% CI
Crowther 2005	31	94	29	105	20.5%	1.19 [0.78, 1.82]
Garner 1997	11	43	8	25	7.6%	0.80 [0.37, 1.72]
Landon 2009	86	264	91	236	71.9%	0.84 [0.67, 1.07]
Total (95% CI)		**401**		**366**	**100.0%**	**0.91 [0.75, 1.11]**
Total events	128		128			

Heterogeneity: Chi² = 2.07, df = 2 (P = 0.35); I² = 4%
Test for overall effect: Z = 0.90 (P = 0.37)

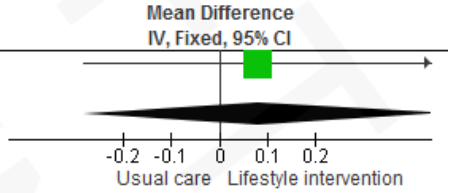


1.42 Adiposity (BMI Z score childhood)

Study or Subgroup	Lifestyle intervention Mean	SD	Total	Usual care Mean	SD	Total	Weight	Mean Difference IV, Fixed, 95% CI
Crowther 2005 (1)	0.49	1.2	94	0.41	1.4	105	100.0%	0.08 [-0.28, 0.44]
Total (95% CI)			**94**			**105**	**100.0%**	**0.08 [-0.28, 0.44]**

Heterogeneity: Not applicable
Test for overall effect: Z = 0.43 (P = 0.66)

Footnotes
(1) Age 4 to 5 years



1.43 Childhood glycaemic control (mmol/L)

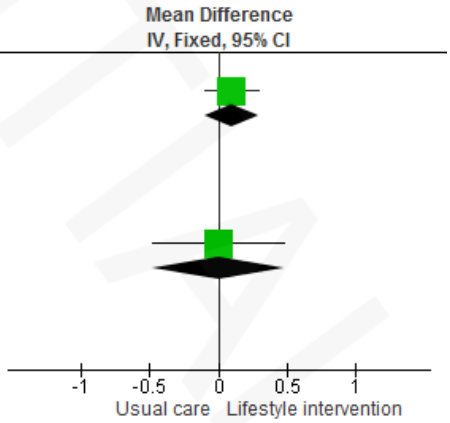
Study or Subgroup	Lifestyle intervention Mean	SD	Total	Usual care Mean	SD	Total	Weight	Mean Difference IV, Fixed, 95% CI
1.43.1 Fasting blood glucose								
Garner 1997	5	0.4	43	4.9	0.4	25	100.0%	0.10 [-0.10, 0.30]
Subtotal (95% CI)			**43**			**25**	**100.0%**	**0.10 [-0.10, 0.30]**

Heterogeneity: Not applicable
Test for overall effect: Z = 0.99 (P = 0.32)

1.43.2 Two-hour postprandial blood glucose								
Garner 1997	5.9	1.2	43	5.9	0.83	25	100.0%	0.00 [-0.48, 0.48]
Subtotal (95% CI)			**43**			**25**	**100.0%**	**0.00 [-0.48, 0.48]**

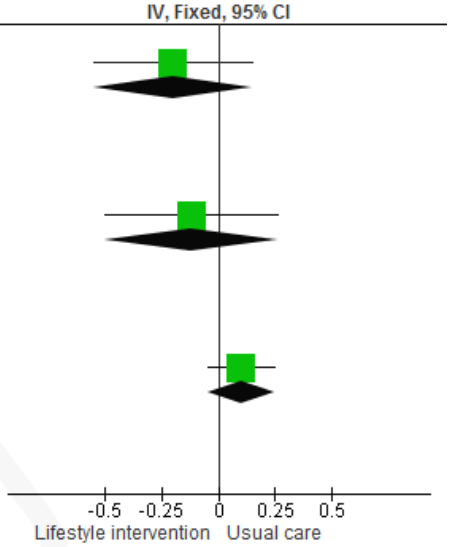
Heterogeneity: Not applicable
Test for overall effect: Z = 0.00 (P = 1.00)

Test for subgroup differences: Chi² = 0.14, df = 1 (P = 0.71), I² = 0%



1.44 Dyslipidaemia or metabolic syndrome (Childhood cholesterol (mg/dL))

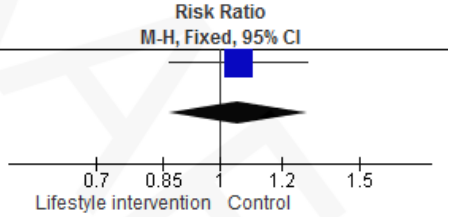
Study or Subgroup	Lifestyle intervention Mean	SD	Total	Usual care Mean	SD	Total	Weight	Mean Difference IV, Fixed, 95% CI
1.44.1 Total cholesterol								
Garner 1997	4.2	0.5	43	4.4	0.8	25	100.0%	-0.20 [-0.55, 0.15]
Subtotal (95% CI)			**43**			**25**	**100.0%**	**-0.20 [-0.55, 0.15]**
Heterogeneity: Not applicable								
Test for overall effect: Z = 1.13 (P = 0.26)								
1.44.2 LDL cholesterol								
Garner 1997	2.4	0.5	43	2.52	0.9	25	100.0%	-0.12 [-0.50, 0.26]
Subtotal (95% CI)			**43**			**25**	**100.0%**	**-0.12 [-0.50, 0.26]**
Heterogeneity: Not applicable								
Test for overall effect: Z = 0.61 (P = 0.54)								
1.44.3 HDL cholesterol								
Garner 1997	1.4	0.3	43	1.3	0.3	25	100.0%	0.10 [-0.05, 0.25]
Subtotal (95% CI)			**43**			**25**	**100.0%**	**0.10 [-0.05, 0.25]**
Heterogeneity: Not applicable								
Test for overall effect: Z = 1.33 (P = 0.19)								



Test for subgroup differences: Chi² = 3.13, df = 2 (P = 0.21), I² = 36.1%

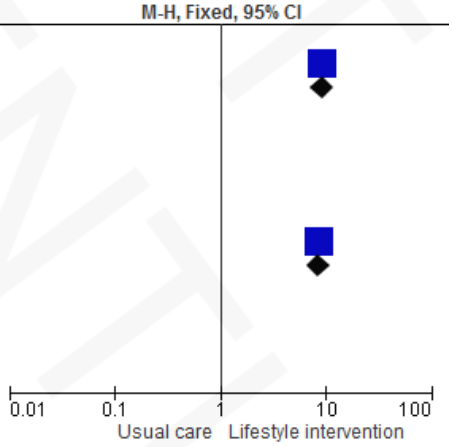
1.45 Number of antenatal visits or admissions

Study or Subgroup	Specific treatment Events	Total	Routine ANC Events	Total	Weight	Risk Ratio M-H, Fixed, 95% CI
Crowther 2005	141	490	139	510	100.0%	1.06 [0.87, 1.29]
Total (95% CI)		**490**		**510**	**100.0%**	**1.06 [0.87, 1.29]**
Total events	141		139			
Heterogeneity: Not applicable						
Test for overall effect: Z = 0.54 (P = 0.59)						



1.46 Number of hospital or health professional visits (including midwife, obstetrician, physician, dietician, diabetic nurse)

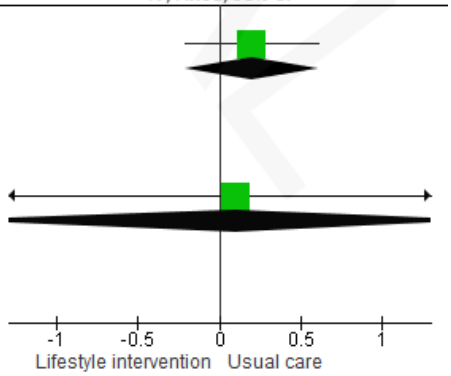
Study or Subgroup	Lifestyle intervention Events	Total	Usual care Events	Total	Weight	Risk Ratio M-H, Fixed, 95% CI
1.46.1 Dietitian						
Crowther 2005	453	490	51	510	100.0%	9.24 [7.12, 12.01]
Subtotal (95% CI)		**490**		**510**	**100.0%**	**9.24 [7.12, 12.01]**
Total events	453		51			
Heterogeneity: Not applicable						
Test for overall effect: Z = 16.66 (P < 0.00001)						
1.46.2 Diabetes educator						
Crowther 2005	460	490	56	510	100.0%	8.55 [6.67, 10.96]
Subtotal (95% CI)		**490**		**510**	**100.0%**	**8.55 [6.67, 10.96]**
Total events	460		56			
Heterogeneity: Not applicable						
Test for overall effect: Z = 16.95 (P < 0.00001)						



Test for subgroup differences: Chi² = 0.18, df = 1 (P = 0.67), I² = 0%

1.47 Number of hospital or health professional visits (including midwife, obstetrician, physician, dietician, diabetic nurse)l

Study or Subgroup	Lifestyle intervention Mean	SD	Total	Usual care Mean	SD	Total	Weight	Mean Difference IV, Fixed, 95% CI
1.47.1 Obstetrician								
Yang 2014	7.8	3	339	7.6	2.5	361	100.0%	0.20 [-0.21, 0.61]
Subtotal (95% CI)			**339**			**361**	**100.0%**	**0.20 [-0.21, 0.61]**
Heterogeneity: Not applicable								
Test for overall effect: Z = 0.95 (P = 0.34)								
1.47.2 Healthcare provider (not specified)								
Ferrara 2011	11.1	5.9	96	11	6.1	101	100.0%	0.10 [-1.58, 1.78]
Subtotal (95% CI)			**96**			**101**	**100.0%**	**0.10 [-1.58, 1.78]**
Heterogeneity: Not applicable								
Test for overall effect: Z = 0.12 (P = 0.91)								



Test for subgroup differences: Chi² = 0.01, df = 1 (P = 0.91), I² = 0%

1.48 Admission to neonatal intensive care unit/nursery

Study or Subgroup	Lifestyle intervention Events	Total	Usual care Events	Total	Weight	Risk Ratio M-H, Random, 95% CI
Bancroft 2000	2	32	6	36	6.9%	0.38 [0.08, 1.73]
Crowther 2005	357	506	321	524	54.4%	1.15 [1.05, 1.26]
Landon 2009	43	477	53	455	38.7%	0.77 [0.53, 1.13]
Total (95% CI)		**1015**		**1015**	**100.0%**	**0.91 [0.59, 1.40]**
Total events	402		380			

Heterogeneity: Tau² = 0.09; Chi² = 6.61, df = 2 (P = 0.04); I² = 70%
Test for overall effect: Z = 0.41 (P = 0.68)