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# The Dietary Management of Diabetic Pregnancies

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## INTRODUCTION

There are general nutritional principles that apply to all pregnancies and there are specific nutritional issues that surround the management of pregnant women with diabetes. The nutritional needs of women with pre-existing Type 1 diabetes and Type 2 diabetes differ, as do those for women who become glucose-intolerant in pregnancy. Ideally nutritional advice should start before pregnancy and continue throughout the pregnancy, being modified as necessary at each antenatal visit. The dietitian is an integral part of the multidisciplinary diabetic–obstetric team and should be involved in all aspects of the patient's care plan. The prescribed diet has to accommodate the metabolic and physiological changes associated with a diabetic pregnancy and the dietitian must be familiar with these changes.

Over the last few decades the Western antenatal population has become older, more obese, less physically active and more ethnically diverse. These demographic changes explain the rise in the numbers of pregnant women with pre-existing Type 2 diabetes and gestational diabetes (1). Although the actual number of pregnant women with pre-existing Type 1 diabetes has remained relatively constant, the duration of diabetes prior to pregnancy has increased, due to women delaying childbirth for personal reasons and the earlier onset of Type 1 diabetes that has occurred over recent years. Both the age of the mother and the duration of her diabetes contribute to the clinical

*Nutritional Management of Diabetes Mellitus*. Edited by G. Frost, A. Dornhorst and R. Moses
 ISBN 0 471 49751 7

complications encountered in a Type 1 diabetic pregnancy. Although pregnancy outcomes continue to improve in women with Type 1 diabetes, perinatal morbidity and mortality remain fourfold higher than for the non-diabetic population (2,3).

Active dietary management for all types of diabetic pregnancies can lessen complications during pregnancy and improve pregnancy outcome for the mother and her child.

## CONSEQUENCES OF A DIABETIC PREGNANCY

Maternal hyperglycaemia results in an excess maternal–foetal transfer of glucose. The placental glucose transporter protein, GLUT1, is increased in diabetic pregnancies, and maternal hyperglycaemia quickly results in foetal hyperglycaemia and foetal hyperinsulinaemia (4). Maternal hyperglycaemia is not only a critical factor in glucose-mediated congenital malformations, but also in many aspects of foetal development, neonatal well-being and future health, see Table 7.1.

An accelerated foetal growth pattern and a large-for-gestational-age (LGA) infant at birth is the hallmark of a poorly controlled diabetic pregnancy. Foetal insulin is the main foetal anabolic hormone and hyperinsulinaemia can cause excess fat accumulation, organomegaly, especially of the heart and liver, and high birthweight. An LGA infant is a potential cause for birth trauma and a high Caesarean rate. Foetal hyperinsulinaemia is also believed to contribute to adverse foetal metabolic complications in late pregnancy including a tendency to high lactate levels and an increased risk of stillbirth. Foetal hyperinsulinaemia at delivery can cause transient hypoglycaemia and hypocalcaemia. There is increasing and tantalising evidence that by optimising maternal glycaemia and avoiding foetal hyperinsulinaemia one can reduce the long-term risk of the child becoming obese and insulin-resistant in adult life (5).

**Table 7.1** The intrauterine influence of maternal hyperglycaemia on foetal and childhood development

| Period of influence | Consequence of maternal hyperglycaemia |
|---|---|
| First trimester | Congenital malformations |
| Second trimester | Foetal cell programming, foetal hyperinsulinaemia |
| Third trimester | Accelerated foetal growth and stillbirth |
| Neonatal period | Transient hypoglycaemia; hypocalcaemia and cardiomyopathy |
| Adolescence | Obesity, impaired glucose tolerance and insulin resistance |
| Adulthood | Insulin resistance, obesity and Type 2 diabetes |

## THE THERAPEUTIC AIM IN THE MANAGEMENT OF DIABETIC PREGNANCIES

The aim in the management of all diabetic pregnancies is to achieve normoglycaemia while avoiding maternal hypoglycaemia. This approach will optimise foetal growth and minimise short- and long-term complications. As the immediate post-prandial period is when maternal glucose levels are at their highest, dietary and insulin therapies need to specifically target this time (6). The glycaemic targets for all types of diabetic pregnancies should be the same, namely a fasting glucose of $< 5$ mmol/l and a 1 h post-prandial glucose $< 7.8$ mmol/l. While these goals will inevitably require insulin in women with pre-pregnancy diabetes, many women with gestational diabetes (GDM) will be able to achieve them with dietary intervention alone, with insulin being reserved for women who, after a trial of dietary therapy, are above these glycaemic target values. The use of oral agents that do not cross the placenta, such as glibenclamide, in the management of GDM, although probably safe, are best suited for women with Type 2 diabetes and GDM in areas of the world where insulin availability is limited (7).

Due to the lack of adequate controlled dietary studies in diabetic pregnancies, conflicting dietary advice is often advocated. Debate still surrounds the total energy content of the diet and the optimal proportions and type of dietary carbohydrate and fat to be prescribed. The benefits, if any, of whether the dietary advice given during pregnancy actually leads to behavioural changes that reduce the future recurrence of GDM or the development of diabetes in the mother are unknown. Also the influence of maternal diets on foetal programming and the future risk of childhood and adult obesity and diabetes are not fully understood.

## PRECONCEPTION NUTRITIONAL COUNSELLING IN DIABETIC PREGNANCIES

All women attempting pregnancy should take a minimum of 400 μg folic acid supplements a day to prevent neural tube defects (8). The higher dose of 5 mg folic acid a day is frequently recommended for diabetic women, despite any actual trial evidence for this, the rationale being that neural tube defects are commoner in this group. In Britain, where the dietary folate intakes are relatively low despite numerous public health campaigns, less than 10% of women actually take folate supplements in early pregnancy (9).

The preconception period is a time when women with diabetes are encouraged to achieve the best glycaemic control possible. Congenital malformations account for approximately 40% of all diabetic perinatal

mortality, and can be significantly reduced when $HbA_{1c}$ levels are within the normal range. To achieve this insulin regimens usually need to be intensified and many women with Type 2 diabetes will be started on insulin for the first time. Dietetic input is required to build confidence, reduce hypoglycaemia and limit unnecessary weight gain (10).

Achieving near-normal glycaemic control is possible in most women with Type 2 and Type 1 diabetes. However, in women with a long duration of Type 1 diabetes and significant autonomic neuropathy the risk of severe hypoglycaemia is high. Poor hypoglycaemia awareness and impaired counter-regulatory hormonal responses increase with the duration of Type 1 diabetes. Dietary advice is essential to ensure adequate carbohydrate is being taken with each meal and that suitable low glycaemic carbohydrate snacks are being consumed between meals.

The preconception period is a good time to encourage weight loss and exercise in obese women with pre-existing Type 2 diabetes or a previous history of gestational diabetes. Maternal obesity is independently associated with increased perinatal morbidity and mortality rates (11). Epidemiological studies suggest that when obesity and diabetes coexist an adverse synergistic effect on pregnancy outcome occurs, including an unexplained increase in congenital malformation rates (12–14). Potentially a weight-reducing diet in obese women prior to conception will improve both glycaemic control and pregnancy outcome.

## GENERAL DIETETIC ADVICE FOR PREGNANCY

Once pregnancy has been confirmed the diet should be reviewed to ensure the recommended vitamin and mineral intakes, including folate and iron, for pregnancy are met. Ensuring adequate amounts of antioxidants in the diet may help to lessen the risk of pre-eclampsia and congenital malformation. Recently dietary supplementation with the antioxidant vitamins C and E have been shown to reduce the incidence of pre-eclampsia in high-risk women (15). Animal, but so far not human, studies have shown that these vitamins also protect embryos from the teratogenic effects of hyperglycaemia (16).

Calcium and vitamin D supplements during both pregnancy and lactation should be considered for Indian/Asian women and others with poor sunlight exposure or low calcium intakes (17,18). Observational studies have linked low vitamin D levels with insulin resistance and diabetes (19,20) and, given the high incidence of diabetes among Asian women, ensuring adequate vitamin D in the diet seems prudent.

All women should be reminded of the dangers of excess alcohol (21), and the potentially harmful effects of uncooked meats and soft cheese.

## RECOMMENDED MATERNAL WEIGHT GAINS IN NON-DIABETIC PREGNANCIES

Optimal weight gain for pregnancy needs to reflect the woman's pre-pregnancy weight (22). The guidelines on recommended maternal weight gains are based on large obstetric surveys in non-diabetic women in the United States (23). The maternal weight gain required to minimise the frequency of small-for-gestational-age (SGA) infants is higher for underweight (BMI $< 19.8\,kg/m^2$) than overweight or obese women, see Table 7.2. As the majority of women with pre-existing Type 2 and GDM are already obese it is important that the dietary advice given does not result in higher post-partum than pre-pregnancy weights.

When the pre-pregnancy BMI is $> 35\,kg/m^2$, the risk of a SGA infant is low and even when little or no maternal weight gain occurs the risk of a SGA infant does not appear to increase (11). Overweight (BMI 26.1–$29\,kg/m^2$) and obese (BMI $> 29\,kg/m^2$) women are more likely to give birth to a LGA infant than normal weight women and this risk increases with increasing maternal weight gain. The US obstetric recommendation for a minimum 7 kg weight gain for all obese women (23,24) may not be universally appropriate (11,25). Nutritional advice given in pregnancy should include appropriate weight gain targets set in early pregnancy and based on pre-pregnancy weight.

## ENERGY REQUIREMENTS IN PREGNANCY

Pregnancy is an anabolic state requiring energy for the products of conception, the foetal–placental unit and the increase in maternal tissues. Newly synthesised maternal tissues account for a 15–26% increase in metabolic rate in pregnancy (26). The total calculated energy cost for pregnancy is around 355 640 kJ (85 000 kcal) and this translates into an extra 1191.3 kJ (285 kcal) a day (27,28). These theoretical energy costs, originally derived in the 1960s by Hytten and Leitch, have been confirmed by more recent physiological measurements (29).

Maternal physiology is highly adaptable and pregnancy can progress during times of extreme food deprivation and/or physical activity (30). Under adverse

**Table 7.2** The 1990 guidelines of the United States Institute of Medicine on maternal weight gain targets according to pre-pregnancy BMI

| | Underweight $< 19.8\,kg/m^2$ | Normal weight 19.8–$26\,kg/m^2$ | Overweight $> 26\,kg/m^2$ |
|---|---|---|---|
| Weight gain term target | 12.5–18 kg | 11.5–16 kg | 7.0–11.5 kg |

environmental conditions maternal adipose deposition is limited, and diet-induced thermogenesis can fall which, when combined with a small decrease in physical activity, can conserve sufficient energy for foetal development (29,31,32). With extreme calorie restriction in the first half of pregnancy, maternal basal metabolic rate can also fall (30).

The energy requirements of pregnancy are seldom, if ever, met by increased dietary intake as shown by cross-sectional and longitudinal nutritional studies (30). Well-nourished women only obtain 20% of their pregnancy energy requirement from increased dietary intake (29). In fact no increase in maternal energy intake is required providing maternal physical activity falls by 20% during pregnancy (27). In women with high physical energy expenditures before pregnancy decreases in physical activity contribute significantly to the overall energy costs of the pregnancy (29,33). Despite these observational studies many of the dietary recommendations for pregnancy are based on providing the total energy costs of pregnancy from increased energy intake (34).

## METABOLIC CHANGES IN NON-DIABETIC PREGNANCY

Metabolic changes occur throughout pregnancy to ensure optimal foetal growth. Maternal glucose is the primary foetal oxidative substrate (35) and by late pregnancy 17–26 g glucose is metabolised per day (36). The maternal respiratory quotient rises during pregnancy as foetal carbohydrate metabolism increases (29). Metabolic changes occur to maximise the maternal–foetal transfer of glucose. Several placental hormones are lipolytic and increase maternal circulating free fatty acids that increase maternal peripheral insulin resistance (37). This increase in maternal insulin resistance diverts glucose away from maternal peripheral tissues to the foetus (35,38,39,40). Post-prandial glucose and insulin concentrations rise during pregnancy in women consuming a typical Western diet. The ability to remain glucose-tolerant while pregnant requires a trebling of insulin secretion by the end of pregnancy to counter this increase in insulin resistance (41). Observational studies suggest that habitual diet and lifestyle factors can influence maternal glucose tolerance and insulin sensitivity in pregnancy (42). Active women consuming low glycaemic index diets have significantly lower post-prandial glucose and insulin levels in pregnancy than women consuming high glycaemic index diets (43,44).

The higher post-prandial insulin levels encountered in pregnancy facilitate maternal fat deposition (45,46), which in well-nourished women approximates to a minimum of 4 kg of adipose tissue (46) and in undernourished women to 2 kg (32). A fall in fatty acid oxidation in late pregnancy also contributes to adipose deposition (29).

Other maternal metabolic changes occur to ensure a steady supply of glucose to the foetus. Lipolytic placental hormones increase maternal lipolysis during the post-absorbative periods, generating sufficient gluconeogenic substrates in the form of ketone bodies and glycerol to provide the necessary glucose for foetal use (45). An increase in maternal hepatic glucose output ensures a necessary glucose supply to the foetus during fasting (48,49). Although ketone bodies can cross the placenta and be used as foetal fuels, non-esterified acids cannot.

## SPECIFIC METABOLIC CHANGES ASSOCIATED WITH TYPE 1 DIABETES

Dietary factors, insulin adjustments and blood glucose values are so interdependent in women with Type 1 diabetes that one should not consider any one in isolation. Women with Type 1 diabetes have an absolute deficiency of insulin and their glycaemic control is totally dependent on exogenous insulin and dietary intake. The metabolic and physiological changes occurring in early pregnancy make these women especially vulnerable to hypoglycaemia, and this is further compounded if food intake falls due to pregnancy-induced nausea or vomiting. In later pregnancy, due to the increase in maternal lipolysis during the post-absorbative and fasting periods, ketoacidosis may develop rapidly. To minimise metabolic complications one needs to continually match and adjust the insulin doses to the carbohydrate intake. Maternal ketosis, as assessed by urine strips, is usually an indication for an increase in both dietary carbohydrate and insulin treatment.

Diets need to be individual and flexible enough to adjust to any of the numerous co-morbidities encountered in pregnancy, such as hyperemesis gravidarum or gastroparesis. If nausea is a problem in early pregnancy the use of liquid meals should be considered, as these are often better tolerated than solids. Going without regular food and insulin in this group is not an option.

## SPECIFIC METABOLIC CHANGES ASSOCIATED WITH TYPE 2 DIABETES

Women with Type 2 diabetes have a relative rather than an absolute deficiency of insulin. These women are already insulin-resistant and with the physiological increase in insulin resistance that occurs in pregnancy their insulin deficiency is further compromised. Very large doses of exogenous insulin are often required to obtain the necessary blood glucose target values. Avoiding excessive weight gain in these obese women being treated with large insulin doses requires considerable dietary education and intervention early in

pregnancy. The use of low-calorie foods and snacks should be encouraged. Appropriate weight targets should be set and a degree of energy restriction considered, see below.

## SPECIFIC METABOLIC CHANGES ASSOCIATED WITH GESTATIONAL DIABETES

A degree of β-cell dysfunction is universal in women with GDM, both during and following pregnancy (50–52). Women who develop GDM not only have insufficient β-cell reserve to remain glucose-tolerant in pregnancy, but higher peripheral and hepatic insulin resistance than glucose-tolerant women (53). The β-cell defect is more apparent in the non-obese than obese GDM women in whom insulin resistance is often a greater contributing factor (54). These metabolic defects result in abnormalities of post-prandial lipoprotein metabolism (55) that can further reduce insulin sensitivity and compromise β-cell function (37,56). The diet should be aimed at lessening these metabolic abnormalities. As with the Type 2 diabetic women, most of the women who develop GDM are obese and weight gain targets should be set and a degree of energy restriction considered, see below. However, unlike the Type 2 diabetic women most can achieve adequate glycaemic control with diet alone. For this reason the dietary recommendations for GDM will be considered in further detail below.

## GENERAL DIETARY RECOMMENDATIONS FOR GDM

A dogmatic approach to the dietary advice for GDM should be avoided as only four randomised trials of primary dietary management of GDM against no treatment were considered to be of sufficient standard to include in a recent Cochrane systematic review (57). This pooled data analysis of 612 women failed to show any benefit of dietary intervention on final birthweight, risk of LGA infants and/or Caesarean deliveries (57). However, ignoring all clinical and observational nutritional studies that have no non-intervention arm is probably unwise, and until definitively controlled studies are done each available study should be considered on its own merit.

The objectives in the dietary management of GDM include glycaemic control, balancing adequate nourishment for the mother and foetus, while limiting excessive weight gain, and establishing healthy eating habits that will continue beyond the pregnancy. Lifestyle changes encompassing diet and exercise should be started during the pregnancy itself, when access to a qualified dietitian is likely to be greater than at any future time.

It is important that women with gestational diabetes understand why dietary intervention during the pregnancy is so important to obstetric care. It is worth stressing that adherence to a diet in pregnancy can in most women improve glycaemic control. Understanding that a diet will reduce her risk of having a very large baby and the need for insulin therapy in pregnancy will help compliance. The importance of avoiding unnecessary weight gain needs to be emphasised, and women need to know that too much weight gain increases the risk of delivering an LGA infant and increased obesity post partum (58). Unnecessary weight gain will also increase the future risk of developing GDM in a subsequent pregnancy (59), and diabetes in later life (60).

### *CALCULATING TOTAL ENERGY FOR THE DIET AND SETTING SAFE WEIGHT GAIN TARGETS*

In our practice we calculate an individual's energy requirement using the pre-pregnancy weight to calculate resting energy expenditure, using Schofield's formula (61), and a physical activity ratio of 1.6. To this we add 200 kcal for the energy requirements for the third trimester. If we wish to induce a mild degree of negative energy balance we subtract 500 kcal from this calculated daily energy requirement to provide the total energy for the diet.

The American Diabetic Association (ADA) have endorsed dietary guidelines for diabetes in pregnancy (62) that are based on pre-pregnancy weights, see Table 7.3.

As previously mentioned, current American guidelines recommend a minimum weight gain of 7.0 kg for all obese (BMI > 29 kg/m$^2$) women, both diabetic (63) and non-diabetic (24). No equivalent weight or daily calorie guidelines exist for the UK. Our own unit limits weight gains in diabetic pregnancies to the bottom rather than the top of those recommended for average, overweight and obese women. For Type 2 diabetic women and those with GDM if the BMI is > 34 kg/m$^2$ we set no minimum weight gain. Ideally we like to achieve no overall weight gain in the overweight woman post partum and weight loss in the morbidly obese woman.

**Table 7.3**

| Pre-pregnancy weight (% ideal body weight) | Daily calorie intake (kcal/kg) |
|---|---|
| < 90% | 36–40 |
| 90–120% | 30 |
| 121–150% | 24 |
| > 150% | 12–18 |

## CALORIE RESTRICTION IN THE OBESE WOMAN WITH GDM

The safety of calorie restriction in pregnancy is not known and genuine concerns exist around infant psychological or physical development. Long-term follow-up of children born to mothers exposed to famine suggests that future health is compromised. Infants born to previously well-nourished Dutch women restricted to 800 kcal/day in late pregnancy during the five months of famine in 1944/5 developed normally, although thinner at birth and at 18 years (64). However, when middle-aged these children had a higher incidence of glucose intolerance and diabetes (65).

Maternal ketosis, induced by calorie restriction, has been implicated to impaired foetal neuro-physiological and cognitive development (66,67). While there is a general reluctance to recommend severe calorie restriction in pregnancy even in obese women, modest calorie constraint for those with GDM may be safe as these women are relatively protected against ketosis by their high hepatic glucose outputs (54,68,69). Theoretically maternal ketosis can be lessened during modest calorie restriction when small frequent meals containing slowly absorbed carbohydrates are taken, as such diets are associated with an attenuated insulin response that delays lipolysis and ketogenesis (70).

We have previously reported that when the daily energy is restricted to 20–25 kcal/kg/day for obese women with GDM (pre-pregnancy BMI $> 28\,kg/m^2$) from the 24th week of gestation, weight gain is half that of women with a similar pre-pregnancy weight who receive no dietary intervention, and their risk of delivering an LGA infant is similarly reduced (71). This degree of modest calorie restraint has also been shown to improve glycaemic control (69). Frequent small meals containing slowly absorbed carbohydrates help to prevent ketosis.

All women receiving a diet that is calorie restricted should have regular foetal ultrasound examinations to ensure that foetal growth is not compromised.

## THE OPTIMAL MIX OF DIETARY CARBOHYDRATE AND FAT FOR GDM

The diet for the diabetic mother needs to limit excess maternal–foetal transfer of glucose. As post-prandial hyperglycaemia is the time of maximal maternal–foetal glucose transfer, treatment interventions need to target this period (6). Controversy exists on how best to achieve this. Some authorities recommend limiting carbohydrate at the expense of increasing dietary fat, while others

favour high-carbohydrate diets with a low glycaemic response. It is the authors' belief that promoting diets that actively limit carbohydrate over fat sends out the wrong lifetime educational message. Clinical studies suggest that it is the type of carbohydrate and fat rather than the absolute amount that dictates the glycaemic and metabolic responses to a meal. As a degree of gastric stasis is common in pregnancy, the glycaemic response of many carbohydrates is blunted.

The American Diabetic Association (62) recommend limiting carbohydrate to 40% of the total energy content by increasing dietary fat to 40%. This advice is based on clinical studies showing women with GDM have better glycaemic control when consuming less than 45%, rather than more than 45%, of their calorie intake as carbohydrate (72,73). The American approach gives no acknowledgement to the fact that different ingested carbohydrates have different glycaemic responses as measured by their glycaemic index (74).

British advice on the diabetic diet in pregnancy does not recommend limiting carbohydrate to 40% of the total energy and indeed suggests this figure should be nearer 55%, with the majority of carbohydrate having a low glycaemic index (75). Low glycaemic index diets can in fact increase insulin sensitivity in both pregnant and non-pregnant individuals (42–44,76). In pregnancy glycaemic control deteriorates when refined carbohydrate contributes more than 45% of the total energy (72). By contrast when refined carbohydrates are exchanged for low glycaemic index carbohydrates, 60% of the total dietary energy can be consumed in this form without any change in glucose tolerance (42–44). As the glycaemic response to rapidly absorbed refined sugars is greatest in the early morning, advice on suitable commercial breakfast cereals should be given (77).

## DIETARY FAT

The short-term dietary studies that demonstrated a benefit of high-fat versus high-carbohydrate diets on post-prandial blood glucose values (72,77) may, as discussed above, have been accounted for by the use of high glycaemic index carbohydrates in these studies. Jovanovic's group (78) have also stated that the addition of dietary saturated fat to a test meal produces a significantly lower glycaemic and insulin response than when the test meal contains the equivalent proportion of monounsaturated fat. The differences may be explained by slower gastric emptying when the meal contains a high saturated fat content. However, we believe that even if the glycaemic response mid-morning can be lowered by increasing the saturated fat content of the breakfast, advocating such a diet to women at future risk of diabetes and cardiovascular disease remains highly questionable, when epidemiological and clinical studies show that high-fat diets are associated with insulin resistance, β-cell dysfunction, and

recurrent GDM pregnancy and future diabetes (56,59). Also the long-term effects of a high maternal saturated fat diet on cardiovascular health is unknown. Animal studies certainly suggest caution as high-fat diets in pregnant rodents can promote cardiovascular disease in the next generation (79,80). Increasing the saturation content of the diet in pregnant rats leads both to unfavourable changes in fatty acid compositions and function of the major arterial vessels. High-fat diets in pregnancy have also been associated with severe hyperemesis gravidarum, with a 5.4-fold increased risk reported for every additional 15 g/day of dietary saturated fat (81).

Increasing the polyunsaturated fat (PUFA) content of the diet while restricting the saturated fat may provide an alternative approach to safely reducing the overall dietary carbohydrate content. A large epidemiological study in China reported that a high habitual intake of dietary PUFA with a correspondingly raised low dietary polyunsaturated to saturated fat ratio protected against gestational diabetes (57). It remains to be proven whether Western women would achieve a similar benefit, as their PUFA intake is highly correlated with saturated fat intake.

The potential benefits of increasing monounsaturated fat (MUFA) intake in pregnancy still need to be shown. A recent small Danish study failed to show any improvement in insulin sensitivity in late pregnancy when women with GDM eat diets high in MUFA rather than high in carbohydrates, although a favourable effect on blood pressure was reported (82). Outside pregnancy improved insulin resistance and lipid profiles have been reported when either a high-carbohydrate diet or a monosaturated-enriched diet replaces dietary saturated fat, with reductions in plasma LDL cholesterol observed (83,84). If high-MUFA diets are to be promoted over a high-carbohydrate diet, one needs to ensure that overall calorie intake leading to unnecessary weight gain does not occur (85).

A large Swedish epidemiological study has suggested that increasing dietary long-chain *n*-3 fatty acids (omega-3 fatty acids) by increasing fish and fish oils may provide some protection against low birth weights and pre-term deliveries (86). Similar diets in Type 2 diabetic subjects have been shown to have some favourable metabolic effects on serum triglycerides but not plasma LDL cholesterol (87,88). Other food sources of *n*-3 polyunsaturated fatty acids include flaxseed and flaxseed oil, canola oil, soybean oil and nuts. Population studies suggest that foods containing *n*-3 fatty acids, specifically eicosapentaenoic acid and docosahexaenoic acid, may provide long-term cardio-protection (89,90). There are therefore potential theoretical benefits for increasing dietary long-chain *n*-3 fatty acids in diabetic women both in and out of pregnancy.

In the face of no real clinical-based studies on the optimal ratio between saturated, poly, mono and fish oils for pregnancy, it is our policy to aim for a ratio of sat:poly:mono of 1:1:1, with the specific advice to eat oily fish three

times a week (91). These recommendations are similar to those for people with diabetes and coronary heart disease.

## DIET AND INSULIN THERAPY FOR GDM

Once diet alone can no longer consistently ensure fasting glucose values below 5.5 mmol/l and a 1 h post-prandial value below 7 mmol/l, the introduction of insulin should be considered (63). It is important to recognise that a small proportion of women will require insulin early in pregnancy and not to assume dietary non-compliance (92). Those requiring insulin are the most metabolically compromised and tend to have both the highest perinatal complications and the fastest deterioration to diabetes after pregnancy (93). Insulin is also occasionally introduced in later pregnancy for obstetric rather than glycaemic reasons; this might occur for accelerated foetal growth or unexplained polyhydramnios (94).

It is important to stress that once insulin is introduced for the management of GDM the dietary management remains equally important. The need to limit weight gain remains for obese women who now need to balance this with having sufficient carbohydrate snacks throughout the day to prevent hypoglycaemia. Although short periods of hypoglycaemia are not detrimental to the foetus they are unpleasant for the woman and frequently result in sudden rises of blood sugar due to the action of counter-regulatory hormones and the consumption of sugary drinks. Frequent episodes of hypoglycaemia often result in women chasing these high-rebound glucose levels by increasing their insulin dosage, which can result in further hypoglycaemic attacks and unnecessary weight gain.

When starting on insulin women should be advised to take low glycaemic index carbohydrates at meal times and for snacks between meals and before bed. Fruit is ideal for snacks as it is low in fat and calories. Fruit, by being slowly absorbed, reduces the risk of hypoglycaemia while allowing post-prandial glucose levels to be lowered without having to increase the insulin dose.

## LONG-TERM DIETARY ADVICE FOR THE MOTHER AND HER CHILD

As most women with GDM are obese and all have at least one child at increased risk of adolescent obesity and diabetes, providing dietary education and advice that extends beyond the pregnancy is extremely important. Lifestyle changes encompassing diet and exercise have been shown to reduce the risk of GDM in subsequent pregnancies as well as delaying the progression to Type 2

diabetes (59,95,96). Women with a history of GDM are an ideal group to target, not only because of their own heightened risk of future diabetes (97,98) but to ensure a healthy lifestyle within the family unit, hence reducing the risk of obesity and future diabetes in the children also.

Ideally all women with GDM should receive lifestyle advice and education in pregnancy that is relevant to after pregnancy. It will be an important challenge to find methods of delivering dietetic education and advice both effectively and cheaply to enable all women with GDM to receive the necessary ongoing support and care they require after pregnancy in the community.

## THE NEED AND FEASIBILITY OF FUTURE DIETARY STUDIES IN PREGNANCY

There remains a lack of good randomised studies on the dietary management of diabetic pregnancies. Such studies are required for both short-term pregnancy outcomes and long-term outcomes for the mother and her child. One of the main difficulties in conducting such studies is the control arm; even when no dietary advice is given, women once diagnosed with GDM make lifestyle changes based on family beliefs or information gathered from a variety of sources. Also if the health care providers are aware of the diagnosis they too unintentionally are likely to influence lifestyle factors. The need to blind both the women and the health care staff to the diagnosis is difficult and often considered unethical, as GDM if ignored can carry a risk to the pregnancy (99). It is hoped that the HAPO Study (Hyperglycaemia Adverse Pregnancy Outcome Study) currently underway, looking at pregnancy outcomes in 25 000 pregnant women in whom lesser degrees of glucose intolerance will go untreated, will help to answer some of these questions.

## SUMMARY

Diabetes is a common complication of pregnancy. Nutritional advice, intervention and education are a central part of the management of all women with diabetes in pregnancy. Dietary intervention, either alone or with insulin, can improve pregnancy outcomes. Appropriate advice to obese women with Type 2 diabetes and GDM should aim to avoid excessive maternal weight gain and worsening glucose tolerance after pregnancy. For women with GDM dietary advice in pregnancy should extend beyond the pregnancy itself, aimed at reducing the lifetime risk of future diabetes for both the mother and her child.

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