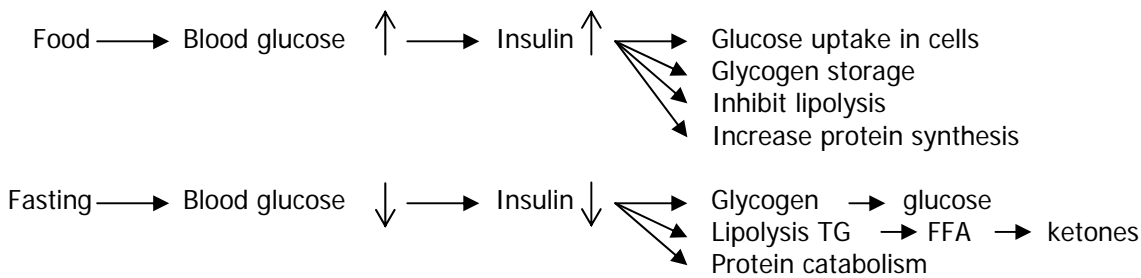# Background Paper to Support 'Diabetes in Pregnancy' (ADHB Guideline)

1. Glucose metabolism

Glucose is maintained within a normal range primarily through the hormone insulin. Following a meal, a subsequent blood glucose rise stimulates increased insulin production from the pancreas. Insulin promotes cellular uptake and utilisation of glucose with excess requirements being stored as glycogen and fat. Insulin also increases protein synthesis and inhibits lipolysis (breakdown of fat).

In the fasting state, as blood glucose levels fall, insulin secretion decreases. This leads to conversion of glycogen to glucose to maintain euglycemia. As fasting continues, triglycerides (TG) are used for further energy production, releasing free fatty acids (FFA) and ketone bodies. Protein stores are also used for energy production with prolonged fasting.

Food ⟶ Blood glucose ↑ ⟶ Insulin ↑
- Glucose uptake in cells
- Glycogen storage
- Inhibit lipolysis
- Increase protein synthesis

Fasting ⟶ Blood glucose ↓ ⟶ Insulin ↓
- Glycogen ⟶ glucose
- Lipolysis TG ⟶ FFA ⟶ ketones
- Protein catabolism

This homeostatic mechanism is stressed when a person has increased visceral fat stores, as these stores (unlike peripheral subcutaneous fat) are metabolically active and readily secrete inflammatory cytokines and excess free fatty acids, which are associated with development of insulin resistance. This means that more insulin has to be secreted to maintain normal glucose levels.

2. Types of diabetes

Type 1 diabetes

When insulin is deficient due to (autoimmune) destruction of pancreatic β-cells this is termed type 1 diabetes. In this disorder exogenous insulin is required to prevent ketoacidosis. This is typically seen in leaner younger people but can be diagnosed throughout life. Most people diagnosed with type 1 diabetes have positive antibodies (e.g. GAD and IA2).

Type 2 diabetes

When insulin is relatively deficient due to inadequate compensation for underlying insulin resistance, this is termed type 2 diabetes. These people usually have visceral adiposity and elevated insulin levels, but the action of insulin is impaired by many factors including interactions with adipocytokines. The main aim of treatment is to reduce insulin resistance with lifestyle interventions. Tablets that decrease insulin resistance (metformin, thiazolidinediones) or stimulate increased insulin secretion (sulphonylureas) are typically used outside pregnancy. Insulin is used if adequate glucose control is not achieved by these measures.

Inherited "monogenic" diabetes

When insulin is relatively deficient as a result of a single gene mutation in pathways involved with the secretion/action of insulin this may present as "MODY" (maturity onset diabetes of the young). This should be considered in a slim young person who otherwise appears to have type 2 diabetes and has a family history reflecting an autosomal dominant pattern of inheritance. Currently six different subtypes of MODY are recognised. Some are occasionally associated with renal or genital tract anomalies (MODY 3 [HNF-1α mutation] and MODY 5). Most of these are associated with progressively worsening insulin secretion over time. An exception is MODY 2 (glucokinase mutation), which is associated with mild hyperglycaemia and a lower risk of complications, as the threshold for recognising elevated glucose levels is set higher rather than any progressive worsening of glucose handling. Another important inherited diabetes is associated with a heteroplasmic mitochondrial gene mutation (usually 3243 A-G), and is therefore inherited through the maternal genes (as mitochondria are maternally transmitted). This maternally inherited diabetes and deafness (MIDD) should be considered in any woman with diabetes who is deaf. It can be associated with other mitochondrial problems termed MELAS (mitochondrial encephalopathy, lactic acidosis, strokes). Overall, monogenic diabetes is likely in only 1 - 2% of women seen in diabetes in pregnancy clinic and tests for diagnosis are expensive. Appropriate testing can be discussed with a diabetes specialist. They are worth considering, as the implications for diagnosis are relevant to the choice of medication, particularly outside pregnancy (MODY 1, 2, 3 may be sensitive to sulphonylurea treatment). Also, with MODY 1 (HNF-4 α mutation) the fetus may be macrosomic if it carries the mutation, independent of maternal glucose control. In utero, this mutation causes fetal hyperinsulinaemia and it is as the child grows up that reduced secretion of insulin develops. With MODY 2 the mother requires tight control if the fetus does not have the mutation, but if the fetus also has glukokinase mutation tight control in the mother may be associated with decreased fetal growth. As the genotype of the fetus is not known

during pregnancy, following the growth may be particularly helpful in this MODY 2 to decide how aggressively to treat the mother.

Impaired glucose tolerance/prediabetes

Prior to the development of diabetes, glucose levels may be elevated above the "normal" range, but not high enough to make a diagnosis of diabetes. Outside pregnancy, if, on a 75g oral glucose tolerance test (OGTT) the 2 hour glucose level is 7.8 - 11.0mmol/l a diagnosis of impaired glucose tolerance is made (IGT). If a fasting glucose is 6.1 - 6.9mmol/l (or 5.6 - 6.9mmol/l in the USA), a diagnosis of impaired fasting glucose is made (IFG). Mostly, these are people who will develop type 2 diabetes unless they institute lifestyle interventions (or sometimes medication such as metformin) to reduce their risk of progression. As glucose homeostasis is not normal outside pregnancy, these women will require treatment during pregnancy. Prediabetes can be diagnosed if an HbA1c outside pregnancy in NZ is 41 - 49mmol/mol.

Gestational diabetes

Glucose homeostasis alters in pregnancy due to the fetus siphoning maternal glucose and the effects of pregnancy hormones. Pregnancy is associated with a maternal inflammatory response, which is associated with insulin resistance. Human placental lactogen, placental growth hormone plus increase estrogen, progesterone and cortisol create insulin resistance. Also, alteration in free fatty acid levels and other cytokines secreted by adipocytes, inflammatory cells and the placenta contribute to insulin resistance (e.g. TNF-α, leptin).

Normal glucose levels during pregnancy in lean women are:

- Fasting glucose levels with continuous tissue testing: mean 4.2±0.67 mmol/L
- Peak postprandial (approx. 60 - 90 mins. after start of meal): mean 6.1±0.89 mmol/l)
- Mean glucose levels: 4.7 ± 1.0 mmol/l
- Insulin levels are increased (may be 2 - 3 times that outside pregnancy) to maintain normal glucose levels
- Obese women have higher postprandial glucose peaks than lean women. It is not known whether increased pregnancy risks associated with obesity relate to any alteration in glucose metabolism

Some women are unable to maintain normoglycaemia as a result of changes in glucose homeostasis in pregnancy and they develop elevated glucoses i.e. diabetes in pregnancy.

It can be seen that the pathogenesis of GDM is similar to type 2 diabetes and many of these women are at increased risk of developing type 2 subsequently. At National Women's Health, almost 30% of women with GDM are reclassified as having IFG, IGT or type 2 diabetes when they do a 75g oral glucose tolerance test (OGTT) at 6 - 8 weeks postpartum. This risk increases with follow up and it is estimated that after 20 years up to 80% of women in this population develop diabetes.

3. Why we look for GDM – a history lesson

Women who have diabetes prior to pregnancy have increased risk for pregnancy complications. Similar complications were seen in women who developed diabetes later in life and it was realised that these women probably had transient diabetes during pregnancy or gestational diabetes.

When initially trying to identify this group with GDM, women were given a glucose tolerance test in pregnancy, and the criteria for diagnosis of GDM were based on the glucose levels that predicted subsequent diabetes. The level of glucose intolerance that corresponded to increased pregnancy risk was not clearly identified. This has led to much controversy as to what should be the criteria for the diagnosis of GDM and different centres use different criteria. Mild abnormalities of glucose metabolism may lead to unnecessary intervention in a pregnancy that is progressing normally. Alternatively, we may miss significant morbidity if the cut off used to define an abnormal glucose tolerance test is too high.

In recent years there have been two randomised prospective trials published, the Australian carbohydrate intolerance study (ACHOIS) and the American maternal fetal medicine network trial that show reductions in adverse perinatal outcomes in women with mild GDM who are treated compared with women who have not been treated (Crowther et al NEJM 2005;352:2477-86, Landon et al NEJM 2009;361:1339-48).

There has been a large non-interventional epidemiological study of over 23,000 women looking at the results of a 75g OGTT at 24 - 32 weeks' gestation and how the glucose levels relate to several pregnancy complications (HAPO NEJM 2008; 358:1991-2002). Risks increased as the maternal glucose levels increased, with no clear cut-off. Subsequently, an international committee has made recommendations about the diagnosis of GDM (screening for GDM).

In New Zealand it is recognised that there are a number of women who are at high risk of having unrecognised diabetes or impaired glucose tolerance when they conceive. There are recommendations about testing to detect these women when they book for pregnancy care. If those results are normal and in all other pregnant women, there are recommendations about testing for gestational diabetes between 24 - 28 weeks gestation. These recommendations are covered in screening for GDM.

4. Pre-pregnancy assessment for women with pre-existing diabetes

Diabetes control prior to pregnancy

It is important to aim for good diabetes control prior to becoming pregnant. One of the most important reasons for doing this is to minimise the risks of:

Congenital anomalies

Women with diabetes have an increased risk of congenital anomalies, which account for 30-50% of the perinatal losses in women with pre-existing diabetes. The background rate of anomalies in the general population is 2 - 3%. A recent review demonstrated that the congenital anomaly rate increases in a linear manner by 2% for every 11mmol/mol increase in HbA1c above the reference range. So, for an HbA1c of 64mmol/mol, the anomaly rate is approximately 8%. In women with type 2 diabetes the recommended aim for a preconception HbA1c is as close to the normal reference range as possible. This is more difficult to achieve in women with type 1 diabetes, and women are often given the go-ahead to conceive once the HbA1c is down to 50 - 55mmol/mol.

It is not known whether periconceptual folate decreases the risk of congenital anomaly associated with hyperglycaemia in women with diabetes but this should be prescribed as for all pregnant women. In Auckland, 0.8mg daily is recommended. There are no data currently to suggest additional vitamin supplements are of benefit.

Spontaneous abortions

There are increased risks of miscarriage in women with poorly controlled diabetes.

Type 1

In type 1, improved control may require further education with diet advice, increased monitoring and usually an intensive insulin regimen. Short acting insulin analogues before meals are recommended. For basal insulin requirements twice daily ispohane insulin is still used a lot in New Zealand, but the long acting analogue glargine is also subsidised for use in type 1 diabetes where significant hypoglycaemia is a problem. There have been theoretical concerns about use of glargine (lantus) and risk of cancer, as glargine has one of the greatest affinities for the IGF binding sites, which are more active in e.g. breast tissue during pregnancy, but there has been no evidence to support this concern and glargine has been used widely in many countries for years. Detemir (levemir) insulin is another option for a long acting insulin analogue in pregnancy and has less IGF affinity but it is not subsidised for use in New Zealand.

Type 2

In type 2**,** many women are managed with lifestyle measures or oral hypoglycaemic agents. Most are obese. They need further diet advice combined with exercise and a weight loss programme. Many centres still stop oral agents prior to pregnancy and change to insulin. However, management needs to be tailored to the individual. In Auckland, many women with type 2 diabetes are managed with night time intermediate

acting insulin and daytime metformin, as this typically improves control without significant weight gain. There is no evidence of teratogenesis with currently used oral agents. If a woman conceives on an oral agent she should continue it until she is reviewed by a diabetes specialist, rather than stop it immediately, as that would expose the fetus to worsening hyperglycaemia.

Sulphonylureas

Many sulphonylureas cross the placenta, though second generation ones cross less and glibenclamide crosses least (about 4%). Sulphonylureas may directly stimulate fetal insulin production, and if taken through pregnancy, we do not know if there are long-term effects. Prolonged neonatal hypoglycaemia is described in offspring of women who take sulphonylureas up to delivery, but this has not been reported as a significant problem with glibenclamide.

Metformin

Metformin crosses the placenta. There are increasing reports describing safe use of metformin through pregnancy. Metformin is also used to improve fertility in women with polycystic ovary syndrome (by improving insulin resistance) and it is continued through pregnancy in some centres. It may decrease the risk of miscarriage and potentially risk of GDM developing in these women. In light of these data, some women with type 2 diabetes choose to remain on metformin during pregnancy.

Thiazolidinediones

Thiazolidinediones have not been studied in human pregnancy, but have adverse effects in animals and currently should not be used.

Acarbose

Acarbose has been used in pregnancy, and is probably safe, but has not real role during pregnancy as it is typically not an adequate treatment to maintain good glucose control.

Diabetes complications and risk assessment prior to pregnancy

It is important to assess complications as they may require intervention prior to pregnancy. Some complications may significantly increase pregnancy risks and the woman needs to be aware of this and make an informed choice about pregnancy.

Relevant issues are:

Retinopathy

- Retinopathy should be assessed by an ophthalmologist or experienced physician prior to pregnancy
- Retinopathy, particularly maculopathy, may progress significantly if diabetes control is suddenly improved. Proliferative retinopathy and maculopathy may require treatment (e.g. laser) prior to improving diabetes control

- Proliferative retinopathy or maculopathy, if treated and stable pre-pregnancy, is usually minimally affected by pregnancy
- More frequent eye review is often necessary in pregnancy. Laser therapy should be administered in pregnancy if indicated

Nephropathy

Nephropathy is characterised by proteinuria (> 0.3mg/day), impaired renal function and hypertension. In the early stages only microalbuminuria may be present. Pregnant women with nephropathy are at increased risk of:

- Hypertensive complications (gestational hypertension and preeclampsia). The risk may be as high as 75% in some women with advanced nephropathy. Proteinuria typically increases in pregnancy and usually improves to pre-pregnancy levels postpartum. Preeclampsia can be hard to distinguish from worsening proteinuria and gestational hypertension
- Fetal growth restriction (15 - 20%)
- Prematurity (30% < 34 weeks)
- Increased perinatal mortality (6 - 7%)

The risks of complications relate especially to the degree of renal impairment at the onset of pregnancy, though the degree of proteinuria and hypertension are important additional factors. Once a woman has a creatinine in the order of 200 mg/dl or higher, her risks of delivering a very premature baby become very high, and careful counselling is required so that the woman understands the implications of potentially having an unsuccessful pregnancy and the risks for the baby associated with very premature birth. Some women defer pregnancy until they have had a successful renal transplant.

Women with nephropathy are often on an ACE-inhibitor before pregnancy to slow the progression of their renal condition and treat hypertension, but ACE inhibitors are generally contraindicated during pregnancy. Retrospective observational data report increased rates of congenital anomalies (especially renal, CNS, cardiac) in women taking an ace-inhibitor during organogenesis (Cooper et al NEJM; 354:2443-51). In the second trimester, ACE-inhibitors are associated with lack of occipital bone formation in the fetus and in later pregnancy, have adverse effects on fetal kidneys that may be irreversible. Clinicians may decide to stop ace-inhibitors pre-pregnancy, particularly if prescribed for microalbuminuria alone. However, if it is an important reno-protective medication for a woman who is reliable about recognising early pregnancy, the data should be discussed and a woman may choose to continue her ace-inhibitor with a plan to stop by 5 weeks' gestation, particularly as it may take months (or years) to conceive. If hypertension is present, an alternative antihypertensive medication should be prescribed. Improved pregnancy outcomes have been reported in this population if aggressive blood pressure control is maintained though pregnancy (Nielsen et al Diab Care 2009; 32:38-44).

Pregnancy does not generally accelerate deterioration of renal function in women with nephropathy, but it may do in some women, especially if their renal function is abnormal pre-conception.

Most women do not have severe nephropathy and the rates of preeclampsia in women with type 1 and type 2 diabetes in Auckland overall is about 15%. Early onset preeclampsia is very uncommon in the diabetes clinic in Auckland.

Neuropathy

Women with autonomic neuropathy have increased risks of adverse pregnancy outcomes. Gastroparesis may cause difficult management problems with vomiting and must be considered in women who present with hyperemesis.

Ischemic heart disease (IHD)

It is important to remember to consider IHD in women with diabetes, particularly if the woman has other risk factors. Symptoms or other features suggestive of IHD should be investigated prior to pregnancy. Women with diabetes who have a myocardial infarct in pregnancy have a high mortality rate, which has been reported to be as high as 50% in the past. This will be less now with present methods of early intervention and management.

Other

Autoimmune antibody screens in type 1 diabetes

Women with type 1diabetes should be screened for thyroid antibodies and thyroid dysfunction, usually every couple of years if previously normal. Also, they should have a screen for celiac disease every 2 - 5 years or earlier if symptoms. Screening for other autoimmune endocrine diseases should be considered on an individual basis (e.g. pernicious anaemia, etc).

Vitamin D deficiency

Many women in Auckland with type 2 diabetes (and GDM) have risk factors for vitamin D deficiency (darker skin colour or very pale, covered, little sunshine exposure). Replacement is considered safe during pregnancy. A prescription for calciferol tablets, 1.25mg, to take as directed can be written and 12 tablets prescribed. If the levels are likely to be very low, all 12 tablets can be given, especially in late pregnancy, to ensure adequate amounts are available to the fetus and for breastfeeding. A more common recommendation is to give six tablets then continue either one tablet monthly or six tablets every six months. Importantly, ongoing replacement is usually recommended, Obviously, it is important to relay this advice to the GP. A template for a letter to the GP is in the resource section.

Vegetarian or vegan

Vegetarian or vegan women and many Indian women have low vitamin B12 levels, so this should also be assessed in at risk women and oral replacement recommended if low. B12 deficiency may be exacerbated by treatment with metformin, which reduces B12 absorption, so checking B12 levels should also be considered in any woman taking metformin (unless used for a short time for example in a woman with GDM). Oral

B12 supplements with B12 "dots", Thompson's Pregnacare (6 microgm), or Elevit (4 microgm) may provide adequate replacement.

5. Future screening recommendations – under discussion

The hyperglycemia and adverse pregnancy outcomes (HAPO) study was an observational study of over 23,000 pregnant women that was published in 2008 (HAPO NEJM 2008; 358:1991-2002). In this study a 75g OGTT was performed between 24 - 32 weeks on a routine low risk pregnancy population and results were not disclosed unless levels were above a pre specified level.

In all the women whose results were not known, data were collected to see the relationship between maternal glucose levels measured by the OGTT at 28 weeks and pregnancy outcomes.

They demonstrated that rates of primary caesarean section, large for gestational age, clinical neonatal hypoglycaemia and cord C-peptide levels were related in a continuous manner to maternal glucose levels. Other important secondary outcomes such as neonatal fat levels and maternal preeclampsia were also related.

Since then, an international committee has further analysed the data to determine cut-off levels for international criteria for the diagnosis of GDM (IADPSG panel Diab Care 2010; 33 (3):676-82).

The committee recommended that all pregnant women should be offered a 75g OGTT during pregnancy between 24 - 28 weeks gestation, except for women at high risk of unrecognised glucose intolerance prior to pregnancy, who should be offered earlier testing, as outlined above.

This means that the 50g glucose screening test would not be required in the future.

The cut-off levels proposed to diagnose GDM following a 75g OGGT are any one of the following:

- Fasting glucose ≥ 5.1mmol/l
- 1 hour glucose ≥ 10.0mmol/l
- 2 hour glucose ≥ 8.5mmol/l

It is also proposed that only a fasting and one hour test can be used, saving the need for women to remain at the laboratory for an additional hour. Only 2% of women with GDM will be missed by omitting the 2 hour test.

In the study population, using these results, 15 - 20% of pregnant women would have a diagnosis of GDM. This has implications for models of care. It is important for this to be discussed and appropriate systems set up before any changes are agreed upon. Otherwise, this could dilute the care of women at highest risk, which are women we see currently.

A possible scenario would be as follows:

If these criteria are adopted, the result may be used as a risk marker, which leads to additional management recommendations, including:

Direct referral to clinic for some women (criteria may be similar to current referrals until data are collected on women with less elevated glucose results).

For all other women diagnosed with GDM they still require:

- Education about diet and lifestyle
- Teaching home blood glucose monitoring and review of accurate results (download or check meter a week later)
- Fetal ultrasound scan for proportionate growth measures
- Documentation of additional risk factors such as BMI, hypertension, maternal age, obstetric history etc
- Referral to clinic will depend on results of these additional assessments

In women who are managed outside the diabetes clinic, they will require ongoing assessments and referral as required.

There will be recommendations about timing of delivery, depending again on risk factors.
In NZ, it is proposed that a trial should be done to decide whether we adopt the international criteria or not.

6. Neonatal complications

Morbidity is related to the ongoing effects of the altered intrauterine environment.

- Neonatal hypoglycaemia: Hypoglycaemia in a neonate is defined as capillary glucose < 2.6 mmol/l. Neonatal hypoglycaemia is a consequence of suboptimal maternal glycaemic control and secondary fetal hyperinsulinemia that takes a while to adapt to external glucose delivery postpartum. Poor glycemic control during labour can also cause this. This may be worse if the baby is premature or growth restricted. The level of hypoglycaemia can be reduced from around 38% to very low levels in centres with strict control. At National Women's Health, babies are monitored within an hour of birth and pre-feed. When glucose is < 2.6 mmol/L a small supplemental feed is given (usually some time on the breast if not too low, a cup feed, *or bottle* up to 25 ml), giving breastmilk if possible. Intravenous dextrose is considered with recurrent levels < 2.3 mmol/l, but depends on response to a supplemental feed and other factors too
- Respiratory distress syndrome (RDS): babies of women with diabetes are at increased risk of RDS, even at late gestations. This may be due to insulin restricting surfactant synthesis and interfering with usual glucocorticoid mediated lung maturation
- Polycythaemia: this may be secondary to fetal hypoxia and can lead to hyperbilirubinaemia and need for phototherapy
- Other effects are seen less frequently but include hypomagnesaemia and hypocalcaemia, which may relate to delay in maturation of homeostatic mechanisms, again possibly related to hyperinsulinaemia
- Birth trauma/shoulder dystocia
- There are data showing that offspring of women with diabetes have increased rates of obesity and type 2 diabetes, partly due to the exposure to hyperglycaemia in utero. Subsequent, fetal hyperinsulinemia results in increased adipose tissue cellularity. The fetus may also develop insulin and leptin resistance in utero. This means these children should have follow up and early lifestyle interventions to improve these risks. Breastfeeding is a very important component in this and should be encouraged whenever possible. Handover to the Well Child Provider regarding childhood follow-up is recommended neonatal follow-up is not routine in this regard

7. Information for obstetric registrars working in National Women's Health diabetes clinics

Introduction to diabetes antenatal clinic

In diabetes clinics the patients are more complex and each patient may be seen by up to 8 different disciplines – obstetricians, physicians, midwives, dieticians, physiotherapists, social workers, Pacific Islanders' support and Smoke Change. Each discipline brings essential expertise for management of pregnancy in the diabetic patient.

The diabetes midwives have a lot of experience, always listen to their advice.

Role of the clinic

a) Pre-pregnancy counselling for those with pre-existing diabetes;

b) Nutritional education and counselling, which includes support and encouragement regarding lifestyle changes;

c) Usual ante-natal care including risk assessment and management;

d) Individualised delivery plan;

e) Contraceptive services;

f) Post partum follow up.

Types of diabetes

a) Type 1 diabetes. These patients have usually had diabetes since their youth and will be dependant on insulin. Short acting insulin is given prior to meals and long acting during the day and at night. These women are more likely to have retinopathy,, nephropathy and neuropathy;

b) Type 2 diabetes. These patients vary considerably, from those with recent disease and early diagnosis to those with long standing poorly treated disease. They may have complications of diabetes; hypertension, raised lipids, impaired renal function, ischaemic heart disease and other vascular complications;

c) Gestational diabetes (GDM) – diabetes only diagnosed during pregnancy. These patients vary from those with small elevations in blood sugar only occurring at the end of pregnancy to undiagnosed type 2 diabetes with vascular disease. These patients range from our lowest to our highest risk patients. Whether true GDM or undiagnosed type 2 these patients vary from being controlled on diet alone to those needing massive doses of insulin;

d) MODY. Be aware of fetal effects as noted in description of MODY above. Usually treated with insulin, but may be on no treatment or on a sulphonylurea, depending on type of MODY.

Booking visit

a) Revise estimated risk factors for mother and fetus, these include;

- type of diabetes
- maternal BMI
- maternal age
- blood sugar control – HbA1c
- complications of diabetes (hypertension, eye disease, IHD, vasculopathies, renal impairment)
- poor social circumstances
- previous obstetric history
- smoking

b) Check booking bloods, swab results and smears;

- MSU
- HbA1C - > 80mmol/mol may be associated with 10 - 20% risk of fetal abnormality and the patient needs appropriate counselling

c) Dating USS;

d) Discuss screening e.g. Nuchal/MSS and CVS/Amnio if appropriate;

e) Consider aspirin prophylaxis if hypertensive/obese/previous IUGR.

Subsequent visits

a) The only "routine" scans are:

- Booking
- Nuchal
- Anatomy
- 28 weeks for growth and liquor and again at 36 - 37 weeks

We do not request "routine serial growth scans" on anyone. If a patient is obese and we are unable to clinically assess fetal growth, then this is an indication for regular growth scans. As are women with clinically poor growth. *This is very important; we struggle to get ultra sound appointments so we don't want them filled with unnecessary scans.*

b) Revise risks factors at each visit. Read the whole USS report, look at liquor volume, distribution of fetal body weight (never rely on the customised growth chart alone, beware the "little fat baby") and growth;

c) Do an SF measurement at each visit, from 24 weeks onwards. If the women has had a growth scan chart this on her CGC growth chart to correlate it with the scan;

d) Try to decide what you will be doing at the next visit, prior to deciding when next she needs follow up. Perhaps she can see the midwife and see the obstetrician at a later visit;

e) Discuss contraception; we can offer puerperal T/L, Essure, Mirena and implants as well as Depo and the COCP;

f) Remember to share care with the private LMC if she has one, this will save on clinic visits. Likewise remember to communicate with the private LMC.

Inductions and delivery

a) As a basic principle we like to keep induction numbers down and so too caesarean sections. Well controlled GDM with no other risk factors may safely go to term. Needing insulin is not a risk factor, poor sugar control is;

b) Women with a very high BMI or a big abdominal apron need a discussion and plan about delivery. These can be very high risk caesareans;

c) A "big baby" is not an indication for induction of labour. We induce labour to save babies lives; induction of labour does not reduce the woman's risk of having a caesarean;

- Increased risks for IUD are: raised maternal age, raised maternal BMI, big baby with low liquor, very big baby particularly if abdominal circumference is disproportionate to the rest of the baby, SGA, small babies with fat tummies, decreasing insulin requirements as well as all the normal risks

d) Patients with GDM but suspected type 2 diabetes are high risk and usually need delivery prior to 40 weeks;

e) Type 2 and type 1 patients tend to be delivered at about 38 - 39 weeks, earlier for each added complication;

f) Patients who have had a previous LSCS and need an elective delivery need careful assessment and consideration. Induction of labour is not always appropriate and an elective LSCS may be safer.