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Gestational diabetes in New Zealand ethnic groups

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Abstract

In New Zealand the rise in gestational diabetes mellitus (GDM) prevalence reflects the current patterns of increasing obesity and diabetes. Approximately 61,000 women give birth in New Zealand every year, and about 4.9% (6.6%) of those women have diabetes. Screening for gestational diabetes is recommended for all pregnant women in New Zealand, unless an earlier diagnosis of diabetes was made. Indigenous Maori women, Pacific Island, and Asian Indian women are populations genetically prone to diabetes, and have high rates of gestational and type 2 diabetes. Exposure of the foetus to maternal diabetes influences changes in birthweight, adiposity, and foetal insulin production. GDM and type 2 diabetes in Pacific Island and Maori new-borns were associated with higher birthweight, skinfold thicknesses, raised cord insulin, insulin peptides, and increased leptin concentrations. Pacific Island and indigenous Maori women gave birth to macrosomic new-borns, who commonly suffered postnatal hypoglycaemia, respiratory distress, and shoulder dystocia. The association between maternal obesity and SGA raises a concern as SGA is less likely to be detected in early pregnancy in obese women, while the association between SGA and socioeconomic status, initially observed in Maori and Pacific women, was explained by high cigarette smoking rates, and obesity in deprived areas.

Abbreviations: GDM: Gestational diabetes mellitus; CVD: Cardiovascular disease; OGTT: Oral glucose tolerance test; NWH: National Women's Hospital; LGA: Large for gestational age; SGA: Small for gestational age; AGA: Average for gestational age; HbA1c: Glycated haemoglobin

Introduction

It is becoming widely accepted that gestational diabetes mellitus (GDM) is related to some degree of glucose intolerance with onset or first recognition during pregnancy, and is the most common metabolic disorder during pregnancy [1,2]. GDM is becoming more common as the epidemic of obesity and type 2 diabetes continues [3]. The prevalence of diabetes for all age groups worldwide was 2.8% in 2000, and is estimated to reach 57% by 2030. During this 30 years period, the number of women of reproductive age is going to double [4].

In New Zealand the rise in GDM prevalence reflects the current patterns of increasing obesity and diabetes. According to the New Zealand Health Survey [5] the prevalence of obesity rose from 26% in 2006/07 to 29.9%. Approximately 61,000 women give birth in New Zealand every year, and about 4.9% (6.6%) of those women have diabetes. The prevalence of type 1 and type 2 diabetes, and GDM is rising among women of South Asian, Pacific Island, and Maori ethnicities [6]. Internationally, the prevalence of GDM varies between 1 and 22% in relation to differences in ethnic composition, age and obesity status [7]. Screening for GDM is recommended for all pregnant women in New Zealand, unless an earlier diagnosis of diabetes was made. Limited data are available on time trends in GDM in New Zealand [8]. Absolute numbers and prevalence rates have been increasing in recent decades. Even two decades ago, the 1994/95 records in South Auckland Hospital showed high prevalence of GDM in Māori (7.9%) and Pacific women (8.1%) who attended oral glucose challenge tests [9]. Pacific women were more likely to be screened (68.5%) when compared with Māori (47.3%), although both ethnic groups have high rates of GDM and type 2 diabetes. Asian Indian women had GDM prevalence of 5.5% emerged as an ethnic group with increasing risk for diabetes.

The number of women with GDM diagnosed at the National Women's Hospital increased from 1.4% of births in 1991 to >7% in 2010. In 2010, the incidence of GDM in the National Women's Hospital Annual Report [10] was 16% for Indian women, 11.3% for Asian women, 10.3% for Pacific women and 5% for Maori women, compared to 3.7% of New Zealand European women [11]. Screening rates for GDM among Maori women are very low, although they are at high risk of diabetes, and also they were overrepresented amongst the obese groups (40% and 62% respectively). Increase in maternal body mass index (BMI) (kg/m^2) was strongly associated with increasing rates of GDM and type 2 diabetes. However, GDM was diagnosed in 13% of overweight or obese women in New Zealand, and 25% of women with BMI over 40. However, the most frequently reported risk factors for GDM were maternal older age, weight and parity, family history of diabetes, and previous delivery of macrosomic new-borns [12].

The aim of this review is to present the current screening and diagnosis of GDM in New Zealand, and factors which contributed to the development of the adverse foetal outcomes in women with obesity and GDM in diverse ethnic groups in New Zealand.

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Screening and diagnosis of GDM in New Zealand

It is difficult to distinguish whether GDM was undiagnosed before pregnancy or induced by pregnancy in women of childbearing age [13]. Newly developed guidelines for screening and diagnosis of gestational diabetes in New Zealand may reveal increased prevalence of this condition. On the initiative of the Ministry of Health, the New Zealand Branch of the Australasian Cochrane Centre and the University of Auckland developed guidelines, and established a multidisciplinary Guideline Development Team which further reviewed the evidence and developed recommendations for screening and diagnosis of GDM [14]. In order to identify women at high risk of GDM all women have to be initially screened before 20 weeks' of gestation using glycated haemoglobin (HbA1c). Women with an HbA1c ≥ 50 mmol/L are referred to a service which specialises in diabetes in pregnancy, while women with an HbA1c 41-49 mmol/L are offered a two hour, 75 g oral glucose tolerance test (OGTT) at 24-28 weeks of gestation by the lead maternity carer (LMC), as they are considered at an increased risk of GDM. If fasting glucose is ≥ 5.5 mmol/L or two hour value is ≥ 9.0 mmol/L women will be referred to a service which specialises in diabetes in pregnancy, where they receive instructions on diet and lifestyle changes [15].

Diagnosis of gestational diabetes mellitus in New Zealand practice

Outside pregnancy an HbA1c of 41-49 mmol/L is classified as prediabetes while HbA1c of 50 mmol/L or more is classified as diabetes [16]. The study by Rowan and co-authors [17], raised the question about whether pregnant women with normal OGTT and raised HbA1c represent a clinically important group of women with GDM. The authors analysed data of eighty pregnant women with normal OGTT and HbA1c > 40 mmol/L, and compared them to data of pregnant women with GDM at the National Women's Hospital (NWH) database. Women with normal OGTT had higher BMIs (32.3 ± 8.1), and were more likely to be of Pacific ethnicity (43.8%). Increased values of HbA1c remained high in 61.8% of women postnataly, and with abnormal OGTT in 20.9% of women. An increase in HbA1c above 40 mmol/L identified women who are likely to undergo pharmacotherapy for their hyperglycaemia in pregnancy. These data also question the possible role of HbA1c in high risk women with non-diagnostic (normal) OGTT, and suggest of whether it is useful to request an HbA1c at other times during pregnancy.

An HbAc1 in early pregnancy will identify women with perhaps undiagnosed diabetes or prediabetes, and the level of risk of developing GDM [14]. The risk of developing type 2 diabetes is estimated to be six to eight times higher in women who were diagnosed with GDM than in women with no history of GDM.

Metabolic changes in normal pregnancy and GDM

Metabolic adaptations during pregnancy include changes in carbohydrate and lipid metabolism in order to provide a continuous supply of nutrients to the foetus despite intermittent maternal food intake [18-21]. During early pregnancy in healthy women, glucose tolerance, peripheral insulin sensitivity, and hepatic basal glucose production are normal. Glucose tolerance shows progressive increase in nutrient-stimulated insulin responses which are consistent with progressive insulin resistance with advancing pregnancy [22]. As a consequence, there is a progressive increase in basal and postprandial insulin concentrations [23-25]. Reduced insulin sensitivity and changes in β cell responsiveness probably occur in parallel with the release of hormones of pregnancy, human chorionic somatostatin (HCS), progesterone, cortisol and prolactin [26]. The cellular mechanism for this complex transition is obscure [27]. In late pregnancy, maternal fat tissue depots decrease, postprandial FFAs levels increase, and insulin-mediated glucose disposal worsens by 40-60% [28]. Reduced insulin secretion in late pregnancy is unable to suppress the whole body lipolysis, which is further reduced in GDM mothers, contributing to a greater postprandial rise in FFAs, increase in hepatic gluconeogenesis, and severe insulin resistance [29,30]. Also, increased serum levels of FFAs may exert toxic effects on β cell function [31].

Lipid metabolism during normal pregnancy is characterised by a physiological hyperlipidaemia [32-34]. In obese women and women with GDM abnormal lipid profile is associated with hyperinsulinemia, increased inflammatory markers, and elevated leptin levels. Alterations in maternal lipid metabolism may also contribute to foetal growth [35]. The enzyme placental lipoprotein lipase breaks down triglycerides to free fatty acids (FFA) that can be transferred to the foetus, increasing foetal adiposity and/or lipase activity of foetal adipose cells. Raised serum levels of FFAs might be responsible for the insulin resistance of GDM [36]. Total serum cholesterol levels remain high throughout the pregnancy in women with GDM [37]. Triglycerides remain significantly elevated across all three trimesters of pregnancy due to decreased lipoprotein lipase activity, particularly towards the end of pregnancy. An elevation of triglycerides in early pregnancy may be predictive of GDM, while increase in triglycerides and FFAs levels indicates a possibility of pre-eclampsia before the onset of clinical pre-eclampsia towards the last trimester of pregnancy [38,39]. However, hypertriglyceridemia and hyperinsulinemia are thought to be the key drivers of foetal macrosomia [40,41].

Predictive adaptive responses

Recent research has shown that environmental and behavioural influences, rather than genetics, are fuelling the current epidemic of obesity and that early life environment is a contributor to later life metabolic health [42]. The "developmental origins of health and disease" model (DOHaD), speculates that the foetus makes predictive adaptations to changes *in utero*, creating permanent adjustments in homeostatic systems to help immediate survival, and improve success in the adverse postnatal environment. These foetal adaptations known as predictive adaptive responses (PARs), may lead to an increased risk of diabetes and CVD in later life, via the inheritance of risk factors and a cycle of disease transmitted across generations [43,44]. For example, being exposed to a maternal diabetic *in utero* environment, new-borns may have a variety of phenotypic expressions. The phenotype of the new-born is generally perceived either as macrosomic or large for gestational age at birth (LGA), or small for gestational age (SGA) [45]. GDM increases the risk for maternal (pre-eclampsia, perineal trauma, and caesarean section) and perinatal complications (macrosomia, hypoglycaemia, shoulder dystocia, respiratory distress, jaundice, birth injuries, and stillbirth) [46]. New-borns of mothers with GDM are at risk of these complications, and probably poor glucose control is directly related to perinatal complications[47]. Proper clinical management of GDM or a tighter glucose control can reduce the risk of adverse pregnancy outcomes.

Ethnic diversity in adaptive predictive responses

The risks of being overweight or obese as a child, after *in utero* GDM exposure, are highly associated with the long-term health risks for type 2 diabetes and cardiovascular disease (CVD) in multi-ethnic

New Zealand, and in particular in Pacific and Maori children . About one in nine children (11%) between two and 14 years of age are obese [48], one in five (22%) are overweight children of Pacific Islands (27%), and the indigenous Maori populations respectively (19%).

Large for gestational age

Macrosomia or large for gestational age (LGA) is defined as a birthweight greater than 90th percentile for gestational age, or a birthweight greater than 4000 or 4500 g [49]. There is a relationship between the perinatal risks and early life complications associated with macrosomia [50]. The authors analysed data of 134 mothers of macrosomic new-borns, in their thirties, who were overweight (39), with previous macrosomia (28), and with family history of diabetes (41). The most of mothers were Europeans (64), Pacific Island women (45), Maori (7), and Asian (15) and others (3). Pregnancies in obese and hyperglycaemic mothers were completed with the induction of labour (58.8%), total (32.5%), and emergency Caesarean sections (19.4%), and birth to 11.3% macrosomic new-borns. Over half of the macrosomic new-borns of mothers screened for GDM (77.8%) suffered recurrent and/or symptomatic hypoglycaemia, and respiratory distress. Other perinatal complications caused by macrosomia were shoulder dystocia, low Apgar score, and Erb's palsy. Most of mothers with macrosomic new-borns were of Pacific Island ethnicity, who were expected to have large babies with respect to their body composition with more lean body mass, compared to European women.

Being macrosomic at birth is now considered a strong indicator that obesity often begins in early life [51]. Prenatal and early life factors that drive obesity in the offspring include maternal obesity, maternal diabetes and diet during pregnancy, socioeconomic factors, no or short breast feeding, and greater weight in early infancy. Gestational diabetes also imposes an inequitable disease burden on indigenous women and their new-borns [52]. The Pacific Island and Maori ethnic groups in New Zealand are genetically-prone populations to both type 2 diabetes and GDM. GDM increases the risk in their offspring of developing obesity and diabetes in later life. However, hyperinsulinaemia is a frequent finding at birth in these populations, and acts as a marker of their risk of increased growth and adiposity [53].

Data from the previous study fully support the results of the study by Simmons [54], where a large number of macrosomic new-borns from obese, glucose tolerant women was associated with the maternal weight and relative hyperglycaemia. A proportion of Pacific macrosomic new-borns with a birth weight above 4000g increased significantly, as the glucose concentration increased. Pacific Island new-borns were heaviest and Maori new-borns lightest. It was suggested that, in Pacific Island women, there might be a greater influence of increased maternal weight and glycaemia, rather than either alone. It is unclear whether it was greater foetal susceptibility to the *in utero* influence of maternal hyperglycaemia, or greater obesity in Pacific Island mothers. Either way, maternal obesity creates a significant risk for their offspring with metabolic compromise already apparent at birth [55].

Neonatal morbidity is common in new-borns of women of Pacific Island and Asian Indian ethnicities with high prevalence of type 2 diabetes and GDM [56]. Two thirds of the women diagnosed with GDM at the NWH ranged from 2% in Caucasian women to 7% in Pacific Island and Indian women, based on the criteria from the European Association for Study on Diabetes [57]. About 427 pregnancies were complicated by the underlying prevalence of GDM (382 new-borns) or type 2 diabetes (60 new-borns) at the NWH. Admission to NICU occurred in 29% of GDM and 40% of type 2 diabetes pregnancies. Hypoglycaemia and respiratory distress were the main neonatal complications requiring NICU admission. Nearly half the admissions were in preterm new-borns with GDM and type 2 diabetes. Macrosomia was the highest in the group of Pacific Island women with type 2 diabetes diagnosed either antenatally or postpartum. Similar to type 2 diabetes, a few women with GDM gave birth to new-borns with cardiovascular, renal and respiratory system abnormalities.

A common risk factor for the development of GDM is excessive weight gain (EWG) during pregnancy, that exposes the developing foetus to persistently raised concentrations of glucose, insulin, amino acids, lipids, and inflammatory cytokines produced by the maternal adipose tissue [58]. High pregravid or early pregnancy BMI is a strong predictor of EWG, which might lead to post-partum weight retention and development of persistent obesity in women of reproductive age and their new-borns [59]. About 163 of Auckland indigenous women (part of the SCOPE study) with singleton pregnancies had EWG, and of these 18 Polynesian and 20 Asian Indian women [60]. Women with high GWG had higher rates of caesarean section in labour, LGA new-borns, and hypertensive disorders of pregnancy.

Small for gestational age

In New Zealand about 3345 SGA new-borns were born in 2014, and accounted for 5.9% - 6.2% of all new-borns born each year from 2005 to 2014 [61]. The incidence of SGA has not been changed for the last decade. SGA new-borns were more commonly born to women in their forties (8.4%), or over and under 20 years of age (7.2%), in Indian and Maori mothers (9.6% and 6.9% respectively), and in mothers who lived in more deprived areas (6.4%).

SGA new-born has been defined as a birthweight of less than the tenth percentile using population based standards, which were derived from cohorts of Europeans in the Western countries, before the epidemic of obesity [62,63]. The implementation of customised birthweight centiles at birth is likely to identify more new-borns at risk of perinatal morbidity and mortality than would be identified by population centiles [64]. In the multi-ethnic general obstetric population in New Zealand, the primary goal was to identify independent risk factors for new-borns who were SGA by customised birthweight centiles [65]. Customised birthweight centiles were generated after birth, adjusted for maternal ethnicity, height, weight, and parity, and new-born's gender. Independent factors which were considered *a priori* a risk or protective for SGA were ethnicity, ethnic specific BMI, maternal age, parity, smoking status, socio-economic status, diabetes, hypertension, antepartum haemorrhage (APH), and pre-existing medical conditions associated with the growth restriction. Pre-pregnancy diagnosis of diabetes included type 1 diabetes mellitus, type 2 diabetes mellitus, and GDM, and/or unknown diabetes status.

The authors utilised a national database of births of 26,254 women with singleton pregnancies, delivered from January 2006 to December 2009 at the NWH. The percent of SGA new-borns by customised birthweight centiles was 11, 8% of the total study population. The authors notified that type 1 diabetes and GDM reduced the risk of SGA, while maternal obesity was found to have 24% increase in risk compared to women with normal gestational weight. Obesity is a clinically important risk factor for customised SGA, and remained independently associated with customised SGA. The association between SGA and maternal obesity raises a concern as SGA is less likely to be detected antenatally in obese women, while the association between customised SGA and socioeconomic status, initially observed in Maori and Pacific women, was explained by high cigarette smoking

rates, and obesity in deprived areas. Also, in association with the foetal growth, gestational hypertension occurred in 3.7% of new-borns and carried a nearly 50% increase in risk of SGA, while APH of unknown origin (4.1%) had a 70% increase in risk by customised birthweight centiles. However, foetal growth in women with GDM correlated with both the degree of glycaemia and presence or absence of vascular damage, which are two dominant and independent regulators of foetal growth [66].

Racial and ethnic origins are clearly associated to size at birth, and might partly influence mother's pre-gravid and gravid weight [67]. Women of Chinese and "other "Asian origins showed an approximately two-fold increased risk of delivering an SGA new-borns in comparison with European women, while Indian women had a four-fold risk. When compared with European women, Indian women remained at significantly increased risk of having an SGA new-born. Mothers of SGA new-borns were shorter and lighter than mothers who delivered AGA (average for gestational age). However, the results of the study indicated that maternal primiparity, Indian ethnicity, being short and light, and maternal hypertension were associated with the delivery of SGA new-borns. At the other end of the spectrum were women of Pacific ethnicity with the lower rates of preterm delivery, and decreased risk for delivering SGA compared to Maori and European and/other women, despite living in areas of high socioeconomic deprivation [68,69]. In Pacific born women, particularly those born in the Pacific islands, SGA was less common compared to those Pacific women born in New Zealand. Preterm birth rates and rates of SGA were the lowest in Pacific Island women, and declined by 30% during 1980-1994, compared to a 25% decline for Maori women and a 19% for European and/other women [70]. The rates of SGA for European and/other women declined the most slowly of the three ethnic groups. The known risk factors that might have played a role include maternal smoking and nutritional factors. However, socio-demographic factors tend to be significantly associated to dietary patterns of pregnant women [71].

The Auckland Birthweight Collaborative (ABC) study showed that mothers who smoked had a significantly increased risk of an SGA [72]. Up to 18% of women of SGA new-borns in the study population of 844 SGA could be related to smoking. Risk factors that also could contribute to SGA were Asian Indian ethnicity, women who developed pre-eclamptic toxaemia and pre-existing hypertension toxaemia. Mothers of SGA new-borns were shorter and reported a low pre-gravid weight.

GDM and type 2 diabetes in pregnancy

Pacific Island and Maori women are ethnic groups at high risk of both type 2 diabetes and GDM during pregnancy [53]. Exposure of the foetus to maternal diabetes influences changes in birthweight, adiposity, and foetal insulin production. In Maori and Pacific Island women with GDM (n=138), and type 2 diabetes (n=39), cord blood was taken from singleton new-borns at Middlemore Hospital, South Auckland, for the measurement of foetal insulin concentrations, insulin propeptides, and leptin, together with the measures of obesity in new-borns such as weight and skinfold thicknesses. Mothers with both GDM and type 2 diabetes were older, more parous, and heavier. Postpartum, 79% (107) of mothers with GDM underwent OGTT, and 20% of women were diagnosed with type 2 diabetes, and 34% of mothers with impaired glucose tolerance (IGT). It has been suggested that mothers with GDM before pregnancy have had pre-existing and clinically undetected IGT. Postpartum, values of HbA1c were significantly increased in mothers with newly diagnosed type 2 diabetes. Moreover, the study by McGrath and colleagues [73], showed that 19% of 110 women with GDM developed type 2 diabetes after a follow up of 2.4 years. The authors suggest that there were no significant differences between women with GDM and type 2 diabetes, although essential hypertension present in 28% of mothers with type 2 diabetes, and only 2% in mothers with GM, and in addition significant rise in HbA1c in mothers with type 2 diabetes at 30 weeks of pregnancy and at the end of pregnancy, might make the difference.

GDM and type 2 diabetes in Pacific Island and Maori new-borns were associated with higher birthweight, skinfold thicknesses, raised cord insulin, insulin peptides, and increased leptin concentrations. Increased insulin and insulin pro-peptides better reflected foetal growth, while leptin concentration at birth reflected adiposity in new-borns of mothers with both GDM and type 2 diabetes [74].

Given the impact of diabetes in pregnancy, particularly undiagnosed type 2 diabetes, GDM screening in early pregnancy will offer potential benefits for indigenous women [75], Pacific Island, and Asian Indian women, populations genetically prone to type 2 diabetes.

Gestational diabetes in twin pregnancies

In New Zealand, the vast majority of women in 2014 (98.5%) gave birth to one new-born and 1.5% (855 women) gave birth to two or more new-borns [61]. During the last decade the proportion of twin and multiple births was not changed significantly and is ranging from .4% to 1.6%, although the proportion of emergency and elective Caesarean sections increased with a number of new-borns. About 25.4% of women with a singleton pregnancies had a Caesarean section compared to 60.3% of women with twin pregnancies, and 90.0% of women with multiple pregnancies.

During twin pregnancies higher levels of hormones such as human placental lactogen, estrogen and progesterone are present than in singleton pregnancies, and due to their insulin antagonistic effects might influence the rise of GDM [76]. GDM is also associated with increased placental mass in twin pregnancies when compared to singleton pregnancies. Literature review showed that there was insufficient data concerning the effects of GDM on perinatal outcomes in twin and multiple pregnancies in New Zealand. The study by Simmons and Yapa [77], showed the association of GDM and twin pregnancies in NZ multi-ethnic population. It was notified that Polynesian women have high incidence of GDM, although there was no ethnic difference in incidence of twin pregnancies. Women with twin pregnancies were older, multiparous, and weighed more than those with a singleton pregnancies at the same gestation. Twin pregnancies were associated with greater risk of GDM and relative hyperglycaemia. There were about 4,939 deliveries at South Auckland Hospital, with 54 (1.1) twin pregnancies Europeans (1.2%), Maori (0.8%), Pacific Island (1.4%) and others (0.4%). Overall incidences of pregnancies with GDM were 11.9% in women with twin pregnancies and 5.1% in women with singleton pregnancies. Since twin pregnancies are more prone to GM, women with twin pregnancies should be tested for GDM not only at 24-28 weeks of gestation but also throughout the pregnancy. Newly developed guidelines for screening ad diagnosis of GDM in New Zealand would probably increase the overall incidence of GDM in twin and multiple pregnancies.

Conclusion

GDM is the most common metabolic disorder during pregnancy. In New Zealand the rise in GDM prevalence reflects the current patterns of increasing obesity and diabetes. The prevalence of type 1

and type 2 diabetes, and GDM is rising among women of South Asian, Pacific Island, and indigenous Maori ethnic groups. Screening for GDM is recommended for all pregnant women in New Zealand, unless an earlier diagnosis of diabetes was made. Screening rates for GDM among Maori and Pacific Island women are very low, although they are at high risk of diabetes, and also they are overrepresented amongst the obese groups (40% and 62% respectively).

The most frequently reported risk factors for GDM are maternal older age, weight and parity, family history of diabetes, and previous delivery of macrosomic new-borns. It is difficult to distinguish whether GDM was undiagnosed before pregnancy or induced by pregnancy in women of childbearing age. GDM increases the risk for maternal and perinatal complications (macrosomia, hypoglycaemia, shoulder dystocia, respiratory distress, jaundice, birth injuries, and stillbirth). Macrosomic new-borns of mothers with GDM are at risk of these complications, and probably poor glucose control is directly related to perinatal complications. Being macrosomic at birth is now considered a strong indicator that obesity often begins in early life. GDM and type 2 diabetes in Pacific Island and Maori new-borns are associated with higher birthweight, skinfold thicknesses, raised cord insulin, insulin peptides, and increased leptin concentrations. Macrosomic new-borns commonly suffer postnatal hypoglycaemia, respiratory distress, and shoulder dystocia as the major complications. Prenatal and early life factors that drive obesity in the offspring include maternal obesity, maternal diabetes and diet during pregnancy, socioeconomic factors, no or short breast feeding, and greater weight in early infancy.

The Pacific Island and Maori ethnic groups in New Zealand are genetically-prone populations to both type 2 diabetes and GDM. Hyperinsulinaemia is a frequent finding at birth in these populations, and acts as a marker of their risk of increased growth and adiposity. It is unclear whether it was greater foetal susceptibility to the *in utero* influence of maternal hyperglycaemia, or greater obesity in Pacific Island and Asian Indian mothers.

Maternal obesity creates a significant risk for SGA new-borns born to women in their forties, or over and under 20 years of age, in Indian and Maori mothers, and in mothers who lived in more deprived areas. Mothers of SGA new-borns were shorter and lighter than mothers who delivered AGA. Indian ethnicity, being short and light, smoking, maternal hypertension and pre-eclamptic toxaemia and pre-existing hypertension toxaemia were associated with the delivery of SGA new-borns. However, socio-demographic factors tend to be significantly associated to dietary patterns of pregnant women.

The implementation of customised birthweight centiles at birth, is likely to identify more new-borns at risk of perinatal morbidity and mortality than would be identified by population centiles. Also, mothers with twin pregnancies were associated with greater risk of GDM and relative hyperglycaemia. Therefore, they should be tested for GDM not only at 24-28 weeks of gestation but also at early pregnancy and throughout the pregnancy. Proper clinical management of GDM or a tighter glucose control can reduce the risk of adverse pregnancy outcomes.

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