

Original Article

Impact of type 2 diabetes, obesity and glycaemic control on pregnancy outcomes

Sally K. ABELL,^{1,2} Jacqueline A. BOYLE,^{3,4} Barbora de COURTEN,^{2,5} Georgia SOLDATOS,^{1,2} Euan M. WALLACE,^{6,7} Sophia ZOUNGAS^{2,8} and Helena J. TEEDE^{1,2}

¹Monash Centre for Health Research and Implementation, School of Public Health and Preventive Medicine, Monash University,

²Diabetes and Vascular Medicine Unit, Monash Health, ³Refugee and Indigenous Women's Health, Monash Centre for Health Research and Implementation, School of Public Health and Preventive Medicine, Monash University, ⁴Monash Women's Services, Monash Health,

⁵Chronic Disease Prevention, Monash Centre for Health Research and Implementation, School of Public Health and Preventive Medicine, Monash University, ⁶Monash Women's Maternity Services, Monash Health, ⁷Department of Obstetrics and Gynaecology, Ritchie Centre, and ⁸Diabetes, Vascular Health and Ageing, Monash Centre for Health Research and Implementation, School of Public Health and Preventive Medicine, Monash University, Melbourne, Victoria, Australia

Background: There are no contemporary cohorts examining pregnancy outcomes in women with type 2 diabetes (T2D) in Australia.

Aim: To compare pregnancy outcomes in women with and without T2D, and assess effects of body mass index (BMI) and glycaemic control on outcomes.

Materials and methods: An historical cohort study was conducted of all singleton births > 20 weeks gestation at a specialist maternity network in Australia from 2010 to 2013. Data were extracted from the Birthing Outcomes System database. Multivariable logistic regression analysis was used to examine associations between presence of T2D and pregnancy outcomes.

Results: Outcomes for 138 pregnancies with T2D and 27 075 pregnancies in women without diabetes were compared (type 1 diabetes and gestational diabetes excluded). Women with T2D were older and more overweight compared to women without diabetes ($P < 0.01$). Their babies were born earlier ($P < 0.01$) with increased risk of large for gestational age (adjusted odds ratio 2.13 (95% CI 1.37–3.32)), hypoglycaemia (4.90 (2.79–8.61)), jaundice (2.58 (1.61–4.13)) and shoulder dystocia (2.72 (1.09–6.78)), but not congenital malformations or perinatal death. Women with T2D had a higher risk of induction (4.03 (2.71–5.99)), caesarean section (2.10 (1.44–3.04)), preterm birth (2.74 (1.78–4.24)) and pre-eclampsia (2.75 (1.49–5.10)). An HbA1c $\geq 6.0\%$ (42 mmol/mol) was associated with increased preterm birth, special care nursery admission, hypoglycaemia and jaundice.

Conclusions: Despite availability of preconception care, good glycaemic control and specialist management, T2D remains associated with increased adverse obstetric and neonatal outcomes. Further research to examine predictors of adverse outcomes may assist in targeted antenatal surveillance and management.

Key words: glycaemic control, obesity, pregnancy outcome, type 2 diabetes.

Introduction

Rising obesity, physical inactivity and later child-bearing have resulted in increased prevalence of type 2 diabetes

(T2D) in pregnancy, which poses a growing public health challenge.¹ T2D in pregnancy results in increased risks to both the mother and child. Microvascular complications can first appear or worsen during pregnancy.² Miscarriage, pre-eclampsia, preterm birth, induction of labour and caesarean section are more common.^{2,3} Babies are at increased risk of congenital malformations, perinatal mortality, macrosomia and related complications.² Better glycaemic control has improved but not eliminated these risks.⁴ Obesity further increases the risk of hypertensive disorders, interventional delivery and prematurity.¹

Recent literature has focused on adverse pregnancy outcomes in women with T2D compared to type 1 diabetes (T1D),^{5–7} or has examined combined risks of both.^{8,9} Controversy remains over which type of diabetes presents

Correspondence: Professor Helena Teede, Monash Centre for Health Research and Implementation, School of Public Health and Preventive Medicine, Monash University, Locked Bag 29 Clayton, Melbourne, Vic. 3168, Australia.
Email: Helena.teede@monash.edu

Conflicts of Interest Statement: There are no conflicts of interest.

Received 5 May 2016; accepted 24 July 2016.

greater risks.^{10–13} Limited recent studies used a non-diabetic comparison group, or did not adequately control for confounders such as ethnicity, obesity and glycaemic control.^{6,14,15} Past literature also comes from settings with variable antenatal healthcare access and expertise.^{8,11} As such, they lack generalisability and clinical applicability.

To our knowledge, there are no recent Australian studies evaluating risk of adverse outcomes in women with T2D compared to women without diabetes. Our purpose was to provide an update in contemporary risk of adverse outcomes, having accounted for potential confounders including age, country of birth, parity and obesity. Further, we aimed to assess the independent effects of obesity, microvascular complications and glycaemic control on outcomes in T2D.

Materials and Methods

An historical cohort study was conducted of all singleton births from 20 weeks gestation at Monash Health in Victoria, Australia from 2010 to 2013. Monash Health includes Clayton, Dandenong and Casey maternity units, and is one of Australia's largest quaternary public health services (7500 births/year). Monash Health provides specialised endocrine, diabetes educator, obstetric, midwifery and neonatal joint services. The study was approved by the Monash Health Human Research Ethics Committee in 2013 (Approval no. 14001Q).

Data was obtained from the Birthing Outcomes System database, where midwives prospectively record pregnancy health information from the booking antenatal visit until delivery and discharge from hospital postnatally. The database includes maternal demographics, family history, medical, obstetric and gynaecological history, antenatal care, birth and neonatal outcomes. The data is standardised and outcomes are well-defined for statutory reporting to a state-wide perinatal database.

Antenatal characteristics, maternal outcomes and neonatal outcomes were collected for women with T2D and women with normal glucose tolerance. Women with T1D and gestational diabetes mellitus (GDM) were excluded. During this period, a two-step diagnostic process was used for universal screening for GDM with a 50 g glucose challenge test and 75 g oral glucose tolerance test (OGTT) at 24–28 weeks gestation (Australasian Diabetes in Pregnancy Society 1998 criteria). 'Pre-existing diabetes' was recorded in the dataset based on patient or midwife report, shown to be reported with high accuracy.¹⁶ Type of diabetes and complications were validated by a clinician using individual medical records. Glycaemic data was obtained from laboratory results. HbA1c % (mmol/mol) was measured using the Arkray Adams high performance liquid chromatography (HPLC) Method (coefficient of variation 1.4%, Japan). HbA1c data was considered 'adequate' if measured in two or three trimesters. Body mass index (BMI) was recorded at the booking antenatal visit, and categorised according to the World Health Organisation.¹⁷

Women with T2D were initially managed in a specialist diabetes clinic then transferred to a multidisciplinary diabetes and maternity service from 12 weeks gestation. Treatment of T2D included diet, insulin and/or metformin. Women without diabetes in pregnancy were managed by midwives and obstetricians through hospital antenatal clinics.

Outcomes

Primary outcomes were large for gestational age (LGA) and small for gestational age (SGA) defined as > 90th and < 10th centiles, respectively, adjusted for gestational age and sex using Australian birthweight percentiles.¹⁸ Macrosomia (≥ 4000 g) and low birth weight (LBW) (< 2500 g) were reported.

Secondary maternal outcomes were induction of labour (IOL), caesarean section, preterm birth (< 37 weeks gestation), gestational hypertension (new onset from 20 weeks with systolic and/or diastolic blood pressure $\geq 140/90$ mmHg) and pre-eclampsia (hypertension with proteinuria > 300 mg/24 h, spot urine protein : creatinine ratio ≥ 0.03 g/mmol and/or other system involvement). In women with T2D, presence of microvascular complications including retinopathy (based on clinician report or clinical review) and nephropathy (micro or macroalbuminuria based on clinician report or urine testing) were reported.

Secondary neonatal outcomes were admission to the special care nursery (SCN) or neonatal intensive care unit (NICU), hypoglycaemia (< 2.6 mmol/L), jaundice requiring phototherapy and respiratory distress syndrome. Shoulder dystocia and Apgar score < 7 at five minutes were reported for vaginal deliveries. Congenital malformations (minor and major) and perinatal deaths (stillbirths after 20 weeks gestation and early neonatal deaths) were reported.

Statistical analyses

Maternal characteristics were analysed using descriptive statistics. Categorical data were compared using the Pearson chi-square test or Fisher exact test. Student's *t*-tests, Mann–Whitney *U*-tests and Kruskal–Wallis tests were used for continuous data as appropriate. Univariable and multivariable logistic regression analysis were used to express odds ratios (OR) and 95% confidence intervals (CI) for each pregnancy outcome in women with T2D relative to women without diabetes (reference category). Covariates were included in the multivariable model if they were clinically or statistically significant ($P < 0.1$) at the univariable level. Model covariates tested included maternal age, BMI, country of birth, parity, smoking status, pre-existing hypertension or pre-eclampsia and gestational age. Interactions were reported if significant. Fewer confounders were included if the number of cases were low ($n < 10$) to avoid overfitting. The likelihood ratio test, Akaike and Bayesian

information criteria, and area under the curve statistics were used to select final models. Logistic regression was adjusted for possible clustering (intraclass correlation) as some women had multiple births during the study. A subanalysis was performed in women with T2D examining the effects of BMI, microvascular complications and HbA1c on pregnancy outcomes. Statistical significance was conveyed by a two-sided *P*-value of 0.05. Analyses were performed using Stata version 12 (StataCorp, College Station, TX, USA).

Results

Women with T2D compared to women without diabetes

There were 138 singleton pregnancies in 124 women with T2D and 27 075 singleton pregnancies in 23 918 women without diabetes. Women with T2D were older and more overweight compared to women without diabetes ($P < 0.001$). Overall, 79.4% of women with T2D were overweight or obese compared to 45.0% of women without diabetes. Women with T2D were more likely to be parous ($P = 0.01$) and to have pre-existing hypertension ($P < 0.001$). Babies of women with T2D were born earlier than babies of women without diabetes ($P < 0.001$) and there was no difference in mean birthweight (Table 1).

In babies of women with T2D compared to those born to women without diabetes, there was no difference in macrosomia (14.5% vs 10.4%, $P = 0.118$) but there were more low birth weight babies (15.9% vs 7.3%, $P < 0.001$). When corrected for gestational age and sex, babies of women with T2D had increased risk of LGA (adjusted OR 2.13 (95% CI 1.37–3.32)), but no difference in risk of SGA (1.38 (0.87–2.20)) (Table 2). Being obese (2.87 (2.55–3.23)) and overweight (1.87 (1.67–2.10)) were associated with increased risk of LGA.

Women with T2D had higher risk of IOL (adjusted OR 4.03 (95% CI 2.71–5.99)), caesarean section (2.10 (1.44–3.04)) and preterm birth (2.74 (1.78–4.24)) compared to women without diabetes (Table 2). Of preterm births, women with T2D were more likely to require caesarean section (54.8% vs 34.6%, $P = 0.019$). Women with T2D had increased risk of pre-eclampsia (2.75 (1.49–5.10)); however, this was not significant if adjusted for pre-existing hypertension (1.91 (0.99–3.67)).

Babies of women with T2D had higher risk of SCN admission (adjusted OR 19.34 (95% CI 12.37–30.25)), hypoglycaemia (4.90 (2.79–8.61)), jaundice (2.58 (1.61–4.13)) and shoulder dystocia (2.72 (1.09–6.78)) compared to babies of women without diabetes. There was no difference in risk of NICU admission, respiratory distress, Apgar score < 7 at five minutes, congenital malformation or perinatal death after adjustment for confounders (Table 2).

Table 1 Maternal demographic and health characteristics for all mothers and neonates

Variable	Type 2 diabetes (<i>n</i> = 138)	No diabetes (<i>n</i> = 27 075)	<i>P</i> -value
Maternal age, mean (SD)	33.6 (5.3)	29.4 (5.4)	<0.001
Maternal BMI in kg/m ² , median (IQR)	32.9 (26.2–36.9)	24.4 (21.7–28.3)	<0.001
Maternal BMI category, <i>n</i> (%)			
Normal weight	28 (20.6)	14 794 (55.0)	<0.001
Overweight	29 (21.3)	7075 (26.3)	
Obese	79 (58.1)	5026 (18.7)	
Country of birth, <i>n</i> (%)			
Australia + New Zealand	64 (46.4)	12 806 (47.3)	0.829
European + American countries	4 (2.9)	1850 (6.8)	0.067
Africa	18 (13.0)	1798 (6.6)	0.003
Asian countries	52 (37.7)	10 621 (39.2)	0.710
Parity, <i>n</i> (%)			
Primiparous	45 (32.6)	11 805 (43.6)	0.009
Parous	93 (67.4)	15 270 (56.4)	
Smoker, <i>n</i> (%)	17 (12.3)	4703 (17.4)	0.118
Pre-existing hypertension, <i>n</i> (%)	17 (12.3)	215 (0.8)	<0.001
Sex of neonate, <i>n</i> (%)			
Male	63 (45.7)	13 897 (51.3)	0.385
Female	75 (54.4)	13 156 (48.6)	
Gestation at birth in weeks, median (IQR)	38.3 (37.1–39.1)	39.4 (38.4–40.4)	<0.001
Neonatal birth weight in grams, mean (SD)	3233 (2854–3660)	3350 (3005–3690)	0.063

Data are presented as count (proportion), mean (SD) or median (IQR).

BMI, body mass index measured at booking visit; IQR, interquartile range; SD, standard deviation.

Table 2 Risk of maternal and neonatal adverse outcomes in women with type 2 diabetes compared to women without diabetes

Outcome	Type 2 diabetes (<i>n</i> = 138)	No diabetes (<i>n</i> = 27 075)	Crude OR (95% CI)	Adjusted OR (95% CI)
LGA†	28 (20.3)	2087 (7.7)	3.05 (1.99–4.67)	2.13 (1.37–3.32)
SGA†	22 (15.9)	3964 (14.7)	1.10 (0.70–1.74)	1.38 (0.87–2.20)
IOL‡	74 (53.6)	5738 (21.2)	4.30 (3.04–6.08)	4.03 (2.71–5.99)
Caesarean section‡	74 (53.6)	7116 (26.3)	3.24 (2.28–4.62)	2.10 (1.44–3.06)
Preterm birth§	31 (22.5)	2186 (8.1)	3.30 (2.16–5.04)	2.74 (1.78–4.24)
Gestational hypertension¶	7 (5.1)	527 (2.0)	2.69 (1.26–5.77)	1.58 (0.73–3.43)
Pre-eclampsia¶	12 (8.7)	645 (2.4)	3.90 (2.15–7.08)	2.75 (1.49–5.10)
SCN**	98 (78.4)	4142 (15.9)	19.21 (12.50–29.52)	19.34 (12.37–30.25)
NICU††	10 (7.4)	727 (2.7)	2.87 (1.50–5.47)	1.94 (0.94–4.01)
Hypoglycaemia¶‡‡	31 (22.5)	1074 (4.0)	7.01 (4.66–10.56)	4.90 (2.79–8.61)
Jaundice¶‡‡	28 (20.3)	1737 (6.4)	3.71 (2.45–5.64)	2.58 (1.61–4.13)
Respiratory distress††‡‡	10 (7.3)	1039 (3.8)	1.96 (1.03–3.71)	0.78 (0.38–1.60)
Shoulder dystocia§§	5/64 (7.8)	498/19 958 (2.5)	3.31 (1.34–8.19)	2.72 (1.09–6.78)
Apgar < 7 at five minutes‡‡§§	5/64 (7.8)	577/19 887 (2.9)	2.83 (1.13–7.12)	0.91 (0.30–2.80)
Congenital malformation	6 (4.4)	996 (3.7)	1.19 (0.52–2.71)	1.00 (0.99–1.01)
Perinatal death	3 (2.2)	394 (1.5)	1.50 (0.48–4.75)	1.40 (0.44–4.46)

Data are presented as count (proportion), crude and adjusted odds ratios (OR) and 95% confidence interval (CI).

All outcomes are adjusted for age and BMI category (normal < 25 kg/m², overweight 25–29.9 kg/m², obese ≥ 30 kg/m²). Additional adjustments:

†Parity, smoking, country of birth.

‡Parity, smoking, pre-eclampsia.

§Smoking, country of birth, pre-eclampsia.

¶Parity.

**Parity, smoking.

††Parity, country of birth.

‡‡Gestation at birth.

§§Apgar score < 7 at five minutes and shoulder dystocia are reported for vaginal delivery only. There was an interaction between the presence of T2D and gestational age on risk of hypoglycaemia: if born at term (8.61 (5.23–14.18)); if born preterm (1.56 (0.68–3.64)).

IOL, induction of labour; LGA, large for gestational age; NICU, neonatal intensive care unit; SCN, special care nursery; SGA, small for gestational age.

T2D subanalysis

Women with T2D had a median HbA1c of 6.4% (46 mmol/mol) (interquartile range 5.9–7.3% (41–56 mmol/mol)) across the pregnancy. Glycaemic data were missing or incomplete in 31 women. Overall, 34 women had HbA1c measured in only one trimester, and 73 women had adequate HbA1c data with measurement in two or three trimesters (*n* = 50 two trimesters, *n* = 23 three trimesters). In the first trimester (*n* = 77 results) median HbA1c was 6.8%, in the second trimester (*n* = 55 results) median HbA1c was 6.2% and in the third trimester (*n* = 71 results) median HbA1c was 6.1%. Women received insulin in 125 pregnancies, insulin and metformin in 21 pregnancies and metformin alone in three pregnancies. Overall, 3/96 (3.1%) women had retinopathy and 8/96 (8.3%) had nephropathy recorded during pregnancy. Treatment type and presence of microvascular complications were not associated with adverse outcomes.

Risk of SCN admission was significantly higher when women were overweight (adjusted OR 6.14 (95% CI 1.19–31.81)) or obese (3.69 (1.05–12.99)) compared to normal weight. Increasing BMI was not associated with other adverse outcomes in women with T2D relative to normal BMI (data not shown).

A 1% increase in mean HbA1c during pregnancy was associated with increased risk of gestational hypertension (adjusted OR 2.23 (95% CI 1.27–3.90)), macrosomia (1.56 (1.07–2.26)), neonatal hypoglycaemia (1.46 (1.03–2.07)), and perinatal death (2.80 (1.09–7.17)) (Table 3). A mean HbA1c ≥ 6.0% (42 mmol/mol) was associated with increased risk of preterm birth, SCN admission, hypoglycaemia and jaundice compared to < 6.0% (all *P* < 0.05) (Table 4).

Discussion

We compared contemporary outcomes in 138 pregnancies in women with T2D and 27 075 pregnancies in women without diabetes managed at one of Australia's largest public diabetes and maternity services. Women with T2D were older, more overweight and more likely to have pre-existing hypertension than women without diabetes. Women with T2D had increased risk of pre-eclampsia, IOL, caesarean section and preterm birth. Their babies had increased risk of LGA, hypoglycaemia, jaundice, shoulder dystocia and SCN admission. There were no increases in congenital malformations after 20 weeks or perinatal death. In women with T2D, obesity was associated with increased risk of SCN admission, but no

Table 3 Glycaemic control during pregnancy in women with type 2 diabetes and risk of maternal and neonatal adverse outcomes

Outcome	Type 2 diabetes <i>n</i> (%)	Crude OR (95% CI)	Adjusted OR (95% CI)
Mean HbA1c during pregnancy (<i>n</i> = 107 women with HbA1c data available)			
LGA	21 (19.6)	1.53 (1.09–2.17)	1.56 (1.07–2.26)
SGA†	17 (15.9)	1.05 (0.71–1.55)	1.28 (0.81–2.01)
IOL‡	59 (55.1)	0.95 (0.71–1.28)	1.05 (0.74–1.51)
Caesarean section†,‡	55 (51.4)	0.99 (0.74–1.33)	1.03 (0.70–1.50)
Preterm birth†,§	23 (21.5)	1.31 (0.94–1.83)	1.32 (0.86–2.04)
Gestational hypertension	6 (5.6)	2.23 (1.31–3.79)	2.23 (1.27–3.90)
Pre-eclampsia	8 (7.5)	1.21 (0.74–1.98)	1.13 (0.65–1.96)
SCN‡	77 (79.4)	1.59 (0.98–2.60)	1.46 (0.89–2.40)
NICU	8 (7.6)	1.28 (0.77–2.13)	0.86 (0.42–1.79)
Hypoglycaemia	25 (23.4)	1.42 (1.03–1.98)	1.46 (1.03–2.07)
Jaundice‡,¶	24 (22.4)	1.08 (0.77–1.52)	0.80 (0.50–1.27)
Congenital malformation	4 (3.7)	1.40 (0.75–2.61)	1.56 (0.81–3.00)
Perinatal death	2 (1.9)	2.37 (1.02–5.49)	2.80 (1.09–7.17)

Data are presented as count (proportion), crude and adjusted odds ratios (OR) and 95% confidence interval (CI).

All outcomes are reported in relation to mean HbA1c for each pregnancy.

All outcomes adjusted for age and BMI.

Additional adjustments:

†Parity.

‡Smoking.

§Pre-eclampsia.

¶Gestation at birth.

IOL, induction of labour; LGA, large for gestational age; NICU, neonatal intensive care unit admission; SCN, special care nursery; SGA, small for gestational age.

Table 4 HbA1c category and rates of adverse maternal and neonatal outcomes in women with type 2 diabetes

Outcome	HbA1c < 6.0% (<42 mmol/mol) <i>n</i> = 36	HbA1c ≥ 6.0% (≥42 mmol/mol) <i>n</i> = 71	<i>P</i> -value
LGA	4 (11.1)	17 (23.9)	0.114
SGA	3 (8.33)	5 (7.04)	1.000
IOL	20 (55.6)	39 (54.9)	0.951
Caesarean section	20 (55.6)	35 (49.30)	0.540
Preterm birth	2 (5.6)	21 (29.6)	0.005
Gestational hypertension	0 (0)	6 (8.5)	0.095
Pre-eclampsia	1 (2.8)	7 (9.9)	0.262
SCN	22 (64.7)	55 (87.3)	0.009
NICU	2 (5.6)	6 (8.7)	0.712
Hypoglycaemia	3 (8.3)	22 (31.0)	0.008
Jaundice	2 (5.6)	22 (31.0)	0.003
Congenital malformations	1 (2.8)	3 (4.2)	1.000
Perinatal death	0 (0)	2 (2.8)	0.549

Data are presented as count (proportion).

IOL, induction of labour; LGA, large for gestational age; NICU, neonatal intensive care unit admission; SCN, special care nursery; SGA, small for gestational age.

other adverse outcomes. An HbA1c ≥ 6.0% (42 mmol/mol) was associated with increased risk of preterm delivery, SCN admission, hypoglycaemia and jaundice.

T2D in pregnancy is increasingly common with important risks for the mother and baby. Up to 30–40% of women with T2D may have an LGA infant and many babies have disproportionate abdominal growth.¹⁹ A population study in South Australia²⁰ (1986–2000) found a relative risk of 2.36 (1.79–3.12) in women with pre-existing diabetes compared to the background population, but they did not adjust for BMI or differentiate risk in T2D. In our more recent cohort of women with T2D with reasonable glycaemic control and specialist care, there was a two-fold risk of LGA (20.3% vs 7.7%) after adjustment for BMI and other confounders. In a systematic review, Gizzo *et al.*²¹ reported macrosomia rates are improving (~21.7% between 2000 and 2012), but studies used varying definitions and did not account for gestational age at birth. Thus, LGA continues to occur in T2D, independent of obesity, despite specialist care and good diabetes control. This has implications for offspring risk of future adiposity and cardiometabolic risk.⁴ Other contributors to excess fetal growth such as gestational weight gain and maternal lipids must also be considered.^{4,21,22}

Hospital protocol often dictates a lower threshold for obstetric intervention and implicates earlier birth for women with T2D. In the US, Knight *et al.*²³ found a four-fold risk of a composite delivery endpoint in T2D compared to a BMI-matched non-diabetic group, adjusting for age, smoking, hypertension and gestational age at delivery. We have advanced this data with

individual delivery outcomes, including a four-fold risk of IOL, two-fold risk of caesarean section and three-fold risk of preterm birth, after adjusting for confounders. Increased obstetric intervention and earlier birth in women with T2D relates to a combination of patient, obstetrician and hospital factors, and in this context it is difficult to discern their individual contributions.

T2D is associated with short-term risks to the neonate requiring close surveillance. We detected a five-fold risk of hypoglycaemia, and two- to three-fold risk of jaundice and shoulder dystocia in babies of women with T2D compared to women without diabetes. Comparison to existing literature is difficult where specific definitions for these outcomes are lacking, but these rates appear comparable, if not better.^{6,21,23} Furthermore, we observed a > 20-fold risk of SCN admission in babies of women with T2D. Intensive monitoring of these babies appears justified and preventative strategies should remain a key priority.

High rates of perinatal mortality and congenital malformations have been reported in T2D. An Australian cohort study²⁰ reported double the risk of congenital anomalies in women with pre-existing diabetes compared to the general population from 1986 to 2000. A small prospective Irish study⁸ ($n = 24$ with T2D, 2006–2007) found perinatal mortality rates 3.5 times and congenital malformation two times higher than the general population. In our larger and more recent cohort we found no difference in risk of congenital malformations after 20 weeks or perinatal death, suggesting improvements in glycaemic control and greater surveillance of women with T2D is effective.¹⁵

Obesity is a known contributor to adverse pregnancy outcomes,²³ but less is known about the independent effects of BMI in T2D. In our cohort, 80% of women with T2D were overweight or obese compared to 45% without diabetes. We observed increased risk of SCN admission in babies of overweight and obese women with T2D, but surprisingly no association with other pregnancy outcomes. It is possible that effects of obesity in T2D are somewhat mitigated by greater attention to antenatal diet, physical activity, weight gain, fetal surveillance and earlier delivery than in their non-diabetic counterparts. Regardless, high rates of obesity are a significant concern for women of reproductive age and this highlights the importance of weight management and education preconception to reduce risk of adverse outcomes in both groups.

HbA1c is a recognised risk factor for congenital malformation and perinatal mortality, but the association with other pregnancy outcomes is less clear.^{24,25} A retrospective study of T2D in the US (2003–2011) found that early pregnancy HbA1c was not a good predictor of composite maternal or fetal outcomes.²⁶ Conversely, third trimester HbA1c was a risk factor for LGA in a prospective cohort with pre-existing diabetes in East Anglia (2006–2009).⁶ In our cohort, a mean pregnancy HbA1c $\geq 6.0\%$ (42 mmol/mol) was associated with increased risk of preterm birth, SCN admission,

hypoglycaemia and jaundice compared to $< 6.0\%$. This emphasises the vital importance of optimising control at both preconception and in pregnancy.

Our cohort is one of the largest and most contemporary studies of pregnancy outcome in T2D and updates Australian literature. We have characterised in detail maternal demographics, antenatal care and pregnancy outcomes with management at a specialist maternity network. We have applied rigorous statistical methodology to adjust for confounders, enhancing generalisability of our results. Given our data captures births from 20 weeks gestation, we may have underrepresented the proportion of pregnancies with congenital malformations and those with miscarriages or terminations that did not continue beyond 20 weeks. Our data is also limited by absence of information regarding diabetes duration and gestational weight gain, and the analysis of HbA1c may be subject to bias due to missing data. Small sample size for some outcomes resulted in decreased precision of effect estimates, and we cannot exclude the possibility of type 2 error. Lastly, it is possible that some women with unrecognised T2D were misclassified as GDM during the study period. We expect this number to be small based on local data following implementation of an early screening program and recognition of newly diagnosed diabetes in pregnancy.

Conclusion

In our large cohort with good glycaemic control and evidence-based management in a multidisciplinary antenatal service, rates of congenital malformations and perinatal mortality similar to background rates were achievable. However, LGA, jaundice, neonatal hypoglycaemia and shoulder dystocia remain increased and an HbA1c $\geq 6.0\%$ (42 mmol/mol) was associated with adverse outcomes. The high prevalence of T2D necessitates understanding of related adverse pregnancy outcomes and their contributors. It is crucial to identify and target these women at preconception, to optimise glycaemic control, weight and other risk factors, and maintain close surveillance during pregnancy to minimise adverse outcomes.

Acknowledgements

HJT, JAB and BdC are National Health and Medical Research Council (NHMRC) postdoctoral fellows and SZ is a National Heart Foundation postdoctoral fellow. SKA holds a NHMRC postgraduate scholarship. The authors would like to acknowledge senior biostatistician Sanjeeva Ranasingha (Monash Centre for Health Research and Implementation, Monash University) who provided advice on study design and statistical methodology, and Amanda Kendell (Monash Women's Services, Monash Health) for her assistance in data acquisition and interpretation.

Funding

No funding was received.

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