A Comparison of Rates, Risk Factors, and Outcomes of Gestational Diabetes Between Aboriginal and Non-Aboriginal Women in the Saskatoon Health District

ROLAND DYCK, MD[1]
HELENA KLOMP, MSC[1]
LEONARD K. TAN, MB[2]
ROGER W. TURNELL, MD[3]
MAKRAM A. BOCTOR, MD[1]

OBJECTIVE — To determine possible differences in gestational diabetes mellitus (GDM) between aboriginal and non-aboriginal people in the Saskatoon Health District.

RESEARCH DESIGN AND METHODS — This was a prospective survey of all women admitted for childbirth to the Saskatoon Royal University Hospital between January and July 1998. We compared prevalence rates, risk factors, and outcomes of GDM between aboriginal and non-aboriginal women.

RESULTS — Information was obtained from 2,006 women, of whom 252 aboriginal and 1,360 non-aboriginal subjects had been tested for GDM. The overall rates of GDM were 3.5% for women in the general population and 11.5% for aboriginal women. For those living within the Saskatoon Health District, GDM rates were 3.7 and 6.4%, respectively. Multivariate analysis demonstrated that aboriginal ethnicity, most notably when combined with obesity, was an independent predictor for GDM. Pregravid BMI ≥27 kg/m^2 and maternal age ≥33 years were the most important risk factors for GDM in aboriginal women, whereas previous GDM, family history of diabetes, and maternal age ≥38 years were the strongest predictors for GDM in non-aboriginal women.

CONCLUSIONS — There may be fundamental differences in GDM between aboriginal and non-aboriginal people. Because GDM contributes to an increased risk for type 2 diabetes in aboriginal women and their offspring, the impact of prevention and optimal treatment of GDM on the type 2 diabetes epidemic in susceptible populations are important areas for further investigation.

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Canadian aboriginal people are experiencing an epidemic of type 2 diabetes and its complications (1–4). Although a combination of genetic and environmental factors associated with the loss of traditional lifestyles is generally acknowledged to have led to this crisis, only recently has attention been directed at the possible contribution of the intrauterine milieu.

North American aboriginal women have higher rates of gestational diabetes mellitus (GDM) than women in the general population (5–10). Women with GDM are more likely to develop type 2 diabetes (11–14), and their offspring may experience increased insulin resistance, increased rates of macrosomia (birth weight >4,000 g), childhood obesity, and a propensity for the early onset of type 2 diabetes (15,16). On northern Saskatchewan reserves, we found increased rates of GDM among aboriginal women, even though the community prevalence of type 2 diabetes was low (17). We also found a dramatic increase in rates of macrosomia from 12.6 to 19.2% between 1975 and 1988 in northern Saskatchewan, compared with a rise of only 10.2 to 12.8% in the south (18). Finally, we recently established that Saskatchewan aboriginal people with diabetes had higher rates of macrosomia than control populations and that this relationship strengthened from the mid to latter part of the last century (19). These findings raise the intriguing possibility that GDM may be a major initiating (as well as perpetuating) factor in the type 2 diabetes epidemic in susceptible populations.

A limitation to reports of GDM among Canadian aboriginal women has been the retrospective nature of the studies and the absence of optimal comparison populations. This result has made it difficult to establish whether or not the high rates of GDM observed in aboriginal women are due to an increased presence of established GDM risk factors or if aboriginal ethnicity constitutes a risk factor in itself. We now report the results of a prospective study that directly compared rates, risk factors, and outcomes of GDM between aboriginal and non-aboriginal women in a defined geographic area.

RESEARCH DESIGN AND METHODS

— This study included an Ethics Committee–approved prospective survey of all women admitted for childbirth to the Saskatoon Royal University Hospital (RUH) between 1 January and 7 July 1998. RUH is one of two tertiary care facilities in Saskatchewan; it is the Mater-

From the Departments of [1]Medicine, [2]Community Health and Epidemiology, and [3]Obstetrics and Gynecology, University of Saskatchewan, Saskatoon, Saskatchewan.

Address correspondence and reprint requests to Dr. Roland F. Dyck, Head, Department of Medicine, Royal University Hospital, 103 Hospital Dr., Saskatoon, Canada S7N 0W8. E-mail: dyck@sask.usask.ca.

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Abbreviations: GDM, gestational diabetes mellitus; OR, odds ratio; RUH, Royal University Hospital; SDH, Saskatoon District Health.

A table elsewhere in this issue shows conventional and Système International (SI) units and conversion factors for many substances.

nal/Child Health Center for Saskatoon District Health (SDH), which serves ~25% of Saskatchewan's 1 million residents. RUH also receives high-risk obstetrical referrals from surrounding health districts and Northern Saskatchewan.

Data were obtained from an admission questionnaire, the obstetrical unit logbook, RUH medical records databases, and hospital charts. The questionnaire asked consenting women to self-report on the following: history of type 1 diabetes, type 2 diabetes, GDM, previous infant with macrosomia (>4,000 g), and family members with diabetes; testing, diagnosis, and treatment of GDM in index pregnancy; height; pregravid weight; weight gain in index pregnancy; weight gain between previous and index pregnancy; ethnic origin; and degree of weekly physical activity throughout the index pregnancy (defined as "walking, swimming, or any type of exercise that kept you active for at least 20 minutes at a time"). Women who had prepregnancy diabetes or who delivered before 20 weeks' gestation were excluded from the study.

Pregravid BMI was calculated using self-reported height and either most recent weight before conception or earliest recorded first trimester weight. Ethnic origin was self-reported by most women or was otherwise obtained from the hospital chart. Ethnic categories were "aboriginal" (AB) (North American Indian, Metis, and Inuit) and "general population" (GP) (ethnicity other than aboriginal).

The obstetrical unit logbook recorded obstetrical history and major complications in index pregnancy, expected and actual date of delivery, pregnancy risk scores, type of delivery, and baby's birth weight, sex, and Apgar scores. RUH medical record information included basic demographics, resident health district, length of hospital stay, attending physician, admission to intensive care units, diagnoses and medical procedures according to International Classification of Diseases codes, type of anesthetic required for delivery, and pregnancy-related readmissions.

Gestational age was determined by ultrasound estimates or the woman's date of last normal menstrual period. An infant (singleton or twin) was determined to be large for gestational age or small for gestational age if birth weight in grams was ≥90th or ≤10th percentile, respectively, for corresponding male and female Saskatchewan newborns (20).

A woman was considered to have GDM if she met one of three criteria: first ($n = 46$), if two or more venous blood glucose values on the 100-g oral glucose tolerance test done during pregnancy met or exceeded the thresholds recommended by the National Diabetes Data Group (21); second ($n = 25$), if a 1-h 50-g oral glucose challenge test done during pregnancy resulted in a blood glucose value of ≥7.8 mmol/l, and there was a physician diagnosis of GDM; and, third ($n = 6$), if highly abnormal blood glucose values were observed in women who were not known to have prepregnancy diabetes and who required treatment with insulin ($n = 4$ of 6) or diet.

Data analyses were performed on Excel 5.0 and SPSS 9.1 software. Univariate analyses were used to determine rates and results of GDM screening. Differences between women tested or not tested for GDM were examined for possible selection bias in both GP and AB groups. Characteristics of GP and AB women tested for GDM were then examined for comparability. Finally, maternal and infant outcomes were compared within and between GP and AB groups.

Categorical variables were compared between groups with χ^2 tests (Yates corrected) or a Fisher's exact test. Comparisons of continuous variables were carried out using independent sample mean t tests. The Mann-Whitney U test was used to confirm analyses demonstrating unequal variances by Levene's test and for comparisons of quantitative (ordinal) variables. Level of significance was set at $P < 0.05$ (two-tailed).

The first step in our multivariate analysis was to select variables using bivariate analysis of these potential risk factors for GDM: age, ethnicity, pregravid BMI, weight gain in pregnancy, parity, family history of diabetes, previous GDM, previous macrosomic infant, previous stillbirth, and amount of physical activity during pregnancy. Variables with P values <0.25 in the bivariate analysis were then carried forth into a series of multivariate analyses using multiple logistic regression. Variables were retained in the model if they were significant at the 0.05 level in the presence of the remaining variables or were known to be biologically important. Continuous variables were rescaled at appropriate cut points. The adjusted odds ratio (OR) and 95% CIs of GDM between the two study groups were calculated. Once the main model was developed, plausible interactions between predictor variables were examined for significance using the likelihood ratio test (set at $P < 0.05$), followed by appropriate data stratification and subgroup analyses.

RESULTS — There were 2,197 women who gave birth to 2,236 infants. A total of 1,904 women (86.7%) completed the questionnaire, and information for 198 women (9.0%) was obtained from hospital charts; 95 (4.3%) declined participation. A total of 191 women were excluded because of nonconsent ($n = 95$), prepregnancy diabetes ($n = 24$), and unavailable ethnic designation ($n = 72$). Of the remaining 2,006 mothers, 394 were not tested for GDM, leaving a final sample of 1,612 women (and 1,635 infants); 252 were AB, and 1,360 were GP. Of the GP women, 95% were Caucasian. There were 1,212 (75.2%) SDH residents.

There were 83% of GP women and 68.5% of AB women screened for GDM ($P < 0.001$). Rates of GDM screening were similar for GP women who lived in and outside SDH (83.5 and 81.9%, respectively; NS); however, AB residents from SDH were tested more frequently than those who lived elsewhere (73.6 and 59.4%; $P < 0.01$). Compared with women who were not screened, women tested for GDM were marginally older (mean age differences: GP, 1 year [$P < 0.001$]; AB, 0.5 years [NS]), had slightly higher pregravid BMIs (mean differences: GP, 1 kg/m^2 [$P < 0.05$]; AB, 1.5 kg/m^2 [$P < 0.05$]), and lower parity (mean differences: GP, 0.1 [NS]; AB, 0.7 [$P < 0.01$]).

Overall, the period prevalence rates of GDM were 3.5% for GP women and 11.5% for AB women (OR 3.6, 95% CI 2.2–5.8). For SDH residents, GDM rates were 3.7 and 6.4%, respectively (OR 1.8, 95% CI 0.9–3.6). For residents outside SDH, GDM rates were 3.1 and 22.8%, respectively (OR 9.2, 95% CI 4.0–20.8).

Table 1 shows rates of GDM according to maternal characteristics. For both GP and AB groups, rates of GDM increased with older age, higher pregravid BMI, higher parity, and less frequent physical activity in pregnancy. For AB women, those relationships were stronger and followed a more progressive pattern. For both groups, rates of GDM were also

Table 1—*Comparability analysis and rate of GDM by maternal characteristics among all non-aboriginal (GP) and aboriginal (AB) women who were tested*

Characteristic	GP (n = 1,360)*		AB (n = 252)*		GP vs. AB
	n	% GDM	n	% GDM	P value
Age (years)					
<20	62	0	48	4.2	
20–24.9	270	3.0	83	4.8	
25–29.9	438	3.7	60	13.3	
30–34.9	404	3.0	42	19.0	
≥35	186	6.5	19	36.8	
Mean ± SD	28.5 ± 5.3		25.0 ± 6.0		<0.001
Pregravid BMI (kg/m^2)					
<20	195	3.6	40	2.5	
20–24.9	688	3.2	114	6.1	
25–26.9	132	1.5	18	0	
27–29.9	151	2.6	29	13.8	
≥30	194	6.7	51	33.3	
Mean ± SD	24.6 ± 5.5		25.3 ± 6.0		NS
Weight gain in index pregnancy (kg)					
<9	236	7.2	41	24.4	
9–18	858	2.7	130	10.0	
>18	266	3.0	80	7.5	
Mean ± SD	14.6 ± 5.6		16.0 ± 7.3		0.005
Previous pregnancies					
0	460	3.3	73	5.5	
1	457	2.8	61	4.9	
2	260	4.6	46	13.0	
≥3	183	4.4	72	22.2	
Mean ± SD	1.3 ± 1.4		1.9 ± 2.0		<0.001
Family history of diabetes in parent or sibling					
No	1102	2.7	137	7.3	
Yes	164	9.1	74	17.6	
%	13.0		35.1		<0.001
Hypertension in index pregnancy					
No	1243	3.2	223	9.4	
Yes	117	6.8	29	27.6	
%	8.6		11.5		NS
Physical activity in index pregnancy (times per week)†					
<1	237	5.5	24	12.5	
1–2	326	4.6	48	14.6	
3–4	404	2.0	81	9.9	
5–7	313	3.2	78	7.7	
% Activity ≥3 times per week	56.0		68.8		<0.001
Among multigravida					
Previous GDM					
No	745	1.6	133	10.5	
Yes	27	44.4	18	38.9	
%	3.5		11.9		<0.001
Previous macrosomic infant (>4,000 g)					
No	574	2.8	86	7.0	
Yes	203	3.9	73	23.3	
%	26.1		45.9		<0.001
Previous stillbirth					
No	728	3.3	151	13.2	
Yes	18	0	6	50.0	
%	2.4		3.8		NS
Number of risk factors for GDM‡					
0	476	2.3	87	3.4	
1	530	3.0	75	4.0	
2	258	3.5	53	17.0	
3	84	8.3	20	20.0	
≥4	12	41.7	17	58.8	
Mean ± SD	1.3 ± 1.0		1.6 ± 1.2		0.005

*Information not complete for all variables; †defined as any kind of continuous aerobic activity or exercise for at least 20 min; ‡risk factor for GDM defined as age ≥30 years, pregravid BMI ≥27 kg/m^2, parent and/or sibling family history of diabetes, previous GDM, previous macrosomic infant (>4,000 g), or previous stillbirth.

increased in those with previous GDM, previous macrosomic infant, family history of diabetes, and hypertension in index pregnancy. Previous stillbirth was associated with increased GDM for AB women only. An inverse relationship was observed between weight gain in pregnancy and rates of GDM for both groups of women; however, women with higher pregravid BMI tended to gain less weight in pregnancy, and those with lower BMI tended to gain more weight (Pearson's correlation coefficient -0.28, $P < 0.01$, for GP women; Pearson's correlation coefficient -0.30, $P < 0.01$, for AB women).

Increasing numbers of risk factors correlated with an increased risk for GDM. This relationship was stronger for AB women, who showed a marked rise in GDM rates at two or more risk factors, compared with a less pronounced gradient for GP women at three or more risk factors. Comparable results were obtained when the analyses in Table 1 were repeated using only SDH residents.

The independent predictors of GDM were previous GDM, pregravid BMI, family history of diabetes, age, and ethnicity (Table 2). Aboriginal women had twice the risk of GDM compared with GP women after adjustment for all other variables. Although age lost significance once rescaled, it was retained in the model because of its biological importance.

Significant interactions were observed in the main model between ethnicity × BMI and ethnicity × previous GDM. After stratification by ethnicity, the strongest risk factors for GDM among GP women were previous GDM and familial diabetes (Table 2). Pregravid BMI was the most important risk factor for GDM among AB women. These observations persisted when each ethnic group was further stratified by SDH residency. With all other variables unaltered, age became a significant predictor for GDM at ≥33 years of age for AB women and ≥38 years of age for GP women. After stratification by pregravid BMI, ethnicity was not a significant predictor of GDM for normal-weight women. In contrast, overweight aboriginal women (BMI ≥27 kg/m^2) had a 4.7-fold risk of GDM compared with overweight GP women.

Table 3 summarizes the delivery and infant outcomes. Compared with their non-GDM counterparts, AB women with GDM had significantly higher rates of pregnancy-related hypertension ($P < 0.01$), higher rates of previous C-section ($P < 0.05$) and current C-section (NS), higher rates of spinal anesthetic ($P < 0.05$), and lower rates of epidural anesthetic during delivery ($P < 0.05$). The differences in anesthetics were due to a preferred use of spinal anesthetics in women undergoing C-section. For GP women with GDM, rates of ≥2° laceration and advanced trauma to perineum/vulva were significantly higher ($P < 0.05$), and rates of episiotomy were lower ($P < 0.05$) than in their non-GDM counterparts. Rates of medical induction, forceps/vacuum/breech, meconium stain, shoulder dystocia, and postpartum hemorrhage did not differ significantly within GP or AB groups. There were also no significant differences with respect to length of stay in hospital, postpartum complications requiring admission, or maternal mortality (there was one maternal death of a GP woman who did not have GDM).

Mean birth weights did not significantly differ within or between populations when mothers did not have GDM. However, AB infants from GDM pregnancies were significantly heavier than their GP counterparts ($P < 0.05$). Also, infants born to AB women with GDM were 2.4 times more likely to be macrosomic (95% CI 1.1–5.6) than their non-GDM counterparts and six times more likely to have birth weight >4,000 g (95% CI 1.7–21.7) than infants born to GP mothers with GDM (not shown).

Compared with their non-GDM counterparts, infants born to GP women with GDM were 2.5 times more likely to have congenital anomalies (95% CI 1.1–5.7). These included an atrial and a ventricular septal defect, two anomalies of the genital organs, one deformity of the skull and face, one dislocation of the hip, and a birthmark.

There were no differences in Apgar scores, rates of birth trauma, or hyperbilirubinemia between the groups. There were also no differences in length of stay in hospital, length of stay in neonatal intensive care unit, neonatal respiratory distress syndrome, or neonatal death (the three infants who died had GP mothers without GDM). Although we detected a higher rate of respiratory conditions other than respiratory distress syndrome among infants of AB women with GDM, these were associated with higher rates of C-section.

Table 2—*Multivariate analysis on GDM risk factors for all women, stratified by ethnicity and BMI*

Variable	All women (n = 1,466)	Stratified by ethnicity		Stratified by BMI	
		GP only (n = 1,263)	AB only (n = 203)	BMI <27 kg/m^2 (n = 1,084)	BMI ≥27 kg/m^2 (n = 382)
Aboriginal ethnicity	1.98 (1.06–3.69)*	—	—	0.65 (0.21, 2.03)	4.66 (1.98, 10.96)†
Pregravid BMI ≥27 (vs. <27)	2.30 (1.35–3.93)‡	1.41 (0.71, 2.78)	8.56 (2.66, 27.55)†	—	—
History of GDM§	12.86 (6.13–26.95)†	26.83 (11.10, 64.80)†	2.67 (0.67, 10.56)	13.56 (4.95, 37.15)†	12.76 (3.95, 41.19)†
Family history of diabetes‖	2.70 (1.53–4.76)†	2.85 (1.41, 5.79)‡	2.13 (0.78, 5.77)	2.77 (1.26, 6.09)‡	2.46 (1.05, 5.78)*
Age ≥30 years (vs. <30 years)	1.27 (0.74–2.18)	0.91 (0.48, 1.74)	2.14 (0.78, 5.88)	1.12 (0.54, 2.32)	1.28 (0.56, 2.92)
Age, further analysis¶	—	3.47 (1.17, 10.25)* at ≥38 years	3.31 (1.08, 10.16)* at ≥33 years	—	—

Data are ORs (95% CI) for presence of GDM, adjusted for all other variables. *$P < 0.05$; †$P < 0.001$; ‡$P < 0.01$; § for primagravida, previous GDM = No; ‖includes only parent and/or sibling; ¶point at which age becomes significant, adjusted for same variables as in above model.

Table 3—*Delivery and infant outcomes in pregnancies with and without GDM according to ethnicity*

Variable	Pregnancies in GP			Pregnancies in AB population		
	With GDM	Without GDM	OR (95% CI)	With GDM	Without GDM	OR (95% CI)
Delivery outcomes						
n	48	1,312	—	29	223	—
Pregnancy-related hypertension	17 (8)	8 (109)	2.2 (1.0–4.8)	28 (8)	9.4 (21)	3.7 (1.5–9.3)*
Epidural anesthetic versus all other	64.6 (31)	58.6 (769)	1.3 (0.7–2.4)	31 (9)	51.1 (114)	0.4 (0.2–0.99)†
Spinal anesthetic Versus all other	10.4 (5)	11.1 (145)	1.0 (0.4–2.4)	27.6 (8)	10.3 (23)	3.3 (1.3–8.3)†
Previous C-section	4.2 (2)	12.0 (158)	0.3 (0.1–1.3)	24.1 (7)	9.9 (22)	2.9 (1.1–7.6)†
C-section versus all other delivery	18.8 (9)	18.1 (237)	1.1 (0.5–2.2)	31.0 (9)	15.7 (35)	2.4 (1.0–5.7)
≥2° laceration and advanced trauma to perineum/vulva	48.3 (21)	28.7 (377)	1.9 (1.1–3.5)†	24.1 (7)	19.7 (44)	1.3 (0.5–3.2)
Shoulder dystocia	2.1 (1)	2.4 (32)	0.8 (0.1–6.4)	0	0.9 (2)	
Mean ± SD total risk score (equals ante + intrapartum)‡	4.6 ± 2.2	3.0 ± 2.1	$P < .001$	5.0 ± 2.4	3.2 ± 2.5	$P < 0.001$
Infant outcomes						
n	50	1,332	—	29	224	—
Mean ± SD gestational age (weeks)	38.8 ± 2.0	39.5 ± 1.7	*t* test*	38.9 ± 2.6	39.4 ± 1.9	*t* test, NS
Mean ± SD birth weight (g)	3,333 ± 568	3,430 ± 531	*t* test, NS	3,668 ± 795	3,496 ± 635	*t* test, NS
Large gestational age >90th percentile	14 (7)	10.2 (136)	1.4 (0.6–3.3)	27.6 (8)	17.9 (40)	1.8 (0.7–4.2)
Small gestational age <10th percentile	8 (4)	8 (107)	1.0 (0.4–2.8)	3.4 (1)	10.3 (23)	0.3 (0.04–2.4)
High birth weight (>4,000 g)	8 (4)	12.5 (167)	0.6 (0.2–1.7)	34.5 (10)	17.9 (40)	2.4 (1.1–5.6)†
Low birth weight (<2,500 g)	4 (2)	5.2 (69)	0.8 (0.2–3.2)	3.4 (1)	7.6 (17)	0.4 (0.1–3.4)
Disorders related to short gestation and low birth weight	14 (7)	6.2 (82)	2.5 (1.1–5.7)†	6.9 (2)	7.6 (17)	0.9 (0.2–4.1)
Hypoglycemia	2 (1)	0.8 (11)	2.5 (0.3–19.4)	6.9 (2)	0.4 (1)	16.5 (1.5–188)†
Any congenital anomalies	14 (7)	6.2 (82)	2.5 (1.1–5.7)†	6.9 (2)	8.9 (20)	0.8 (0.2–3.4)

Data are % (*n*) unless otherwise indicated. *$P < 0.01$; †$P < 0.05$; ‡interpretation of total score: 0–2, low risk; 3–6, moderate risk; >6, high risk.

CONCLUSIONS — Although several recent articles have reported increased rates of GDM in Canadian aboriginal women, this is the first Canadian study that has shown higher rates among aboriginal (6.4%) compared with non-aboriginal (3.7%) women in a prospective study that included all births within a defined geographical area. These findings would be more robust had a universal GDM screening program been in place, but we did not detect major differences in GDM risk factors between those women tested and not tested. We also observed a higher rate of GDM among aboriginal women from outside compared with within the SDH (22.8 vs. 6.4%); however, we were unable to determine whether this was because of a larger proportion of women with GDM among those referred because of high-risk pregnancies or whether it confirms a higher reported rate of GDM in aboriginal women from northern Saskatchewan (17). Both possibilities likely contribute to this observation; if an increased concentration of GDM among high-risk pregnancy referrals were the only reason for this finding, we also would have expected higher rates of GDM in non-aboriginal women living outside SDH (3.1%) compared with those living inside SDH (3.7%).

Perhaps the most important finding from this study was that aboriginal ethnicity is an independent predictor of GDM, even in the presence of other known GDM risk factors. The interaction between ethnicity and BMI was key in the complexity of this relationship, such that overweight aboriginal women had a dramatically higher risk of GDM compared with overweight GP women; however, ethnicity was not a determinant of GDM among normal-weight women. This interaction effect is consistent with a recent report by Rodrigues et al. (22). We also report differences between aboriginal and non-aboriginal women in the importance of other known GDM risk factors. For non-aboriginal women, previous GDM and family history of diabetes were most predictive. Increasing age was an important determinant of GDM in both ethnic groups but became significant at a younger age among aboriginal women.

Despite current recommendations for universal screening (23), aboriginal women in our study population were screened less frequently than non-aboriginal women. The reasons for this observation are not clear; however, there may be a lack of awareness among some health care professionals and aboriginal women with respect to the importance of GDM screening, women may not wish to be tested, and/or some aboriginal women may present to the health care system too late in their pregnancies for timely screening.

What were the immediate implications of GDM for mothers and their newborns? Although our study may not have had the power to detect all possible differences between aboriginal and non-aboriginal women and their infants, we did detect distinctive features between the two groups that did not appear to be due to referral bias. Aboriginal women with GDM were more likely to have hypertension and to have experienced a previous and current C-section. Non-aboriginal women with GDM had higher rates of significant laceration and trauma from childbirth than those who did not have GDM.

Aboriginal newborns from GDM pregnancies had the highest mean birth weight and were more likely to be macrosomic. Infants from GDM pregnancies were often hypoglycemic, although this was only significant for aboriginal newborns. An unexpected finding was that non-aboriginal newborns of GDM mothers were more than twice as likely to have congenital anomalies than those whose mothers did not have GDM. Although there was no pattern to these anomalies, this finding warrants further investigation.

Our findings suggest that there may be fundamental differences in GDM between aboriginal and non-aboriginal people. Not only have we shown that aboriginal ethnicity is an independent GDM risk factor contributing to higher GDM rates, but we have also shown differences in immediate GDM outcomes between the two populations. There may also be differences in the long-term implications of GDM. Both aboriginal women who have had GDM and the offspring of women with diabetes during pregnancy experience an increased risk of type 2 diabetes (16). In a case-control study, we recently showed that Saskatchewan aboriginal people with diabetes were born with higher rates of macrosomia than aboriginal people without diabetes (19). The latter is probably explained in part by high rates of GDM in the mothers of diabetic case subjects. We have proposed a new paradigm: the "hefty fetal phenotype" hypothesis (24). It is consistent with the "thrifty genotype" hypothesis (25,26) and, like the "thrifty phenotype" (27) and "surviving small baby genotype" (28) hypotheses, provides an explanation for the prenatal origin of type 2 diabetes. The "hefty fetal phenotype" hypothesis attempts to explain how an ancient survival mechanism, which may have evolved to produce well-nourished infants, has become a modern liability leading to increased rates of GDM and an epidemic of type 2 diabetes in susceptible populations.

These and related findings perhaps raise more questions than they answer. Whereas we have shown some immediate adverse consequences of GDM, the possible long-term impact of GDM for aboriginal people is at the same time more troubling, as well as a reason for hope. If GDM is a significant initiating and perpetuating factor in the type 2 diabetes epidemic in aboriginal populations, will the prevention and optimal treatment of GDM lead to lower rates of type 2 diabetes in successive generations? Pregnancy (and the pregravid period) represent optimal times for the initiation of intervention programs for reasons we have outlined (29). This and other studies (22) suggest that targeting obesity in pregravid women may be particularly beneficial. Finally, we are interested in the possibility that exercise during pregnancy may prevent GDM in those women who are at highest risk (30). It is noteworthy that, in this study, women who were most physically active had the lowest prevalence of GDM.

Hopefully, the results of this study, as well as the questions it raises, will provoke further research into the role of GDM in the type 2 diabetes epidemic among aboriginal people and into the implications for its prevention and treatment. This and other recent Canadian reports demonstrate the importance of promoting GDM screening programs and healthy pregnancies in this high-risk population.

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