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Treatment of Gestational Diabetes Mellitus and Maternal Risk of Diabetes After Pregnancy

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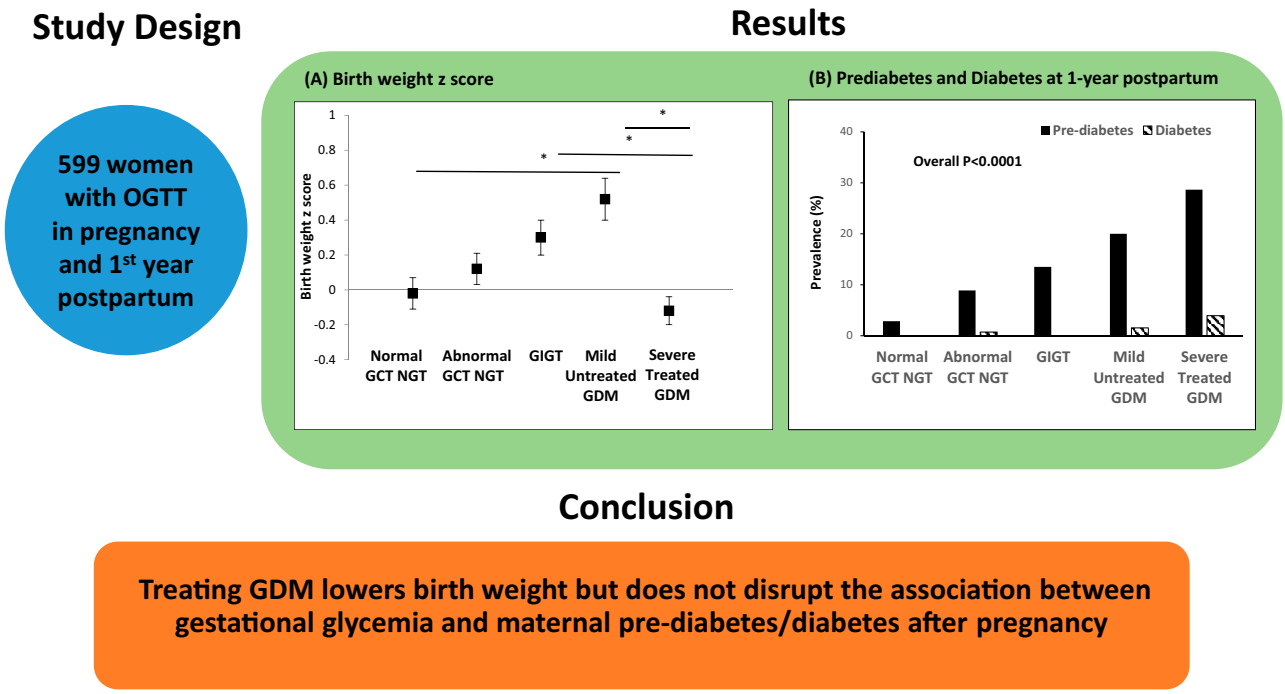
Summary of the study design, results, and conclusions. *A:* Birth weight z scores. *B:* Prediabetes/diabetes at 1 year postpartum. **P* < 0.05 by pairwise comparison. GCT, glucose challenge test; GDM, gestational diabetes mellitus; GIGT, gestational impaired glucose tolerance; NGT, normal glucose tolerance; OGTT, oral glucose tolerance test.



ARTICLE HIGHLIGHTS

- Whether treating gestational diabetes mellitus (GDM) affects the association between gestational glycemia and maternal risk of prediabetes/diabetes after pregnancy is unknown.
- We compared postpartum glucose tolerance between women with severe treated GDM and those with mild, untreated GDM.
- Although treatment of severe GDM yielded lower birth weight than mild, untreated GDM, the risk of maternal postpartum prediabetes/diabetes remained higher in women with severe treated GDM.
- Postpartum metabolic surveillance is essential in women with GDM, irrespective of the effect of their antenatal treatment on birth weight at delivery.

Treatment of Gestational Diabetes Mellitus and Maternal Risk of Diabetes After Pregnancy

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BRIEF REPORT

OBJECTIVE

To compare postpartum glucose tolerance between women treated for gestational diabetes mellitus (GDM) and those not treated.

RESEARCH DESIGN AND METHODS

Metabolic testing was performed at 3 and 12 months postpartum in 599 women comprising the following gestational glucose tolerance groups: *1*) normal glucose challenge test (GCT) and oral glucose tolerance test (OGTT) during pregnancy, *2*) abnormal GCT with normal OGTT, *3*) gestational impaired glucose tolerance, *4*) mild untreated GDM, and *5*) severe treated GDM.

RESULTS

Birth weight progressively increased across groups 1–4 before falling steeply in treated GDM ($P < 0.0001$). In contrast, at 3 and 12 months, insulin sensitivity and β-cell function progressively decreased across the five groups, mirrored by rising fasting and 2-h glucose (all $P < 0.0001$). Accordingly, prevalence of prediabetes/diabetes at 12 months increased in a stepwise manner across groups 1–5 (2.8%, 9.6%, 13.5%, 21.5%, and 32.6%, respectively; $P < 0.0001$).

CONCLUSIONS

Treating GDM lowers birth weight but does not disrupt the association between gestational glycemia and maternal prediabetes/diabetes after pregnancy.

Continuous associations exist between maternal glycemia in pregnancy and both neonatal birth weight and future maternal risk of prediabetes/diabetes (1–3). Antenatal treatment of gestational diabetes mellitus (GDM) can lower birth weight and disrupt the former association (4); however, whether this treatment affects the latter association is unclear (5). Thus, we compared postpartum glucose tolerance between women treated for GDM and those not treated.

RESEARCH DESIGN AND METHODS

The study protocol has been previously described in detail (6). In brief, 599 women underwent a 50-g glucose challenge test (GCT) and 3-h 100-g oral glucose tolerance test (OGTT) during pregnancy, followed by a 2-h 75-g OGTT at both 3 and 12 months postpartum. Per institutional practice, pregnant women who met National Diabetes Data Group (NDDG) criteria for GDM (7) (Supplementary Table 1) were treated with antenatal lifestyle modification (diet and physical activity), targeting a fasting glucose <5.3 mmol/L and 2-h postprandial glucose <6.7 mmol/L on self-monitoring. Women

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exceeding these targets >50% of the time in 1 week were treated with insulin therapy, with doses titrated to the targets.

Since NDDG thresholds for diagnosing GDM are higher than those of the American Diabetes Association (ADA) (8), ADA criteria can define two groups of women within this study population: those meeting both ADA and NDDG criteria (who received treatment for GDM) and those meeting only ADA criteria (who were not treated). Accordingly, by applying ADA criteria to the antepartum OGTT (Supplementary Table 1), we stratified the population into the following groups: *1*) severe treated GDM, which included women meeting ADA and NDDG criteria for GDM; *2*) mild untreated GDM, which included women meeting only ADA criteria; *3*) gestational impaired glucose tolerance (GIGT), which included women with only one glucose value meeting ADA criteria; *4*) abnormal GCT normal glucose tolerance (NGT), which included women with an abnormal GCT followed by NGT on OGTT (ADA criteria); and *5*) normal GCT NGT, which included women with normal GCT and OGTT (ADA criteria).

Outcomes and Statistical Analyses

Maternal outcomes of prediabetes and diabetes on the OGTT at 3 and 12 months postpartum were defined according to Diabetes Canada clinical practice guidelines (9) (Supplementary Table 1). Women were notified if the OGTT showed prediabetes/diabetes. On each OGTT, insulin sensitivity/resistance was assessed by Matsuda index and HOMA of insulin resistance (HOMA-IR), and β-cell function was assessed by insulin secretion-sensitivity index 2 (ISSI-2) and insulinogenic index/HOMA-IR (10–12). Neonatal outcomes were birth weight *z* score and large-for-gestational-age (LGA) delivery based on Canadian birth weight centiles for sex and gestational age (13).

Statistical analyses were performed using SAS 9.4 software (SAS Institute, Cary, NC). Birth weight *z* score and LGA rate at delivery and maternal prediabetes/diabetes at 3 and 12 months postpartum were compared across the five groups (Fig. 1). Maternal metabolic function at 3 and 12 months postpartum was compared across the groups by multiple linear regression, adjusted for age, ethnicity, family history of diabetes, current BMI, and duration of breastfeeding (Fig. 2). Multiple logistic regression analyses were performed to determine whether study groups were independently associated with prediabetes/diabetes at 3 and 12 months postpartum, after adjustment for the same covariates (Fig. 3).

RESULTS

Table 1 shows the characteristics of the five groups defined by gestational glucose tolerance status and treatment of GDM as follows: *1*) normal GCT NGT, *2*) abnormal GCT NGT, *3*) GIGT, *4*) mild untreated GDM, and *5*) severe treated GDM. As anticipated, these groups exhibited a progressively more severe metabolic phenotype by OGTT in pregnancy, characterized by rising glycemia, declining insulin sensitivity, and worsening β-cell function (all $P < 0.0001$). At delivery, infants of women with severe treated GDM had the lowest birth weight (mean ± SD 3,235 ± 481 g) and length of gestation (38.2 ± 1.9 weeks). Birth weight *z* score

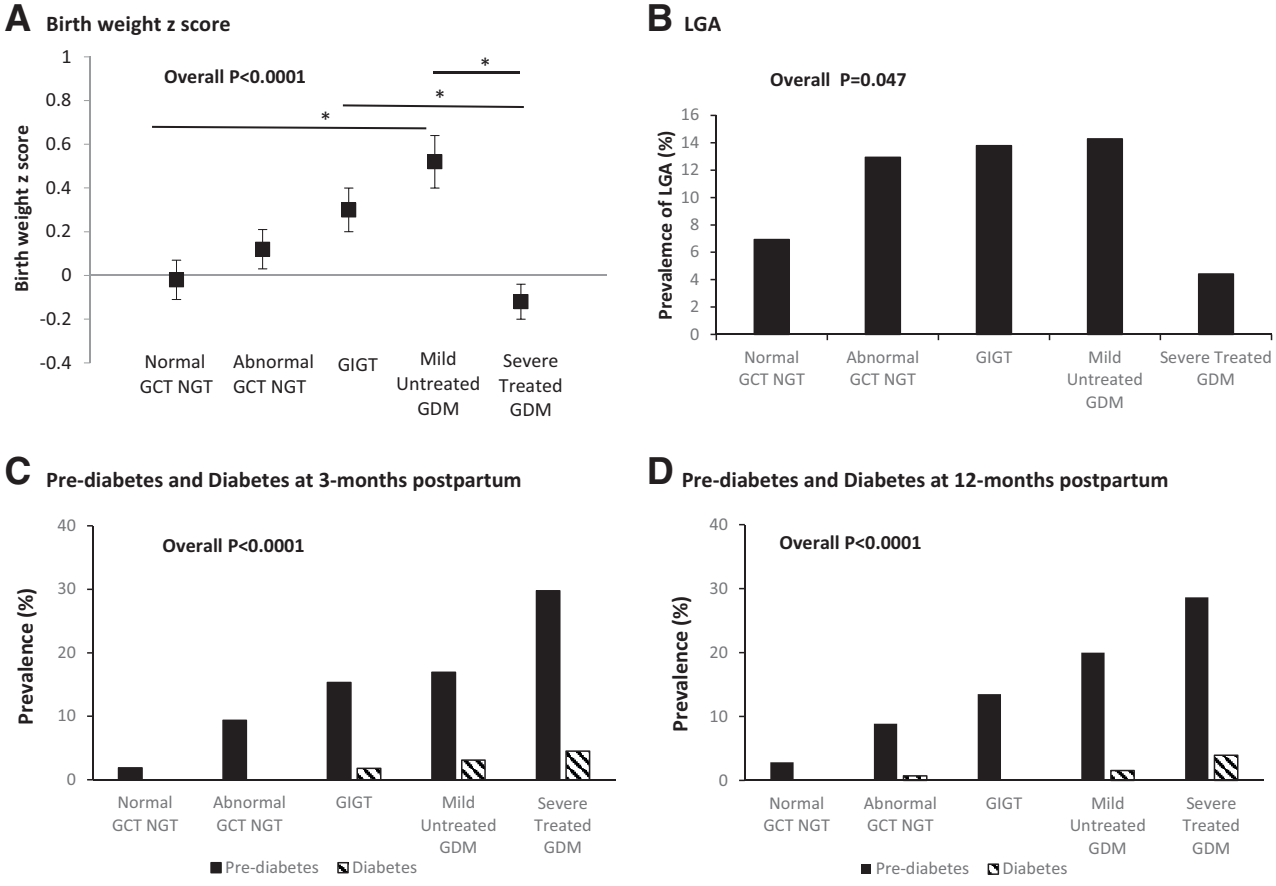


Figure 1—Gestational glucose tolerance status/treatment and maternal/neonatal outcomes. *A*: Birth weight *z* score. *B*: LGA. *C*: Maternal prediabetes and diabetes at 3 months postpartum. *D*: Maternal prediabetes and diabetes at 12 months postpartum. *$P < 0.05$ by pairwise comparison.

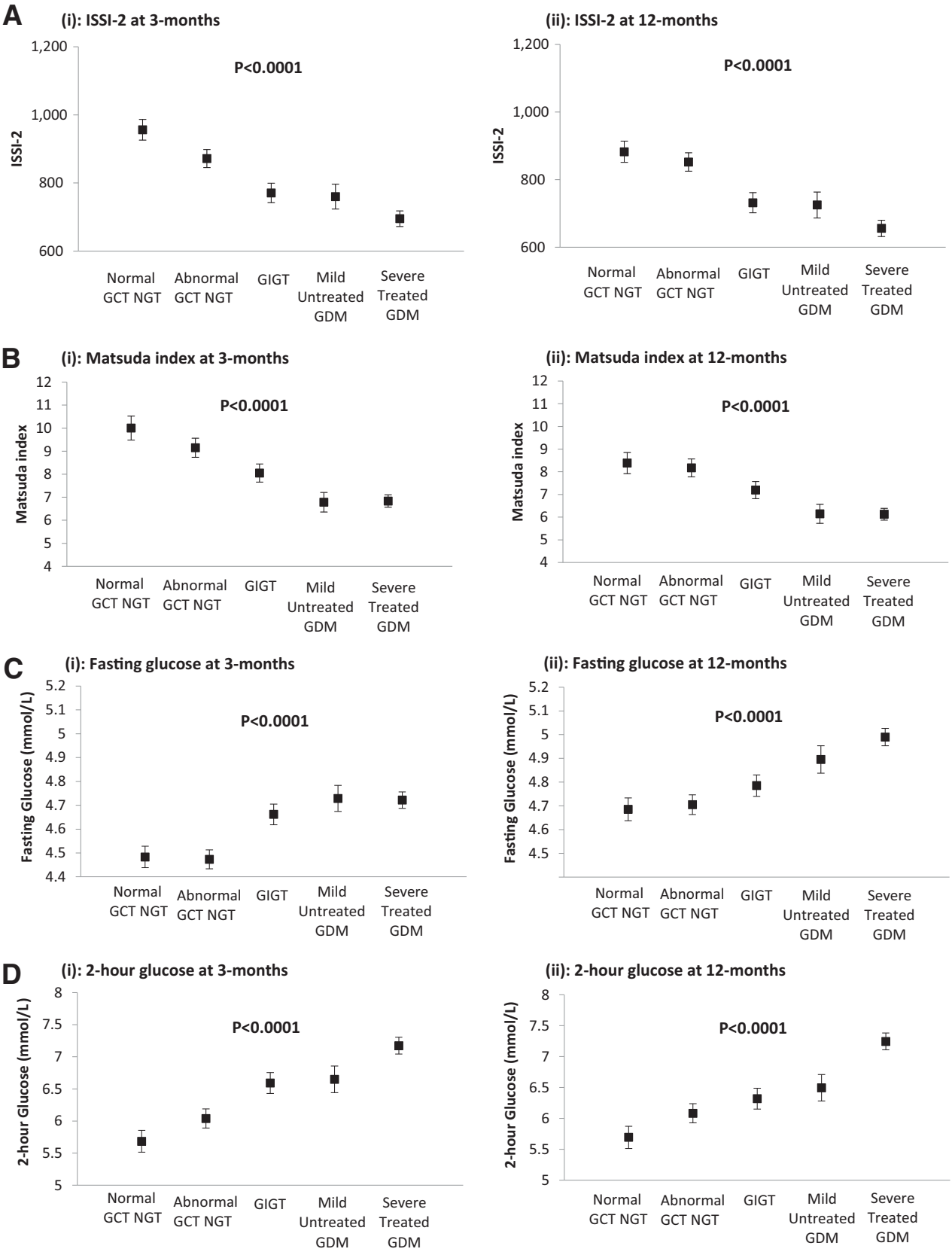
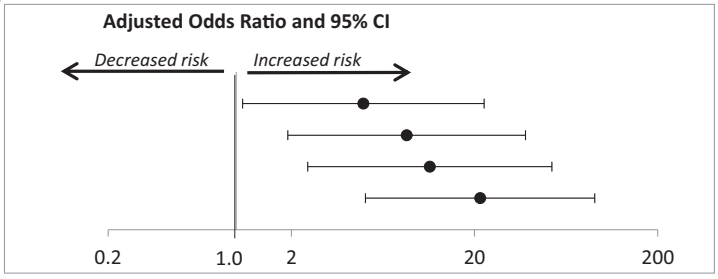


Figure 2—Maternal metabolic function at 3 and 12 months postpartum by study group. *A*: ISSI-2. *B*: Matsuda index. *C*: Fasting glucose. and *D*: Two-hour glucose on OGTT. Data are mean ± SE. All data are adjusted for age, ethnicity, family history of diabetes, current BMI, and duration of breastfeeding. Adjusted means and SEs were obtained using the least squares method for each group at 3 and 12 months postpartum.

A **Risk of pre-diabetes/diabetes at 3-months postpartum**

	Adjusted OR (95% CI)
Normal GCT NGT	*Reference*
Abnormal GCT NGT	4.9 (1.1 to 22.6)
GIGT	8.5 (1.9 to 38.0)
Mild Untreated GDM	11.4 (2.5 to 52.9)
Severe Treated GDM	21.5 (5.1 to 90.7)



B **Risk of pre-diabetes/diabetes at 12-months postpartum**

	Adjusted OR (95% CI)
Normal GCT NGT	*Reference*
Abnormal GCT NGT	3.2 (0.9 to 12.0)
GIGT	3.7 (1.0 to 13.8)
Mild Untreated GDM	8.3 (2.2 to 31.2)
Severe Treated GDM	12.9 (3.8 to 43.8)

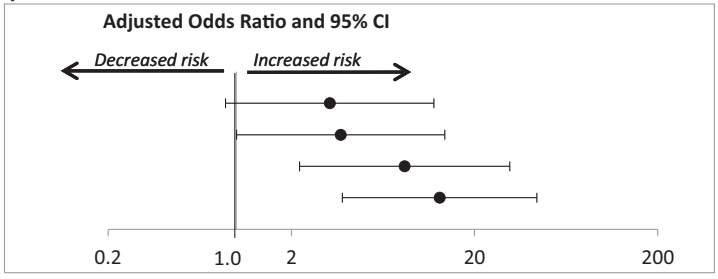


Figure 3—Adjusted odds ratio (OR) for each gestational glucose tolerance group in predicting prediabetes/diabetes at 3 and 12 months postpartum. *A*: Three months postpartum. *B*: Twelve months postpartum. Each OR is adjusted for maternal age, ethnicity, family history of diabetes, BMI, and duration of breastfeeding. Reference group is normal GCT NGT.

progressively increased from normal GCT NGT to abnormal GCT NGT to GIGT to mild untreated GDM before falling precipitously in women with severe treated GDM ($P < 0.0001$) (Fig. 1*A*). LGA rates showed the same pattern, rising from 6.9% to 12.9% to 13.8% to 14.3% before falling to 4.4% in women treated for GDM ($P = 0.047$) (Fig. 1*B*). Treatment of GDM thus disrupts the continuous association between maternal glycemia and infant birth weight.

In contrast, the association between gestational glycemia and maternal risk of prediabetes/diabetes showed no such disruption. Indeed, at both 3 and 12 months postpartum, the prevalence of prediabetes and diabetes progressively increased across the five groups (both $P < 0.0001$) (Fig. 1*C* and *D*). Thus, antenatal treatment of GDM did not appear to influence maternal risk of prediabetes/diabetes.

At both 3 and 12 months postpartum, mean adjusted ISSI-2 progressively decreased from normal GCT NGT to abnormal GCT NGT to GIGT to mild untreated GDM to severe treated GDM (both $P < 0.0001$) (Fig. 2*A*). The secondary measure of β-cell function, insulinogenic index/HOMA-IR, showed the same pattern (both $P < 0.0001$) (data not shown). Mean adjusted insulin sensitivity (Matsuda index) also displayed this pattern (both $P < 0.0001$) (Fig. 2*B*), coupled with concordant findings for HOMA-IR (data not shown). These patterns of declining β-cell function and insulin sensitivity across the five groups were mirrored by a rising mean adjusted fasting glucose and 2-h glucose at both 3 and 12 months postpartum (all $P < 0.0001$) (Fig. 2*C* and *D*). Furthermore, adjusted odds ratios for prediabetes/diabetes at 3 and 12 months postpartum progressively increased across the groups from normal GCT NGT (reference) to abnormal GCT NGT to GIGT to mild untreated GDM to severe treated GDM ($P < 0.0001$) (Fig. 3*A* and *B*).

Finally, we sought to determine whether insulin treatment of GDM affected postpartum metabolic function compared with management with lifestyle modification alone (Supplementary Table 2). At the antepartum OGTT, women who subsequently required insulin therapy ($n = 53$) had lower insulin sensitivity, poorer β-cell function, and greater glycemia than those in whom GDM was managed with lifestyle alone ($n = 125$). Though birth weight did not differ between these groups, the differences in insulin sensitivity, β-cell function, and glycemia persisted at both 3 and 12 months postpartum (Supplementary Table 2), suggesting no enduring metabolic effect of antenatal insulin therapy. This interpretation was further supported when comparing all six groups (Supplementary Table 3), as well as baseline-adjusted changes between 3 and 12 months (Supplementary Fig. 1).

CONCLUSIONS

The question of whether antenatal treatment of GDM affects postpartum risk of diabetes would be best addressed with a clinical trial in which women with GDM are randomly assigned to either treatment or no treatment and then undergo postpartum metabolic testing. However, such a trial would be difficult to perform now that antenatal glucose-lowering therapy (lifestyle or pharmacologic) is standard management for GDM (4). Thus, in the absence of such a trial, we postulated that relevant insight might be obtained from observational data by determining whether treating GDM disrupts the continuous association of maternal glycemia with postpartum prediabetes/diabetes (as it does for the association of maternal glycemia with birth weight).

In this study, the reduction in birth weight with treatment of GDM appeared greater than that observed in previous trials (14,15), possibly reflecting the greater severity of GDM herein and subsequent treatment to stringent glycemic targets, which may have collectively yielded a greater glycemic contrast between treated

Table 1—Characteristics of study population, stratified according to gestational glucose tolerance status and treatment of GDM

	Normal GCT NGT (*n* = 106)	Abnormal GCT NGT (*n* = 139)	GIGT (*n* = 111)	Mild untreated GDM (*n* = 65)	Severe treated GDM (*n* = 178)	*P*
At OGTT during pregnancy						
Age (years)	34.2 ± 4.4	33.9 ± 3.9	34.9 ± 3.9	35.3 ± 4.2	34.9 ± 4.4	0.09
Ethnicity						0.28
Caucasian	81 (76.4)	92 (66.2)	74 (66.7)	46 (70.8)	112 (62.9)	
Asian	7 (6.6)	18 (13.0)	16 (14.4)	10 (15.4)	32 (18.0)	
Other	18 (17.0)	29 (20.8)	21 (18.9)	9 (13.8)	34 (19.1)	
Family history of diabetes	50 (47.2)	80 (58.0)	76 (68.5)	33 (50.8)	120 (67.4)	0.002
Prepregnancy BMI (kg/m^2)	23.3 (21.3–26.7)	22.7 (21.0–25.6)	24.8 (22.2–27.7)	24.1 (21.9–27.3)	24.7 (21.6–30.0)	0.003
Smoking status						0.58
Remote	37 (34.9)	39 (38.1)	28 (25.2)	17 (26.2)	42 (23.7)	
Current	1 (0.9)	2 (1.4)	3 (2.7)	2 (3.1)	2 (1.1)	
Never	68 (64.2)	98 (70.5)	80 (72.1)	46 (70.8)	133 (75.1)	
Insulin sensitivity and resistance						
Matsuda index	6.0 (4.2–8.6)	5.5 (3.8–7.6)	3.9 (2.7–5.5)	3.2 (2.7–4.0)	3.0 (2.1–4.4)	<0.0001
HOMA-IR	1.5 (0.9–2.0)	1.3 (1.0–2.1)	1.8 (1.3–2.9)	2.3 (1.6–2.9)	2.3 (1.4–3.7)	<0.0001
β-Cell function						
ISSI-2	930 ± 267	910 ± 246	679 ± 162	616 ± 155	528 ± 155	<0.0001
IGI/HOMA-IR	13.7 (9.7–20.2)	13.9 (10.0–21.1)	8.9 (6.8–13.0)	6.5 (4.3–10.0)	5.8 (3.3–8.8)	<0.0001
Glucose on OGTT (mmol/L)						
Fasting	4.2 ± 0.4	4.4 ± 0.3	4.6 ± 0.5	4.8 ± 0.5	4.9 ± 0.7	<0.0001
1 h	7.5 ± 1.3	8.1 ± 1.2	9.2 ± 1.3	10.5 ± 1.0	11.3 ± 1.5	<0.0001
2 h	6.6 ± 1.0	7.1 ± 1.0	8.1 ± 1.1	8.7 ± 1.0	10.4 ± 1.4	<0.0001
3 h	5.7 ± 1.2	5.9 ± 1.2	6.9 ± 1.3	6.9 ± 1.5	8.2 ± 1.7	<0.0001
At delivery						
Length of gestation (weeks)	39.0 ± 1.6	38.8 ± 1.5	39.1 ± 1.4	38.7 ± 1.6	38.2 ± 1.9	0.0007
Male infant	46 (43.4)	72 (52.9)	48 (44.9)	40 (64.5)	83 (50.0)	0.07
Birth weight (g)	3,373 ± 511	3,402 ± 487	3,482 ± 457	3,546 ± 624	3,235 ± 481	<0.0001
At 3 months postpartum						
Breastfeeding (months)	3 (3–3)	3 (3–3)	3 (3–4)	3 (3–4)	3 (2–3)	0.02
BMI (kg/m^2)	26.1 ± 4.6	25.8 ± 5.2	27.3 ± 4.9	27.1 ± 4.4	27.3 ± 6.0	0.06
At 12 months postpartum						
Breastfeeding (months)	11 (6–12)	9 (6–12)	11 (6–12)	10.5 (4.5–12)	9 (3–12)	0.03
BMI (kg/m^2)	24.9 ± 4.4	25.0 ± 5.2	26.5 ± 5.3	26.2 ± 4.9	26.8 ± 6.8	0.02

Data are mean ± SD (if normal distribution), median (interquartile range) (if skewed distribution), or *n* (%). IGI, insulinogenic index.

and untreated GDM than in the earlier trials. However, there were no observed effects on β-cell function, insulin sensitivity, or glucose tolerance at either 3 or 12 months postpartum. Indeed, these findings are not surprising when one considers that although the spectrum of gestational glycemia identifies gradients of risk for both neonatal macrosomia and future maternal diabetes (1–4), it does so through very different mechanisms. Specifically, macrosomic risk is driven by the anabolic effects of hyperglycemia-induced fetal insulin secretion that can be modified by lowering maternal glycemia (through lifestyle or pharmacotherapy). In contrast, future maternal risk of diabetes arises over time because of progressive worsening of β-cell compensation for insulin resistance (16,17) that exists before, during, and after pregnancies complicated by GDM (18–20). Thus, to reduce future risk of prediabetes/diabetes in these women, antenatal treatment of GDM would likely need to modify postpartum β-cell function or its deterioration over time (neither of which was observed). In the same way, studies of preconception/early pregnancy intervention to prevent GDM have been similarly unsuccessful, reflecting the difficulty of modifying this chronic pathophysiology.

A limitation of this study is the observational design wherein only severe GDM was treated, while milder GDM was not, such that the two GDM groups differed both in severity and treatment. This design precludes definitive attribution of causality to the impact of GDM treatment. However, recognizing the improbability of conducting a trial of treated versus untreated GDM, the current analysis provided an analytic approach for addressing the research question with observational data. Another limitation is that generalizability may be limited since the study population was 67.6% Caucasian. It is also possible that some women in the severe treated GDM group could have had undiagnosed prepregnancy diabetes. In addition, β-cell function and insulin sensitivity were measured with surrogate indices on OGTT rather than by clamp studies. Finally, participant awareness of prediabetes/diabetes at 3 months postpartum could have influenced lifestyle and glucose tolerance at 12 months.

In conclusion, these data suggest that while treatment of GDM appeared to disrupt the association between gestational glycemia and birth weight, there

was no discernible effect on the association between gestational glycemia and postpartum maternal metabolic function. Thus, the clinical implications of these findings are support for antenatal glucose-lowering therapy in women with GDM, coupled with the reminder that postpartum metabolic surveillance remains essential in this patient population, irrespective of the effect observed at delivery.

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Author Contributions. R.R. wrote the manuscript. R.R. and C.Y. verified the data. C.Y. performed the statistical analyses. R.R., A.J.H., P.W.C., M.S., and B.Z. designed and implemented the study. All authors critically revised the manuscript for important intellectual content and approved the final manuscript. R.R. is the guarantor of this work and, as such, had full access to all the data in the study and takes responsibility for the integrity of the data and the accuracy of the data analysis.

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