An Abnormal Screening Glucose Challenge Test in Pregnancy Predicts Postpartum Metabolic Dysfunction, Even When the Antepartum Oral Glucose Tolerance Test is Normal

Ravi Retnakaran, MD^{1,2}, Ying Qi, MSc¹, Mathew Sermer, MD³, Philip W Connelly, PhD^{2,4}, Anthony JG Hanley, PhD^{1,2,5}, and Bernard Zinman, MD^{1,2}

¹ Leadership Sinai Centre for Diabetes, Mount Sinai Hospital, Toronto, Canada

² Division of Endocrinology, University of Toronto

³ Division of Obstetrics and Gynecology, Mount Sinai Hospital

⁴ Keenan Research Centre in the Li Ka Shing Knowledge Institute of St. Michael's Hospital, Toronto

⁵ Department of Nutritional Sciences, University of Toronto

Abstract

Objective—In pregnancy, a normal result on the oral glucose tolerance test (OGTT) that follows an abnormal screening glucose challenge test (GCT) is considered a reassuring finding, requiring no further intervention. The obstetrical and metabolic implications of this presentation, however, have not been well-studied. Thus, we sought to characterize the obstetrical and postpartum metabolic significance of an abnormal GCT in women with normal glucose tolerance (NGT) on antepartum OGTT.

Design/Patients/Measurements—259 women with NGT on antepartum OGTT (166 with an abnormal GCT and 93 with normal GCT) underwent (i) metabolic evaluation in pregnancy, (ii) assessment of obstetrical outcome at delivery and (iii) repeat metabolic characterization by OGTT at 3-months postpartum.

Results—Neither infant birthweight nor Caesarian-section rate differed between the abnormal GCT and normal GCT groups. At 3-months postpartum, however, compared to the normal GCT group, the abnormal GCT group exhibited greater glycemia (mean area-under-the-glucose-curve (AUC_{gluc}) 19.6 vs 18.3, $p=0.0021$), lower insulin sensitivity (median IS_{OGTT} 9.5 vs 11.3, $p=0.0243$) and poorer beta-cell function (median insulinogenic index/HOMA-IR 9.8 vs 14.1, $p=0.0013$). On multiple linear regression analyses, an abnormal GCT emerged as (i) the strongest independent predictor of postpartum AUC_{gluc} ($t=2.77$, $p=0.006$) and (ii) the strongest independent negative predictor of log insulinogenic index/HOMA-IR ($t=-2.36$, $p=0.0191$). Furthermore, the GCT was the antepartum parameter that best predicted postpartum pre-diabetes (area-under-receiver-operating-characteristic-curve=0.754).

Conclusions—An abnormal antepartum GCT, even when followed by a normal OGTT, is associated with postpartum glycemia and beta-cell dysfunction, factors that may portend an increased future risk of diabetes in this patient population.

Keywords

glucose challenge test; pregnancy; glucose intolerance; postpartum

INTRODUCTION

Gestational diabetes mellitus (GDM) is associated with (i) adverse obstetrical outcomes, particularly related to fetal macrosomia, and (ii) an increased risk of developing type 2 diabetes (T2DM) in the years following the index pregnancy (1,2). As such, standard obstetrical practice involves screening pregnant women for the presence of GDM. Although universal consensus regarding the appropriate screening protocol is lacking, one common approach is the performance of a 50g glucose challenge test (GCT) in all women in late 2nd trimester, followed by a diagnostic oral glucose tolerance test (OGTT) in those women in whom the GCT is abnormal. Women identified with GDM are then (i) treated with dietary or insulin therapy in order to reduce glucose levels and decrease the risk of infant macrosomia and (ii) advised to undergo postpartum glucose tolerance testing (3,4).

Recently, it has emerged that even women with mildly elevated glucose levels on antepartum GDM screening are still at risk of fetal macrosomia (5). Similarly, we have reported that women with mild glucose intolerance in pregnancy (ie. less severe than GDM) are also at increased risk of developing pre-diabetes or diabetes at 3-months postpartum (6). Thus, taken together, these data suggest that the obstetrical and postpartum metabolic risks of GDM extend to much lower levels of maternal glycemia than currently recognized, a possibility that may have important implications for a large number of women with mild abnormalities on GDM screening.

The mildest possible abnormal category on antepartum GDM screening is represented by those women with normal glucose tolerance (NGT) on the OGTT but an isolated abnormal GCT result. Although NGT on the OGTT following an abnormal GCT is generally considered a reassuring result (one that does not require any clinical intervention), it should be noted that there has been limited study to date of the obstetrical and postpartum metabolic implications of this presentation. Thus, in light of the aforementioned observations regarding the significance of mild dysglycemia in pregnancy, our objective in the current analysis was to systematically evaluate obstetrical outcomes and postpartum metabolic function in women with an abnormal GCT followed by NGT on antepartum OGTT, as compared to a control group of women with both a normal GCT and NGT.

RESEARCH DESIGN AND METHODS

This analysis was conducted in the setting of an ongoing observational study of early events in the natural history of T2DM, in which a cohort of women recruited at the time of GDM screening is undergoing longitudinal metabolic characterization in pregnancy and the postpartum period (6,7). Standard obstetrical practice at our institution involves universal

screening for GDM in all pregnant women at 24–28 weeks' gestation by 50g GCT followed by, if the GCT is abnormal (1-hour post-challenge plasma glucose ≥ 7.8 mmol/L), referral for a diagnostic OGTT. In the study, healthy pregnant women are recruited either prior to or just after their GCT. Regardless of the GCT result, all study participants undergo a 3-hour 100g OGTT for determination of glucose tolerance status in pregnancy. At 3-months postpartum, participants undergo re-assessment by 2-hour 75g OGTT. The study protocol has been approved by the Mount Sinai Hospital Research Ethics Board and all participants have provided written informed consent. The current analysis was restricted to those women with NGT on the antepartum OGTT (n=259), as defined by National Diabetes Data Group criteria (8). For this analysis, these women were stratified into 2 groups: (i) those with an abnormal GCT (n=166) and (ii) those with a normal GCT (n=93).

Participant Assessments

On the morning of the 3-hour 100g OGTT in pregnancy, data pertaining to personal history of previous GDM and family history of diabetes were collected by interviewer-administered questionnaire. Anthropometric measurements of height and weight were also obtained using a medical scale.

At delivery, data on obstetrical outcome was entered into a database that tracks labour and delivery data at Mount Sinai Hospital. Large-for-gestational-age (LGA) was defined as sex-specific birthweight for gestational age above the 90th percentile of Canadian population fetal growth curves (9). Macrosomia was defined as birthweight $\geq 4,000$ grams.

At 3-months postpartum, participants returned to the clinical investigation unit for a 2-hour 75g OGTT. Physical examination was also performed, including measurement of weight and waist circumference.

Laboratory Measurements and Physiologic Indices

For all OGTTs, participants arrived in the morning after overnight fast. Venous blood samples were drawn for measurement of glucose and insulin at fasting and at 30-, 60- and 120-minutes (and 180-minutes in pregnancy). Specific insulin was measured using the Roche Modular system and the electrochemiluminescence immunoassay kit (Roche Diagnostics catalogue number 12017547122). This assay shows 0.05% cross-reactivity to intact human proinsulin and the primary circulating split form (Des 31,32).

At both baseline and follow-up, glycemia was assessed by the total area-under-the-glucose-curve (AUC_{gluc}) during the OGTT, calculated using the trapezoidal rule. Glucose tolerance status on OGTT at 3-months postpartum was defined according to Canadian Diabetes Association guidelines, as previously described (6,10). Pre-diabetes refers to (i) impaired glucose tolerance (defined as 2-hour glucose between 7.8–11.0 mmol/L inclusive), (ii) impaired fasting glucose (defined as fasting glucose between 6.1–6.9 mmol/L inclusive) or (iii) both (10). Insulin sensitivity was measured using the insulin sensitivity index (IS_{OGTT}) of Matsuda and DeFronzo (11). In pregnant women, IS_{OGTT} exhibits better correlation with insulin sensitivity measured by euglycemic-hyperinsulinemic clamp than either Homeostasis Model Assessment of Insulin Resistance (HOMA-IR) or Quantitative Insulin Sensitivity Check Index (12). Beta-cell function was assessed by the insulinogenic index divided by

HOMA-IR (13,14). The insulinogenic index was calculated as the incremental change in insulin concentration during the first 30 minutes of the OGTT divided by the incremental change in glucose during the same time period (15).

Statistical Analyses

All analyses were conducted using the Statistical Analysis System (SAS 9.1, SAS Institute, Cary NC). Continuous variables were tested for normality of distribution (by descriptive histogram and theory-driven Q-Q plot) and natural log transformations of skewed variables were used, where necessary, in subsequent analyses. In Table 1, baseline characteristics at the time of the OGTT in pregnancy (Panel A), obstetrical outcomes (Panel B), and metabolic characteristics at 3-months postpartum (Panel C) are presented for the 2 study groups (normal GCT and abnormal GCT). Continuous variables are presented as median followed by interquartile range if skewed, or mean followed by standard error if normally distributed, while categorical variables are presented as proportions. Univariate differences between the groups were assessed using Analysis of Variance for continuous variables and χ^2 test for categorical variables. Conservative Bonferroni adjustment for multiple comparisons was performed. In Table 2, multiple linear regression analysis was used to identify the antepartum factors that independently predicted the following metabolic parameters at 3-months postpartum: (i) log IS_{OGTT} (model A), and (ii) log insulinogenic index/HOMA-IR (model B). Covariates included age, pre-pregnancy BMI, family history of T2DM, previous GDM, ethnicity (with Caucasian as reference group) and abnormal GCT (with normal GCT as reference group). Receiver-operating-characteristic (ROC) analysis was performed to assess the discriminative capacity of antepartum metabolic measures in predicting postpartum pre-diabetes (Figure 1). The following antepartum measures were evaluated: GCT, AUC_{gluc}, insulinogenic index/HOMA-IR, IS_{OGTT} and each glucose value from the OGTT in pregnancy.

RESULTS

Baseline Characteristics of Study Groups

Table 1 Panel A shows the baseline characteristics of the study subjects stratified into two groups: (i) normal GCT (n=93) and (ii) abnormal GCT (n=166). The antepartum OGTT was performed slightly later in pregnancy in the normal GCT group than in the abnormal GCT group (mean 32.2 vs 29.1 weeks, $p<0.0001$). Otherwise, the two groups were not significantly different with respect to age, pre-pregnancy BMI, family history of T2DM, previous GDM, ethnicity, insulin sensitivity (IS_{OGTT}) and beta-cell function (insulinogenic index/HOMA-IR) in pregnancy. As would be expected, mean GCT value was significantly higher in the abnormal GCT group (8.6 vs 5.8 mmol/L, $p<0.0001$). More importantly, although all of the women had NGT on the OGTT, almost all glycemic measures from the OGTT (including fasting glucose, 1-hr glucose, 2-hour glucose, and AUC_{gluc}) were significantly higher in the abnormal GCT group compared to the normal GCT group (all $p<0.005$).

Obstetrical Outcomes of Study Groups

Having demonstrated greater antepartum glycemia in the women with an abnormal GCT, we next compared the study groups with respect to obstetrical outcomes (Table 1 Panel B). This comparison revealed that infant birthweight was not significantly different between the abnormal GCT (3395 $\pm$ 574g) and normal GCT group (3360 $\pm$ 526g) ($p=0.6355$). There were also no significant differences with respect to length of gestation, infant gender, Apgar scores, and Caesarian-section rates. Although rates of macrosomia and LGA were both higher in the abnormal GCT group, neither difference reached statistical significance ($p=0.2542$ and $p=0.0943$, respectively).

Postpartum Metabolic Characteristics of Study Groups

At 3-months postpartum (Table 1 Panel C), neither BMI nor waist circumference differed between the two groups. Nevertheless, clear metabolic differences between the groups were apparent. Specifically, at 3-months postpartum, women with an abnormal GCT in pregnancy had lower insulin sensitivity (IS_{OGTT} : $p=0.0243$) and poorer beta-cell function (insulinogenic index/HOMA-IR: $p=0.0013$) than their peers who had had a normal GCT. These features translated into greater glycemia in the abnormal GCT group, as reflected by 2-hr blood glucose ($p=0.0074$) and AUC_{gluc} ($p=0.0021$). Most importantly, the prevalence of pre-diabetes was significantly higher in the abnormal GCT group than in the normal GCT group (10.2% vs 3.2%, $p=0.0429$).

Antepartum Predictors of Postpartum Metabolic Dysfunction

Having demonstrated that an abnormal GCT was associated with postpartum metabolic defects, we next sought to determine if these associations were independent of other antepartum factors. Indeed, on multiple linear regression analysis, an abnormal GCT was the strongest independent predictor of postpartum AUC_{gluc} ($t=2.77$, $p=0.006$) (data not shown). The mechanism linking the abnormal GCT group to postpartum dysglycemia did not appear to involve decreased insulin sensitivity, as abnormal GCT was not significantly associated with $\log IS_{OGTT}$ at 3-months postpartum, after adjustment for age, pre-pregnancy BMI, family history of T2DM, previous GDM and ethnicity (Table 2 model A). In contrast, however, abnormal GCT was the sole independent negative predictor of $\log$ insulinogenic index/HOMA-IR ($t=-2.36$, $p=0.0191$) (Table 2 model B), thereby implicating beta-cell dysfunction as a likely factor contributing to postpartum dysglycemia in the abnormal GCT group.

Finally, we compared the discriminative capacity of the GCT with that of other antepartum metabolic measures for the prediction of postpartum pre-diabetes. As shown in Figure 1, the area-under-the-ROC curve (AROC) for the GCT (0.754) exceeded that of other metabolic measures in pregnancy, including AUC_{gluc} (0.592), insulinogenic index/HOMA-IR (0.564) and IS_{OGTT} (0.483), reflecting the superior discriminative capacity of the GCT in this context. The GCT was also superior to each glucose measure on the OGTT in pregnancy, including fasting glucose (AROC=0.541) (data not shown).

DISCUSSION

In this report, we demonstrate that, in women with normal glucose tolerance on antepartum OGTT, an abnormal GCT in pregnancy is associated with postpartum metabolic dysfunction. Specifically, the abnormal GCT is an independent predictor of both glycemia and beta-cell dysfunction at 3-months postpartum. Furthermore, when compared to other antepartum metabolic measures on ROC analysis, the GCT emerged as the best indicator of those women at risk of postpartum pre-diabetes. Overall, these data suggest that the finding of an isolated abnormal GCT in pregnancy may hold significance by potentially identifying a population of women at increased risk of developing T2DM in the future.

Pregnancy provides a natural test of a woman's gluco-regulatory physiology, wherein the pancreatic beta-cells must compensate for the acquired insulin resistance of late gestation, if normoglycemia is to be maintained (16). In this context, it stands to reason that any abnormal glucose homeostasis in pregnancy should reflect some degree of underlying beta-cell dysfunction and hence should be associated with an increased risk of postpartum glucose intolerance. Indeed, consistent with this concept, we have recently demonstrated that any abnormal finding on antepartum GDM screening predicts an increased risk of postpartum pre-diabetes (6,7). In the current analysis, we extend these data by specifically studying the metabolic features of those women with the mildest such abnormality (an abnormal GCT followed by NGT on OGTT) in comparison to their peers with truly normal glucose tolerance in pregnancy (i.e. a normal GCT and NGT on OGTT). Remarkably, the seemingly mild abnormality of an isolated elevated GCT is found to be associated with postpartum metabolic dysfunction, including dysglycemia/glucose intolerance, lower insulin sensitivity and poorer beta-cell function. As such, these data raise the important possibility that an abnormal GCT with NGT in pregnancy may identify a population of women at increased risk of developing T2DM in the future.

To date, there has been very limited study of the postpartum metabolic implications of an abnormal GCT with NGT on antepartum OGTT. In a single previous report, Carr et al used administrative data to demonstrate that women with GCT results above 5.4 mmol/L have a >1.7-fold higher risk of developing diabetes over median follow-up of 8.8 years when compared to their peers with lower GCT values, after limited adjustment for age, primigravidity and pre-term delivery (17). The current study extends this data by (i) systematic evaluation of all subjects by GCT and OGTT in pregnancy (i.e. thereby confirming that all of the women under study had NGT on OGTT), (ii) postpartum metabolic characterization of all subjects by OGTT, (ii) full adjustment for covariates associated with glucose intolerance (including age, BMI, family history of T2DM, previous GDM and ethnicity), and (iii) assessment of insulin sensitivity and beta-cell function. Specifically, we demonstrate that, in women with NGT in pregnancy, an abnormal GCT independently predicts postpartum glycemia and beta-cell dysfunction. The association with poorer postpartum beta-cell function is particularly important, since chronic beta-cell dysfunction is likely the underlying basis for the enhanced risk of T2DM in women with a history of previous GDM (1). Thus, taken together with the findings of Carr et al (17), the current analysis (i) suggests that an abnormal GCT with NGT in pregnancy indeed identifies a population of women at risk of future diabetes and (ii) implicates beta-cell dysfunction as a

potential mechanism underlying this increased risk. Further study will be needed to determine if this group of women warrants enhanced postpartum surveillance for the development of pre-diabetes/diabetes.

As an isolated elevated GCT with NGT on OGTT represents the mildest possible abnormality on antepartum GDM screening, it is to be expected that the increased risk of postpartum glucose intolerance in this group of women will be less than that of their peers with GDM or gestational impaired glucose intolerance (ie. ≥ 2 or 1 abnormal value(s) on OGTT in pregnancy, respectively). As such, if postpartum surveillance is to even be considered in this patient population (abnormal GCT with NGT), the identification of those women within this group at greatest risk of future glucose intolerance emerges as an important issue. In this regard, our ROC analysis suggests that the discriminative capacity of the GCT for predicting postpartum pre-diabetes amongst women with NGT in pregnancy exceeds that of other antepartum metabolic measures. While further study will be needed to determine the optimal strategy for identifying those women at greatest risk, the current data nevertheless highlights the potential significance of the GCT result in women with NGT in pregnancy.

It should be recognized that the usefulness of the GCT as a predictor of postpartum pre-diabetes may be limited by the variability associated with this test. Specifically, a woman's glycemic response to the 50g GCT has been shown to be influenced by several factors, including (i) weeks gestation at the time of testing (18), (ii) time of day of the test (19,20), and (iii) the amount of time since her last meal (21,22). As none of these data were available in this study, we cannot determine whether the predictive capacity of the GCT with respect to postpartum pre-diabetes is truly independent of these factors. Thus, further study aimed at elucidating the role of the GCT for predicting postpartum pre-diabetes will need to adjust for each of these factors (weeks gestation, time of day and the amount of time since the last meal) in order to evaluate the potential independent value of the GCT in this context.

In contrast to the metabolic findings, we did not observe evidence of adverse obstetrical outcomes in this patient population. In this regard, previous studies have reported mixed results. In some cases, abnormal GCT with NGT has been associated with adverse perinatal outcomes and LGA (23–25). Moreover, consistent with these findings, Bevier et al showed that dietary counselling with a euglycemic diet (consisting of 40% carbohydrate, 20% protein and 40% fat) and home glucose monitoring of fasting and postprandial glucose levels can reduce infant birthweight and Caesarian-section rates in this patient population (26). In contrast, other investigators have not found evidence of fetal macrosomia or adverse perinatal outcomes in women with an abnormal GCT followed by NGT (27,28), rendering the obstetrical implications of this presentation somewhat uncertain.

A limitation of the current study is that the sample size ($n=259$) may not have provided sufficient power to detect associations with obstetrical outcomes. Since many other factors may impact fetal growth (29,30), limitations in power could obscure an otherwise modest effect of antepartum glycemia on obstetrical outcomes. Although possibly under-powered for obstetrical associations, this report nevertheless represents the first demonstration of

postpartum metabolic dysfunction in this patient population and should lead to further studies.

In summary, an abnormal GCT in pregnancy, even in the presence of normal glucose tolerance on antepartum OGTT, is associated with abnormalities in postpartum metabolic function. As such, these data raise the important possibility that pregnant women presenting in this way may represent a patient population at increased risk of developing T2DM in the future. Further study is thus warranted, as this population of young women may benefit from enhanced postpartum surveillance.

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Figure 1. Receiver-operating-characteristic (ROC) curves of antepartum metabolic parameters (GCT, AUC_{gluc}, insulinogenic index/HOMA-IR, IS_{OGTT}) for the prediction of pre-diabetes at 3-months postpartum

Table 1

Comparison of women with normal GCT NGT and women with abnormal GCT NGT with respect to (Panel A) antepartum characteristics, (Panel B) obstetrical outcomes, and (Panel C) metabolic characteristics at 3-months postpartum

Panel A: Antepartum Characteristics			
	Normal GCT NGT n=93	Abnormal GCT NGT n=166	p
Age (yrs)	34.0 [4.4]	33.8 [4.2]	0.7632
Weeks gestation	32.2 [2.6]	29.1 [2.4]	<0.0001
Pre-pregnancy BMI (kg/m ²)	23.0 [21.5 – 26.1]	23.5 [21.1 – 27.5]	0.5501
Family history of DM (%)	41.9	50.6	0.1811
Previous GDM/macrosomia (%)	0.0	3.6	0.0633
Ethnicity:			0.8444
Caucasian (%)	79.6	79.5	
Asian (%)	7.5	9.0	
Other (%)	12.9	11.5	
IS _{OGTT}	5.4 [3.6 – 7.3]	5.4 [3.6 – 7.3]	0.9248
Insulinogenic Index/HOMA-IR	13.9 [9.7 – 19.6]	12.8 [8.6 – 18.1]	0.3535
Glucose metabolism:			
Glucose challenge test (mmol/L)	5.8 [1.0]	8.6 [0.8]	<0.0001
OGTT:			
Fasting glucose (mmol/L)	4.2 [0.5]	4.4 [0.4]	0.0033
1-hr glucose (mmol/L)	8.0 [7.0 – 8.5]	8.6 [7.9 – 9.3]	<0.0001
2-hr glucose (mmol/L)	6.6 [5.9 – 7.5]	7.6 [6.7 – 8.3]	<0.0001
3-hr glucose (mmol/L)	6.1 [5.2 – 6.8]	6.0 [4.8 – 6.9]	0.6351
AUC _{gluc}	19.6 [18.3 – 21.1]	21.3 [19.9 – 22.7]	<0.0001
Panel B: Obstetrical Outcomes			
	Normal GCT NGT n=93	Abnormal GCT NGT n=166	p
Length of gestation (wks)	39 [38 – 40]	39 [38 – 40]	0.4001
Infant gender (% M/F)	44/56	50/50	0.3757
1 minute Apgar < 7 (%)	8.9	5.2	0.2617
5 minute Apgar < 7 (%)	0	0	-----
Caesarian-section (%)	28.6	36.1	0.1221
Infant birthweight (g)	3360 [526]	3395 [574]	0.6355
Macrosomia (%)	7.5	12.1	0.2542
LGA (%)	5.6	12.2	0.0943
Panel C: Metabolic Characteristics at 3-months Postpartum			
	Normal GCT NGT n=93	Abnormal GCT NGT n=166	p
Months postpartum	3.2 [2.9 – 3.6]	3.2 [2.9 – 4.1]	0.2198
BMI (kg/m ²)	24.6 [22.6 – 27.9]	25.8 [23.4 – 29.8]	0.3272
Waist circumference (cm)	86.6 [10.9]	87.1 [11.8]	0.7149
IS _{OGTT}	11.3 [8.2 – 15.7]	9.5 [6.6 – 14.2]	0.0243
Insulinogenic Index/HOMA-IR	14.1 [9.6 – 20.0]	9.8 [7.1 – 14.9]	0.0013

Panel A: Antepartum Characteristics

	Normal GCT NGT n=93	Abnormal GCT NGT n=166	p
Glucose metabolism:			
OGTT:			
Fasting glucose (mmol/L)	4.4 [0.4]	4.5 [0.4]	0.1395
2-hr glucose (mmol/L)	5.6 [1.1]	6.0 [1.3]	0.0074
AUC _{gluc}	18.3 [3.1]	19.6 [3.4]	0.0021
Glucose tolerance status:			0.0429
NGT (%)	96.8	89.8	
Pre-diabetes (%)	3.2	10.2	

Continuous data are presented as median followed by interquartile range, if skewed, or mean followed by standard deviation, if normally-distributed. Categorical data are presented as percentages. p-values refer to overall differences across groups as derived from ANOVA analysis for continuous variables (parametric test for normally-distributed variables and nonparametric test for skewed variables) or χ^2 test for categorical variables.

Table 2

Multiple linear regression analyses of the following dependent variables at 3-months postpartum: (Model A) log IS_{OGTT} and (Model B) log insulinogenic index/HOMA-IR

Model A: Dependent variable log IS_{OGTT} at 3-months postpartum				
Variable	Parameter Estimate	Standard Error	t value	p
Age	0.00198	0.00755	0.26	0.7934
Pre-pregnancy BMI	-0.04212	0.00623	-6.76	<0.0001
Family history of diabetes	-0.05934	0.06575	-0.90	0.3677
Previous GDM	-0.82694	0.21090	-3.92	0.0001
Ethnicity:				
Asian ethnicity	-0.40628	0.12000	-3.39	0.0008
Other non-white ethnicity	-0.12875	0.10446	-1.23	0.2190
Abnormal GCT	-0.08882	0.06760	-1.31	0.1902
Model B: Dependent variable log insulinogenic index/HOMA-IR at 3-months postpartum				
Variable	Parameter Estimate	Standard Error	t value	p
Age	-0.01887	0.01199	-1.57	0.1168
Pre-pregnancy BMI	-0.01754	0.00972	-1.80	0.0725
Family history of diabetes	0.08028	0.10348	0.78	0.4386
Previous GDM	-0.12335	0.32976	-0.37	0.7087
Ethnicity:				
Asian ethnicity	0.17288	0.18744	0.92	0.3573
Other non-white ethnicity	0.16976	0.16889	1.01	0.3159
Abnormal GCT	-0.25147	0.10656	-2.36	0.0191