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Hypertension Risk Subsequent to Gestational Dysglycemia Is Modified by Race/Ethnicity

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Abstract

Gestational diabetes mellitus is associated with an increased risk of type 2 diabetes mellitus and hypertension. Additionally, gestational dysglycemia has been associated with an increased risk of type 2 diabetes mellitus but not yet associated with hypertension subsequent to pregnancy in long-term follow-up. Therefore, we set out to examine this relationship as well as the role of race/ethnicity in modifying this relationship. We analyzed a prospective observational cohort followed between 1998 and 2007. There were 17 655 women with self-reported race/ethnicity and full-term, live births. A 1-hour 50 g oral glucose-load test and a 3-hour 100 g oral glucose-tolerance test enabled third trimester stratification of women into 1 of 4 glucose-tolerance groups: (1) normal ($n=15\,056$); (2) abnormal glucose-load test ($n=1558$); (3) abnormal glucose-load and -tolerance tests ($n=520$); and (4) gestational diabetes mellitus ($n=521$). Women were then followed for a mean $\pm$ standard deviation of 4.1 ± 2.9 years after delivery for the development of hypertension. Although gestational diabetes mellitus was associated with an increased risk of hypertension after pregnancy (odds ratio [95% confidence interval]: 1.58 [1.02, 2.45]; $P=0.04$), dysglycemia defined by an abnormal glucose-load test predicted hypertension only among black women (4.52 [1.24, 16.52]; $P=0.02$). The risk of hypertension after pregnancy among dysglycemia groups not meeting criteria for gestational diabetes mellitus varied based on the race/ethnicity of the population. Further research on the implications of the intersection of race/ethnicity and gestational dysglycemia on subsequent hypertension is warranted.

Keywords

diabetes mellitus; ethnic groups; gestational; glucose intolerance; hypertension; pregnancy

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Disclosures

None.

Gestational diabetes mellitus (GDM), defined as diabetes mellitus diagnosed during the second or third trimester that is not clearly overt diabetes mellitus,¹ affects $\approx 9.2\%$ of pregnancies.² GDM has been associated with adverse maternal outcomes subsequent to pregnancy, such as increased incidence of type 2 diabetes mellitus,³ hypertension,⁴ and cardiovascular disease (CVD).^{5,6} Furthermore, women with gestational dysglycemia not meeting criteria for GDM are at increased risk of type 2 diabetes mellitus,⁷ metabolic syndrome,⁸ and overt CVD.⁹ However, because the effect of race/ethnicity in these relationships has not been investigated, we sought to examine this relationship more closely.

Race/ethnicity has been shown to differentially impact several CVD risk factors, including type 2 diabetes mellitus,¹⁰ metabolic syndrome,¹¹ and hypertension.⁴ Similarly, GDM has been shown to be more prevalent among racially/ethnically diverse than among non-Hispanic white populations.¹² A recent study by Bentley-Lewis et al found that black and Hispanic women with a history of GDM were more likely to develop hypertension and did so more rapidly compared with white women with a history of GDM. In addition, they observed that women with GDM had greater hypertension risk relative to their race/ethnicity-specific counterparts without GDM in race-stratified analyses.⁴ However, this investigation did not examine the impact of variable degrees of glucose intolerance on the progression to hypertension. Consequently, our population-based cohort study will extend the current literature by examining this previously published cohort study enhanced by additional racial/ethnic diversity and stratified by glycemia ranging from normal glucose tolerance to GDM. We then followed the cohort for the subsequent development of hypertension, hypothesizing that women with gestational dysglycemia not meeting criteria for GDM would be at an increased risk of future hypertension independent of intercurrent type 2 diabetes mellitus. Furthermore, we hypothesized that women of any race/ethnicity with gestational dysglycemia would be at an increased risk of hypertension compared with their normoglycemic race/ethnicity-specific counterparts.

Methods

We analyzed a prospective observational cohort of women who presented for prenatal care to the Massachusetts General Hospital Obstetric Department between September 1998 and January 2007. Details of this population have been previously described.⁴ In brief, from an initial population of 23 223 women representing 31 140 pregnancies, women were studied if they delivered full-term (gestational age ≥ 37 weeks), live infants; had no antepartum history of pregestational diabetes mellitus or CVD identified by medical record review or International Classification of Diseases, ninth revision codes; and had complete third trimester (24–28 weeks gestational age) race/ethnicity and biochemical data, including a 1-hour glucose-load test (GLT) and a 3-hour glucose-tolerance test (GTT). Race/ethnicity was self-reported as singularly white, black, Asian, or Hispanic. Institutional review board approval was granted by the Partners Human Research Committee before study initiation, and all study participants provided informed written consent.¹³

The primary exposure, glucose tolerance, was defined by the results from the GLT and GTT, which enabled stratification of participants into 1 of 4 glucose-tolerance groups: (1) normal GLT (60 $\leq$ GLT < 140 mg/dL); (2) abnormal GLT (140 $\leq$ GLT < 200 mg/dL and normal GTT);

(3) abnormal GLT/GTT (140 GLT<200 mg/dL and 1 abnormal GTT); and (4) GDM (140 GLT<200 mg/dL and 2 abnormal GTT). Abnormal values on the GTT were based on Carpenter–Coustan criteria.¹⁴ The primary outcome, hypertension, was identified by International Classification of Diseases, ninth revision code 401.xx obtained from electronic medical records comprising inpatient and outpatient data.

Additional clinical data included height, weight, and baseline seated blood pressure measured at the first prenatal visit. Blood pressures were measured as part of usual clinical care after at least 5 minutes of rest in a seated upright position. Measurements were obtained in a quiet office by clinicians experienced and well-trained in the conduct of mercury sphygmomanometer blood pressure acquisition by Korotkoff sound auscultation.¹⁵ Body mass index (BMI) was calculated as weight (kg)/height (m²). Gestational weight gain was calculated as the difference between the initial prenatal visit weight in the first trimester and the third trimester visit weight measured proximate to delivery. Gravidity, parity, and breastfeeding at discharge were abstracted from the medical record. Smoking history was abstracted through an electronic medical record review of each woman’s Massachusetts General Hospital encounters within 1 year after delivery. Years of education and marital status were obtained through maternal self-report. Length of follow-up was calculated from date of delivery to the date of last Massachusetts General Hospital encounter.

Characteristics of the study population stratified by glycemic status were described by number (percentage) for categorical variables and mean±SD for continuous variables. Independent samples *t* tests and χ^2 tests were used to compare demographic and clinical characteristics by exposure status. One-way analysis of variance was used to summarize race- and glycemic status–stratified characteristics. Multiple comparisons were performed as necessary using post hoc adjustments. Follow-up time was summarized using mean±SD.

Women with normal GLT were used as the reference group for all analyses. Women were censored if they developed type 2 diabetes mellitus before the development of hypertension to minimize confounding because having type 2 diabetes mellitus is a significant risk factor for CVD.¹⁶ If no hypertension developed, women were censored at the time of their last Massachusetts General Hospital encounter. Statistical comparisons among glycemia groups were conducted using repeated measures logistic regression models with generalized estimating equations accounting for correlations between repeated pregnancies in the same mother.

Multivariable Cox proportional hazards models with time-varying covariates were used to examine the independent association between glycemic status and risk of hypertension after delivery, adjusted for age, race, gravidity, parity, systolic blood pressure, BMI, gestational weight gain, birth weight for gestational age percentile, breastfeeding at discharge, smoking status, marital status, and years of education. Covariates were selected based on statistical significance among glycemia groups in age-adjusted analyses. Additional clinical and socioeconomic factors were selected if considered to be potential confounders of the relationship between dysglycemia and hypertension. For instance, prior studies have suggested that breastfeeding results in lower maternal blood pressure;¹⁷ marriage is associated with a lower risk of hypertension;¹⁸ smoking is a significant independent risk

factor for hypertension;¹⁹ and education is inversely associated with hypertension risk factors, such as smoking, BMI, and systolic blood pressure.²⁰

To examine effect modification between glycemic status and race, race-stratified Cox proportional hazards models were performed. Multivariable race-stratified models retained the same terms as overall models except for the race covariate. *P* values <0.05 were considered statistically significant. All statistical analyses were performed using SAS for Windows, version 9.2 (SAS Institute Inc., Cary, NC).

Results

The women included in the analytic population (n=17 655; Figure) were followed for a mean±SD of 4.1±2.9 years after delivery for the development of hypertension. Baseline characteristics are summarized in Table 1 with additional covariates listed in Table S1 in the online-only Data Supplement. Notably, women with abnormal GLT (n=1558), abnormal GLT/GTT (n=520), and GDM (n=521) were significantly older, had higher BMI, had higher systolic and diastolic blood pressures, higher baby weight, and were of a greater birth weight for gestational age percentile compared with women with normal GLT (n=15 056). Women with GDM also had greater gestational weight gain compared with women with normal GLT.

Regarding risk factors for hypertension, we observed no significant difference in smoking status across the 4 glycemia groups. Women with GDM had significantly fewer years GLT/GTT were more likely to be married than women with normal GLT. Comparing within each racial/ethnic group, black and Hispanic women were most likely to have GDM and white women were most likely to have normal GLT. Asian women were more likely to have abnormal GLT, abnormal GLT/GTT, or GDM than normal GLT.

When considering the association between glycemic status and subsequent hypertension (Table 2), only women with GDM had an increased risk of hypertension compared with women with normal GLT after full multivariable adjustment. However, women with abnormal GLT had an increased risk of hypertension before adjustment for socioeconomic status. Notably, these data are in contrast to the risk of type 2 diabetes mellitus, which is increased at all levels of dysglycemia regardless of statistical adjustments (Table S2). In race-stratified analyses (Table 3), there was no significant difference in hypertension risk among Asian women with dysglycemia compared with Asian women without dysglycemia. Both white and Hispanic women with GDM had a significantly higher risk of hypertension compared with their racially/ethnically-congruent counterparts without GDM after adjusting for age, gravidity, and live births; however, these results did not remain significant after introducing additional covariates. Black women with abnormal GLT or GDM were at significantly higher risk of hypertension compared with black women with normal GLT when adjusting for age, gravidity, and live births. After full multivariable adjustment, only black women with abnormal GLT had a significantly increased risk of hypertension compared with black women with normal GLT.

Discussion

In this study, we examined the ability of varying degrees of dysglycemia to predict hypertension independent of intercedent type 2 diabetes mellitus and after adjusting for several demographic, clinical, and socioeconomic status covariates. We observed no relationship between non-GDM levels of dysglycemia and incidence of hypertension in the general population. However, when examining the population stratified by race/ethnicity, we observed that black women with an abnormal GLT did have an increase in subsequent hypertension relative to black women with normal GLT.

An investigation by Retnakaran et al examined the association between gestational dysglycemia and subsequent hypertension.²¹ They observed that women with impaired glucose tolerance or GDM had significantly higher systolic blood pressures and a greater use of antihypertensive medications than women with normoglycemia at 3 months postpartum.²¹ However, this study did not perform a prepregnancy assessment of cardiovascular risk factors, thereby limiting our ability to determine whether the increased CVD risk was present before pregnancy.

Additionally, to our knowledge, our study is the first published examination of the impact of race/ethnicity on the relationship between gestational dysglycemia and subsequent hypertension. Comparing within each racial/ethnic groups, black and Hispanic women were more likely to have GDM than normal GLT, and white women were more likely to have normal GLT than GDM. Asian women were more likely to have any degree of dysglycemia than normal GLT. In race/ethnicity-stratified analyses of the non-GDM dysglycemia groups, only black women with abnormal GLT were at a greater risk for hypertension compared with black women with normal GLT. This finding was only evident in the abnormal GLT group and not the groups with greater degrees of dysglycemia, potentially reflecting the smaller sample size of the abnormal GLT/GTT group of black women and a lack of incremental impact of race on the background of dysglycemia. Because our sample size of black and Asian women within each glycemia group was limited relative to the numbers of white or Hispanic women, further research in larger, racially/ethnically diverse populations may enhance our understanding in this area. Nonetheless, our data reveal a differential risk of hypertension based on degree of glucose intolerance, and this risk is further impacted by population race/ethnicity.

The strengths of our study lie in the examination of the impact of race/ethnicity on the relationship between degrees of gestational dysglycemia and hypertension while eliminating potential confounding by intercedent type 2 diabetes mellitus. We statistically adjusted our models for several demographic, clinical, and socioeconomic factors, which have been shown to influence the risk of hypertension.^{17–20} The mean follow-up period subsequent to delivery of 4.1 years enabled assessment of hypertension development beyond the postpartum period. Additionally, the racial/ethnic distribution of our population of pregnant women is comparable to and, therefore, generalizable to that of the United States.²²

Despite the rigorous methodology by which we obtained and analyzed our data, we acknowledge several potential limitations to our study. First, the limited sample size and

consequent limited racial/ethnic diversity among black and Asian women potentially limited our ability to detect differences in these subgroups. Additionally, the use of electronic medical records may have limited our ascertainment of data that could impact hypertension risk, such as changes in smoking, physical activity, or BMI. Moreover, we may have missed data if women presented for medical care at outside facilities during the study time period. Nonetheless, we are confident in the data obtained and in our ability to draw conclusions from these data, while recognizing that these limitations prompt further research in a larger, more racially/ethnically diverse population of women.

Perspectives

Gestational dysglycemia that does not meet criteria for GDM could be considered among the risk factors for hypertension depending on an individual's race/ethnicity. For instance, our study identified a significant positive association between gestational dysglycemia and hypertension risk subsequent to pregnancy among black women, even after adjusting for several clinical, demographic, and socioeconomic status characteristics. However, because black women represented only 4.5% of the dysglycemia group, further study in populations with larger race/ethnicity subgroups is warranted to confirm that gestational dysglycemia is an independent risk factor for hypertension. Nonetheless, recognition of the potential adverse health outcomes associated with varying degrees of glucose tolerance may enhance risk stratification for women at risk for hypertension after pregnancy.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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References

1. American Diabetes Association. 2. Classification and diagnosis of diabetes. Diabetes Care. 2015; 38:S8–S16.10.2337/dc15-S005
2. DeSisto CL, Kim SY, Sharma AJ. Prevalence estimates of gestational diabetes mellitus in the United States, Pregnancy Risk Assessment Monitoring System (PRAMS), 2007–2010. Prev Chronic Dis. 2014; 11:E104.10.5888/pcd11.130415 [PubMed: 24945238]
3. Bellamy L, Casas JP, Hingorani AD, Williams D. Type 2 diabetes mellitus after gestational diabetes: a systematic review and meta-analysis. Lancet. 2009; 373:1773–1779.10.1016/S0140-6736(09)60731-5 [PubMed: 19465232]
4. Bentley-Lewis R, Powe C, Ankers E, Wenger J, Ecker J, Thadhani R. Effect of race/ethnicity on hypertension risk subsequent to gestational diabetes mellitus. Am J Cardiol. 2014; 113:1364–1370.10.1016/j.amjcard.2014.01.411 [PubMed: 24576544]
5. Shah BR, Retnakaran R, Booth GL. Increased risk of cardiovascular disease in young women following gestational diabetes mellitus. Diabetes Care. 2008; 31:1668–1669.10.2337/dc08-0706 [PubMed: 18487472]

6. Bentley-Lewis R. Late cardiovascular consequences of gestational diabetes mellitus. *Semin Reprod Med.* 2009; 27:322–329.10.1055/s-0029-1225260 [PubMed: 19530066]
7. Retnakaran R, Qi Y, Sermer M, Connelly PW, Hanley AJ, Zinman B. Glucose intolerance in pregnancy and future risk of pre-diabetes or diabetes. *Diabetes Care.* 2008; 31:2026–2031.10.2337/dc08-0972 [PubMed: 18628572]
8. Negrato CA, Jovanovic L, Rafacho A, Tambascia MA, Geloneze B, Dias A, Rudge MV. Association between different levels of dysglycemia and metabolic syndrome in pregnancy. *Diabetol Metab Syndr.* 2009; 1:3.10.1186/1758-5996-1-3 [PubMed: 19825195]
9. Retnakaran R, Qi Y, Sermer M, Connelly PW, Hanley AJ, Zinman B. The postpartum cardiovascular risk factor profile of women with isolated hyperglycemia at 1-hour on the oral glucose tolerance test in pregnancy. *Nutr Metab Cardiovasc Dis.* 2011; 21:706–712.10.1016/j.numecd.2011.02.010 [PubMed: 21703831]
10. Brancati FL, Kao WH, Folsom AR, Watson RL, Szklo M. Incident type 2 diabetes mellitus in African American and white adults: the Atherosclerosis Risk in Communities Study. *JAMA.* 2000; 283:2253–2259. [PubMed: 10807384]
11. Ford ES, Giles WH, Dietz WH. Prevalence of the metabolic syndrome among US adults: findings from the third National Health and Nutrition Examination Survey. *JAMA.* 2002; 287:356–359. [PubMed: 11790215]
12. Lawrence JM, Contreras R, Chen W, Sacks DA. Trends in the prevalence of preexisting diabetes and gestational diabetes mellitus among a racially/ethnically diverse population of pregnant women, 1999–2005. *Diabetes Care.* 2008; 31:899–904.10.2337/dc07-2345 [PubMed: 18223030]
13. Partners Healthcare Notice for Use and Sharing of Protected Health Information. *Partners.* 2013:1–8.
14. Coustan DR, Carpenter MW. The diagnosis of gestational diabetes. *Diabetes Care.* 1998; 21(suppl 2):B5–B8. [PubMed: 9704220]
15. Pickering TG, Hall JE, Appel LJ, Falkner BE, Graves J, Hill MN, Jones DW, Kurtz T, Sheps SG, Roccella EJ. Subcommittee of Professional and Public Education of the American Heart Association Council on High Blood Pressure Research. Recommendations for blood pressure measurement in humans and experimental animals: Part 1: blood pressure measurement in humans: a statement for professionals from the Subcommittee of Professional and Public Education of the American Heart Association Council on High Blood Pressure Research. *Hypertension.* 2005; 45:142–161.10.1161/01.HYP.0000150859.47929.8e [PubMed: 15611362]
16. Gu K, Cowie CC, Harris MI. Diabetes and decline in heart disease mortality in US adults. *JAMA.* 1999; 281:1291–1297. [PubMed: 10208144]
17. Ebina S, Kashiwakura I. Influence of breastfeeding on maternal blood pressure at one month postpartum. *Int J Womens Health.* 2012; 4:333–339.10.2147/IJWH.S33379 [PubMed: 22870047]
18. Lipowicz A, Lopuszanska M. Marital differences in blood pressure and the risk of hypertension among Polish men. *Eur J Epidemiol.* 2005; 20:421–427. [PubMed: 16080590]
19. Dochi M, Sakata K, Oishi M, Tanaka K, Kobayashi E, Suwazono Y. Smoking as an independent risk factor for hypertension: a 14-year longitudinal study in male Japanese workers. *Tohoku J Exp Med.* 2009; 217:37–43. [PubMed: 19155606]
20. Eggen AE, Mathiesen EB, Wilsgaard T, Jacobsen BK, Njølstad I. Trends in cardiovascular risk factors across levels of education in a general population: is the educational gap increasing? The Tromsø study 1994–2008. *J Epidemiol Community Health.* 2014; 68:712–719.10.1136/jech-2013-203428 [PubMed: 24675289]
21. Retnakaran R, Qi Y, Connelly PW, Sermer M, Zinman B, Hanley AJ. Glucose intolerance in pregnancy and postpartum risk of metabolic syndrome in young women. *J Clin Endocrinol Metab.* 2010; 95:670–677.10.1210/jc.2009-1990 [PubMed: 19926711]
22. Ventura SJ, Abma JC, Mosher WD, Henshaw SK. Estimated pregnancy rates by outcome for the United States, 1990–2004. *Natl Vital Stat Rep.* 2008; 56:1–25. 28. [PubMed: 18578105]

Novelty and Significance

What Is New?

- Black women with gestational dysglycemia, not fulfilling criteria for gestational diabetes mellitus, are at increased risk for developing hypertension subsequent to pregnancy compared with black women with normal gestational glucose tolerance.

What Is Relevant?

- The consideration of alternative approaches to identify women at increased risk for hypertension may inform strategies targeting primary prevention.

Summary

Race/ethnicity impacts the effect of gestational dysglycemia on the risk for hypertension subsequent to pregnancy. Further understanding of this relationship may enhance our approach to hypertension risk-reduction efforts in women postpregnancy.

Figure.

Study population flow. From our initial Massachusetts General Hospital (MGH) Obstetric Database population of 23 223 women, women were selected who delivered live-born, infants at term (n=19 310); had no history of cardiovascular disease (CVD) or diabetes mellitus (DM) before delivery (n=18 412); and had complete biochemical and race/ethnicity data (n=17 655). Based on a 1-hour glucose-load test (GLT) and 3-hour glucose-tolerance test (GTT), we stratified participants into 1 of 4 glucose-tolerance groups: normal GLT (60 GLT<140 mg/dL; n=15 056), abnormal GLT (140 GLT<200 mg/dL and normal GTT; n=1558), abnormal GLT/GTT (140 GLT<200 mg/dL and 1 abnormal GTT; n=520), and gestational diabetes mellitus (GDM; 140 GLT<200 mg/dL and 2 abnormal GTT; n=521).

Table 1

Baseline Characteristics of Population by Glycemia Group

Characteristic	Normal GLT	P Value	Abnormal GLT	P Value	Abnormal GLT/GTT	P Value	GDM	P Value
Maternal								
N	15 056		1558		520		521	
Age, y	30±5	Ref	31±5	<0.001*	32±5	<0.001*	32±5	<0.001*
BMI, kg/m ²	25.3±5.2	Ref	25.5±5.2	0.02*	26.7±5.9	<0.001*	28.9±7.0	<0.001*
Systolic BP, mm Hg	110±11	Ref	111±11	0.003*	112±12	0.003*	113±12	<0.001*
Diastolic BP, mm Hg	68±8	Ref	69±8	0.04*	70±9	<0.001*	70±8	<0.001*
Heart rate, bpm	76±12	Ref	76±13	0.45	80±16	0.04*	78±12	0.08
Parity		Ref		0.17				0.70
Primiparous, n (%)	7174 (47.7)		773 (49.6)		239 (46.0)		245 (47.0)	
Multiparous, n (%)	7729 (51.3)		775 (49.7)		227 (53.3)		273 (52.4)	
Gravidity (n)	3±1	Ref	2±1	0.88	3±2	0.04*	3±2	<0.001*
Smoking status		Ref		0.39				0.74
Never smoked, n (%)	5531 (36.7)		612 (39.3)		204 (39.2)		204 (39.2)	
Current smoker, n (%)	930 (6.2)		101 (6.5)		36 (6.9)		31 (6.0)	
Past smoker, n (%)	2328 (15.5)		231 (14.8)		87 (16.7)		91 (17.5)	
Race		Ref		<0.001*		0.002*		<0.001*
Asian, n (%)	943 (6.3)		167 (10.7)		62 (11.9)		61 (11.7)	
Black, n (%)	891 (5.9)		70 (4.5)		27 (5.2)		34 (6.5)	
Hispanic, n (%)	3971 (26.4)		382 (24.5)		137 (26.4)		165 (31.7)	
White, n (%)	9251 (61.4)		939 (60.3)		294 (56.5)		261 (50.1)	
Follow-up time (years)	4.1±2.9	Ref	4.1±2.9	0.76	4.3±2.9	0.15	4.4±3.0	0.07
Obstetric								
Gestational age at delivery, weeks	39.7±1.1	Ref	39.7±1.2	0.30	39.5±1.2	<0.001*	39.3±1.2	<0.001*
Gestational weight gain, kg	28.5±12.0	Ref	28.5±11.6	0.88	28.2±11.6	0.51	23.5±11.9	<0.001*
Baby weight, g	3455±464	Ref	3484±481	0.02*	3544±487	<0.001*	3571±525	<0.001*
Preeclampsia, n (%)	68 (0.5)	Ref	12 (0.8)	0.15	5 (1.0)	0.24	6 (1.2)	0.14
Breast feeding at discharge, n (%)	11 520 (76.5)	Ref	1182 (75.9)	0.45	411 (79.0)	0.53	381 (73.1)	0.12

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BMI indicates body mass index; BP, blood pressure; GDM, gestational diabetes mellitus; GLT, glucose-load test; and GTT, glucose-tolerance test. Normal GLT (60 GLT<140 mg/dL); abnormal GLT (140 GLT<200 mg/dL and normal GTT); abnormal GLT/GTT (140 GLT<200 mg/dL and 1 abnormal GTT); GDM (140 GLT<200 mg/dL and 2 abnormal GTT).

* Significant at P<0.05.

Table 2**Risk of Essential Hypertension by Glycemia Group**

Glycemia Group	HR (95% CI)	P Value
Age adjusted		
Normal GLT	1.0	Ref
Abnormal GLT	1.26 (0.99, 1.62)	0.07
Abnormal GLT/GTT	1.17 (0.78, 1.74)	0.46
GDM	2.52 (1.90, 3.36)	<0.001 *
Demographic adjusted [†]		
Normal GLT	1.0	Ref
Abnormal GLT	1.31 (1.02, 1.68)	0.03 *
Abnormal GLT/GTT	1.16 (0.78, 1.74)	0.47
GDM	2.46 (1.84, 3.28)	<0.001 *
Demographic+clinical adjusted [‡]		
Normal GLT	1.0	Ref
Abnormal GLT	1.35 (1.03, 1.79)	0.03 *
Abnormal GLT/GTT	1.11 (0.72, 1.70)	0.65
GDM	1.83 (1.32, 2.54)	<0.001 *
Demographic+clinical+SES adjusted [§]		
Normal GLT	1.0	Ref
Abnormal GLT	1.21 (0.84, 1.76)	0.31
Abnormal GLT/GTT	1.22 (0.72, 2.07)	0.46
GDM	1.61 (1.04, 2.09)	0.03 *
Demographic+clinical+SES+follow-up time adjusted		
Normal GLT	1.0	Ref
Abnormal GLT	1.22 (0.84, 1.76)	0.30
Abnormal GLT/GTT	1.22 (0.72, 2.06)	0.46
GDM	1.58 (1.02, 2.45)	0.04 *

CI indicates confidence interval; GDM, gestational diabetes mellitus; GLT, glucose-load test; GTT, glucose-tolerance test; HR, hazard ratio; SES, socioeconomic status. Normal GLT (60 GLT<140 mg/dL); abnormal GLT (140 GLT<200 mg/dL and normal GTT); abnormal GLT/GTT (140 GLT<200 mg/dL and 1 abnormal GTT); GDM (140 GLT<200 mg/dL and 2 abnormal GTT).

* Significant at P<0.05.

[†]Demographic=age, race, gravidity, parity.

[‡]Clinical=systolic blood pressure, body mass index, gestational weight gain, birth weight for gestational age percentile.

[§]SES=smoking status, breast feed at discharge, marital status, years of education.

Table 3

Risk of Essential Hypertension by Glycemia Group and Race

Glycemia Group	Asian			Black			Hispanic			White		
	HR (95% CI)	P Value	HR (95% CI)	HR (95% CI)	P Value	HR (95% CI)	HR (95% CI)	P Value	HR (95% CI)	HR (95% CI)	P Value	
Age adjusted												
Normal GLT	1.0	Ref	1.0	2.09 (1.10, 3.97)	Ref	1.0	1.09 (0.64, 1.85)	Ref	1.0	1.23 (0.88, 1.71)	Ref	
Abnormal GLT	1.57 (0.57, 4.29)	0.38	2.09 (1.10, 3.97)	0.03 *	0.03 *	1.0	1.09 (0.64, 1.85)	0.76	1.0	1.23 (0.88, 1.71)	0.22	
Abnormal GLT/GTT	0.71 (0.10, 5.39)	0.74	2.26 (0.97, 5.25)	0.06	0.06	1.0	1.42 (0.72, 2.79)	0.31	0.79 (0.41, 1.54)	0.79 (0.41, 1.54)	0.49	
GDM	0.74 (0.10, 5.57)	0.77	2.51 (1.14, 5.52)	0.02 *	0.02 *	1.0	2.34 (1.43, 3.81)	<0.001 *	2.50 (1.66, 3.76)	2.50 (1.66, 3.76)	<0.001 *	
Demographic adjusted [†]												
Normal GLT	1.0	Ref	1.0	2.03 (1.06, 3.86)	Ref	1.0	1.10 (0.64, 1.87)	Ref	1.0	1.23 (0.89, 1.71)	Ref	
Abnormal GLT	1.58 (0.57, 4.39)	0.38	2.03 (1.06, 3.86)	0.03 *	0.03 *	1.0	1.10 (0.64, 1.87)	0.73	1.23 (0.89, 1.71)	1.23 (0.89, 1.71)	0.22	
Abnormal GLT/GTT	0.63 (0.08, 4.86)	0.66	2.31 (0.99, 5.37)	0.05	0.05	1.0	1.43 (0.73, 2.83)	0.30	0.80 (0.41, 1.56)	0.80 (0.41, 1.56)	0.51	
GDM	0.75 (0.10, 5.69)	0.78	2.63 (1.19, 5.82)	0.02 *	0.02 *	1.0	2.29 (1.40, 3.75)	<0.001 *	2.53 (1.68, 3.80)	2.53 (1.68, 3.80)	<0.001 *	
Demographic+clinical adjusted [‡]												
Normal GLT	1.0	Ref	1.0	3.60 (1.58, 8.19)	Ref	1.0	1.07 (0.60, 1.92)	Ref	1.0	1.28 (0.90, 1.82)	Ref	
Abnormal GLT	1.85 (0.46, 7.50)	0.39	3.60 (1.58, 8.19)	0.002 *	0.002 *	1.0	1.07 (0.60, 1.92)	0.81	1.28 (0.90, 1.82)	1.28 (0.90, 1.82)	0.18	
Abnormal GLT/GTT	1.12 (0.13, 9.74)	0.92	2.1 (0.75, 6.05)	0.16	0.16	1.0	1.52 (0.74, 3.14)	0.26	0.73 (0.38, 1.44)	0.73 (0.38, 1.44)	0.37	
GDM	1.14 (0.13, 10.04)	0.91	1.86 (0.74, 4.72)	0.19	0.19	1.0	1.83 (1.04, 3.22)	0.04 *	1.76 (1.10, 2.79)	1.76 (1.10, 2.79)	0.02 *	
Demographic+clinical+SES adjusted [§]												
Normal GLT	1.0	Ref	1.0	4.52 (1.23, 16.55)	Ref	1.0	1.10 (0.52, 2.30)	Ref	1.0	1.10 (0.68, 1.76)	Ref	
Abnormal GLT	1.05 (0.05, 22.13)	0.98	4.52 (1.23, 16.55)	0.02 *	0.02 *	1.0	1.10 (0.52, 2.30)	0.81	1.10 (0.68, 1.76)	1.10 (0.68, 1.76)	0.70	
Abnormal GLT/GTT	5.34 (0.31, 92.30)	0.25	3.03 (0.91, 10.14)	0.07	0.07	1.0	1.43 (0.56, 3.62)	0.45	0.80 (0.33, 1.94)	0.80 (0.33, 1.94)	0.62	
GDM	2.33 (0.16, 34.24)	0.54	1.54 (0.42, 5.71)	0.52	0.52	1.0	1.35 (0.59, 3.08)	0.48	1.59 (0.86, 2.97)	1.59 (0.86, 2.97)	0.14	
Demographic+clinical+SES+follow-up time adjusted												
Normal GLT	1.0	Ref	1.0	4.52 (1.24, 16.52)	Ref	1.0	1.07 (0.51, 2.26)	Ref	1.0	1.09 (0.68, 1.75)	Ref	
Abnormal GLT	1.18 (0.05, 27.64)	0.92	4.52 (1.24, 16.52)	0.02 *	0.02 *	1.0	1.07 (0.51, 2.26)	0.85	1.09 (0.68, 1.75)	1.09 (0.68, 1.75)	0.72	
Abnormal GLT/GTT	7.00 (0.32, 155.12)	0.22	2.86 (0.82, 9.98)	0.10	0.10	1.0	1.46 (0.58, 3.71)	0.42	0.81 (0.33, 1.98)	0.81 (0.33, 1.98)	0.64	
GDM	3.92 (0.20, 78.17)	0.37	1.51 (0.40, 5.67)	0.55	0.55	1.0	1.32 (0.58, 3.00)	0.52	1.52 (0.82, 2.84)	1.52 (0.82, 2.84)	0.19	

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CI indicates confidence interval; GDM, gestational diabetes mellitus; GLT, glucose-load test; GTT, glucose-tolerance test; HR, hazard ratio; and SES, socioeconomic status. Normal GLT (60 GLT < 140 mg/dL); abnormal GLT (140 GLT < 200 mg/dL and normal GTT); abnormal GLT/GTT (140 GLT < 200 mg/dL and 1 abnormal GTT); GDM (140 GLT < 200 mg/dL and 2 abnormal GTT).

* Significant at $P < 0.05$.

[†] Demographic = age, gravidity, live births.

[‡] Clinical = systolic blood pressure, body mass index, gestational weight gain, birth weight for gestational age percentile.

[§] SES = smoking status, breast feed at discharge, marital status, years of education.