

RACIAL/ETHNIC DIFFERENCES IN PREGNANCY-RELATED HYPERTENSIVE DISEASE IN NULLIPAROUS WOMEN

Objective: Hypertension and cardiovascular disease rates vary by race/ethnicity in nonpregnant adults. We aimed to examine racial/ethnic differences in prevalence and severity of hypertensive diseases during pregnancy in nulliparous women.

Design, Setting, Participants: Nulliparous women with singleton deliveries and electronic medical record data on demographics and pregnancy outcomes ($n=56,617$) were selected from the Consortium on Safe Labor (2002–2008). Multivariable logistic regression was performed to calculate the adjusted odds of gestational hypertension, mild preeclampsia, severe preeclampsia, eclampsia, chronic hypertension, superimposed preeclampsia, and unspecified hypertension for women who were non-Hispanic Black, Hispanic, Asian/Pacific Islander, and multiracial/other race/ethnicity, compared with non-Hispanic White women.

Results: Non-Hispanic Black women had higher odds of entering pregnancy with chronic hypertension (adjusted odds ratio (AOR)=1.43, 95% confidence interval (CI) 1.11–1.84) and had higher odds of developing mild (AOR=1.26, 95% CI 1.10–1.45), severe (AOR=1.31, 95% CI 1.10–1.57) or superimposed preeclampsia (AOR=1.98, 95% CI 1.40–2.80) compared to non-Hispanic White women. Hispanic women and Asian/Pacific Islanders had higher odds of remaining normotensive (AOR=1.22, 95% CI 1.12–1.33 and AOR=1.55, 95% CI 1.31–1.84, respectively).

Conclusions: Odds for specific gestational hypertensive diseases varied by race/ethnicity among women during their first pregnancy. Non-Hispanic Black women experienced more severe disease, while Hispanic women and Asian/Pacific Islanders had an overall decreased risk compared to non-Hispanic Whites. Patterns of racial/ethnic variation associated with hypertensive diseases during pregnancy were similar to racial/ethnic associations reported for adult-onset cardiovascular disease, suggesting that there may be common pathways and shared risk factors. (*Ethn Dis.* 2014;24[3]:283–289)

Key Words: Ethnic Groups, Hypertension, Pregnancy-induced, Pre-eclampsia

From Epidemiology Branch (GG, JG, TM, PM, SKL), and Biostatistics and Bioinformatics Branch (ZC, YX), Eunice Kennedy Shriver National Institute of Child Health and Human Development, National Institutes of Health, Bethesda, Maryland.

Gaurav Ghosh, BS; Jagteshwar Grewal, PhD; Tuija Männistö, MD, PhD; Pauline Mendola, PhD; Zhen Chen, PhD; Yunlong Xie, PhD; S. Katherine Laughon, MD, MS

INTRODUCTION

Women with preeclampsia, a new-onset hypertension in pregnancy with proteinuria, have an approximately 1.7-fold higher risk of dying from cardiovascular disease later in life.¹ Pregnancy has been thought of as a stress test, in that women who develop a hypertensive disease during pregnancy are also at risk for later cardiovascular disease even though elevated blood pressure in pregnancy is typically transient and resolves postpartum.^{2,3} Hypertension during pregnancy and later-life cardiovascular disease may share common pathways and risk factors such as race/ethnicity or other factors associated with race/ethnicity. Outside of pregnancy, non-Hispanic Black women have a higher risk for cardiovascular disease, while Asian and Hispanic women have a lower risk, compared to non-Hispanic Whites.⁴ Moreover, non-Hispanic Black women have consistently been found to have an increased risk of hypertension during pregnancy.^{5–11}

The definitions of gestational hypertensive disorders have changed over time and prior studies often used outcome measures that failed to distinguish between disorders, using combined categories such as pregnancy-induced hypertension, and they were limited by small sample sizes and/or lacked the ability to adjust for

individual clinical data because they were based on birth certificates.^{5–16} Examining specific gestational hypertensive disorders is critical because the evidence suggests that the various disorders may develop through distinct pathways. For example, elevated blood pressure alone during pregnancy (ie, gestational hypertension) may have a different etiology than preeclampsia, and earlier onset preeclampsia may have a different etiology compared to later onset preeclampsia.^{12,17–22} Yet whether racial/ethnic differences in preeclampsia vary by severity of disease, or early- vs later-onset of the disorder, remains unknown.

The primary aim of our study was to evaluate the risk of gestational hypertensive diseases by maternal race/ethnicity in nulliparous women from a large, contemporary US cohort. Our secondary aim was to explore the association of race/ethnicity with severity of hypertensive diseases and timing of delivery/onset.^{19,20}

METHODS

Study Population

The Consortium on Safe Labor, conducted by the Eunice Kennedy

The primary aim of our study was to evaluate the risk of gestational hypertensive diseases by maternal race/ethnicity in nulliparous women from a large, contemporary US cohort.

Address correspondence to S. Katherine Laughon, MD, MS; Epidemiology Branch, Division of Intramural Population Health Research, Eunice Kennedy Shriver National Institute of Child Health and Human Development; National Institutes of Health; 6100 Executive Blvd., Room 7B03; Rockville, MD 20852; 301.435.6935; 301.402.2084 (fax); laughonsk@mail.nih.gov

Shriver National Institute of Child Health and Human Development, National Institutes of Health, involved 12 clinical centers (19 hospitals) from nine American College of Obstetricians and Gynecologists districts between 2002 and 2008.²³ The study was approved by the institutional review boards of all participating institutions. Maternal demographics (including race/ethnicity), medical history, prenatal complications, maternal and neonatal outcomes, delivery summary, and postpartum and newborn information were captured from electronic medical records. Data on race/ethnicity was as recorded in the medical record and was mapped to six predefined categories based on race and ethnic standards for federal statistics and administrative reporting: non-Hispanic White, non-Hispanic Black, Hispanic, Asian/Pacific Islander, multiracial, or other.²⁴ The last two categories were combined for this analysis due to small sample size. To reduce confounding by previous obstetric history, we restricted the analysis to nulliparous women with singleton pregnancies ($n=89,281$).²⁵ Since maternal race/ethnicity was a primary variable of interest, all women who were missing data on maternal race/ethnicity ($n=4,360$) were excluded. Finally, women missing data on covariates, including prepregnancy body-mass index (BMI, calculated as weight in kg/height in m^2) ($n=27,531$), maternal age ($n=28$), and marital status ($n=745$), were also excluded. The series of exclusions yielded a final sample size of 56,617 deliveries.

Classification of Hypertensive Diseases

Hypertensive diseases were initially captured from the medical records as gestational hypertension, preeclampsia, eclampsia, chronic hypertension, superimposed preeclampsia and unspecified hypertension. Information on who made the diagnosis or managed the care was not available. We supplemented these data using the electronic hospital

discharge summary International Classification of Diseases 9th Revision (ICD-9) codes as follows: gestational hypertension (642.3), mild preeclampsia (642.4), severe preeclampsia (642.5), eclampsia (642.6), chronic hypertension (642.0, 642.1 or 642.2), superimposed preeclampsia (642.7), and unspecified hypertension (642.9). Women with no recorded hypertensive disease were considered normotensive. Hypertensive disease diagnoses from discharge ICD9 codes and medical records were generally in agreement. Analyses performed using either medical record diagnosis or ICD-9 codes yielded similar results. We chose to present analyses using ICD-9 codes since the capture of hypertensive diseases in medical records varied somewhat by site.

Data Analysis

Multivariable logistic regressions were performed to calculate the odds ratios (ORs) with 95% confidence intervals (CIs) of gestational hypertension, mild preeclampsia, severe preeclampsia, eclampsia, and unspecified hypertension among women who were non-Hispanic Black, Hispanic, Asian/Pacific Islander, or of multiracial/other race/ethnicity, compared to non-Hispanic White women. We also examined whether the risk of either entering the pregnancy with chronic hypertension or developing superimposed preeclampsia varied for different races/ethnicities. All ORs are adjusted for study site and the fully adjusted models also included maternal age, prepregnancy BMI, insurance status, smoking during pregnancy, and marital status (married, divorced/widowed, single). Study site was deemed as an important covariate to adjust for in all analyses to account for geographical differences in reporting as well as potential underlying differences in unmeasured risk factors. A non-linear relationship between maternal age and hypertensive disease was evaluated using a quadratic term; however, the coeffi-

cients for the main effects were not significantly different, so this term was not included in the final models. A secondary analysis was performed stratifying data by delivery at <34 and ≥ 34 weeks of gestation, as a proxy for early- vs late-onset of gestational hypertensive disease. This analysis was limited to women who were normotensive at the beginning of pregnancy (women with chronic hypertension or superimposed preeclampsia were excluded) and the hypertensive disease categories were collapsed into a composite since the numbers were small for earlier delivery.

To address concerns about potential selection bias in our analytic sample, we compared the maternal, obstetrical, and hospital characteristics of women with missing data on covariates to those with complete data using Chi-squared tests. Deliveries with missing maternal race/ethnicity were less likely to have pregnancies complicated by preeclampsia (5.3% vs 6.1%, $P<.001$), and more likely to involve women who remained normotensive (91.24% vs 88.03%, $P<.001$), when compared to pregnancies where maternal race/ethnicity was known. Women with missing BMI had a higher, but clinically insignificant, percentage of hypertensive diseases compared to those with known BMI (13.4% vs 11.3%, $P<.001$). The similarities in prevalence of gestational hypertensive diseases between women with complete data and women missing data on race/ethnicity or other covariates suggested that excluding cases for these reasons did not appreciably affect our results. We also performed sensitivity analyses to address missing data on maternal prepregnancy BMI. For these analyses, data on prepregnancy BMI were categorized with missing values included as an unknown category in the analyses.²⁶ Second, we analyzed only women with normal prepregnancy BMI (between 18.5 and 24.9 kg/m^2). Analyses were performed using SAS version 9.2 (SAS Institute Inc. Cary, NC).

Table 1. Description of study cohort by race/ethnic groups

	Non-Hispanic White <i>n</i> =30,499 (53.9%)	Non-Hispanic Black <i>n</i> =11,584 (20.5%)	Hispanic <i>n</i> =10,476 (18.5%)	Asian/Pacific Islander <i>n</i> =2,696 (4.8%)	Multiracial/ other <i>n</i> =1,362 (2.4%)	<i>P</i>
Maternal age, years, mean (SD)	26.4 (5.9)	22.7 (5.8)	24.4 (6.1)	29.2 (5.3)	25.3 (6.6)	<.0001
Marital status, <i>n</i> (%)						<.0001
Married	22,602 (74.1)	2,080 (18.0)	4,459 (42.6)	2,272 (84.3)	669 (49.1)	
Divorced/widowed	295 (1.0)	53 (.5)	82 (.8)	11 (.4)	14 (1.0)	
Single	7,602 (24.9)	9,451 (81.6)	5,935 (56.7)	413 (15.3)	679 (49.9)	
Health insurance, <i>n</i> (%)						<.0001
Private	21,222 (69.6)	3,258 (28.1)	2,579 (24.6)	1,648 (61.1)	373 (27.4)	
Public	5,676 (18.6)	6,147 (53.1)	4,640 (44.3)	328 (12.2)	402 (29.5)	
Self-pay	221 (.7)	199 (1.7)	164 (1.6)	46 (1.7)	33 (2.4)	
Other	3,380 (11.1)	1,980 (17.1)	3,093 (29.5)	674 (25.0)	554 (40.7)	
BMI at admission, kg/m ² , mean (SD)	29.9 (5.6)	31.7 (7.0)	30.3 (5.6)	27.9 (4.5)	30.2 (6.1)	<.0001
Prepregnancy BMI, kg/m ² , mean (SD)	24.1 (5.4)	26.1 (6.9)	24.4 (5.2)	22.0 (4.1)	24.2 (5.7)	<.0001
Gestational age at delivery, weeks, mean (SD)	38.9 (2.1)	38.5 (2.9)	38.8 (2.4)	38.9 (1.9)	38.4 (2.9)	<.0001
Gestational diabetes, <i>n</i> (%)	676 (2.2)	225 (1.9)	375 (3.6)	127 (4.7)	58 (4.3)	<.0001
Smoking during pregnancy, <i>n</i> (%)	2,084 (6.8)	611 (5.3)	332 (3.2)	51 (1.9)	69 (5.1)	<.0001
Birth weight, grams, mean (SD)	3279.6 (560.1)	3062.8 (642.9)	3221.1 (597.4)	3193.3 (511.0)	3128.5 (656.1)	<.0001

^a *P* for marital status, health insurance, gestational diabetes, and smoking during pregnancy were obtained from Chi-squared tests. Mean maternal age, BMI at admission, prepregnancy BMI, gestational age at delivery, and birthweight were compared using ANOVA tests.

RESULTS

An overview of the study cohort and description of the maternal characteristics by race/ethnicity are presented in Table 1. Asians/Pacific Islander women were oldest (29.2 ± 5.3 years) but had the lowest prepregnancy BMI (22.0 ± 4.1 kg/m²), whereas non-Hispanic Black women were youngest (22.7 ± 5.8 years) and had highest prepregnancy BMI (26.1 ± 6.9 kg/m²). Non-Hispanic White women were most likely to be privately insured (69.6%) and Hispanic women were least likely (24.6%). Asians/Pacific Islander women were most likely to be married (84.3%) and non-Hispanic Black women were least likely (18.0%). Non-Hispanic White women were also more likely to smoke (6.8%) and Asians/Pacific Islander women were least likely to smoke (1.9%).

Non-Hispanic Black women were more likely to enter pregnancy with chronic hypertension, exhibiting a 1.43-fold increased odds compared to non-Hispanic White women (Table 2). They also had lower odds of remaining normotensive during pregnancy (adjust-

ed odds ratio (AOR)=.85, 95% CI .78–.93). Overall, Hispanic women and Asian/Pacific Islanders were 1.22- and 1.55-fold, respectively, more likely to remain normotensive during pregnancy, than non-Hispanic White women. Relative to non-Hispanic White women, Asian/Pacific Islanders were 38% less likely to develop mild preeclampsia. Non-Hispanic Black women had higher odds of mild preeclampsia (AOR=1.26, 95% CI 1.10–1.45), severe preeclampsia (AOR=1.31, 95% CI 1.10–1.57) and superimposed preeclampsia (AOR=1.98, 95% CI 1.40–2.80). Rates of eclampsia were similar across race/ethnic groups, although the number of cases was small.

When stratifying by timing of delivery, the patterns of risk appeared similar as in the main analysis. Hispanic and Asian/Pacific Islander women had lower odds of developing any pregnancy related hypertensive disease compared to non-Hispanic White women (Table 3). These associations were stronger with earlier delivery compared to later delivery, although the confidence intervals overlapped and were not as robust in the women with deliveries <34 weeks due to smaller sample size. In non-Hispanic

Black women, the effect of early delivery was less clear, as we observed these women only had increased risk of hypertensive disorders with delivery at ≥ 34 weeks.

All results were similar when including women with missing prepregnancy BMI in the analyses, which indicated little selection bias due to missing covariate data. Also, the results remained similar when restricting the analyses to women with normal prepregnancy BMI, indicating that the results are not solely driven by differences in weight distributions by different race/ethnic groups (data not shown).

DISCUSSION

In this large, nationwide, contemporary cohort study with a diverse racial/ethnic obstetrical population, non-Hispanic Black women were more likely to begin pregnancy with chronic hypertension and to develop mild, severe or superimposed preeclampsia, while Hispanic women and Asians/Pacific Islanders were more likely to

Table 2. Prevalence and odds ratios of hypertensive diseases during pregnancy by race/ethnicity^a

	Non-Hispanic White <i>n</i> =30,499 (53.9%)	Non-Hispanic Black <i>n</i> =11,584 (20.5%)	Hispanic <i>n</i> =10,476 (18.5%)	Asian/Pacific Islander <i>n</i> =2,696 (4.8%)	Multiracial/Other <i>n</i> =1,362 (2.4%)
Normotensive					
<i>n</i> (%)	27,209 (89.2)	9,817 (84.8)	9,437 (90.1)	2,540 (94.2)	1,220 (89.6)
OR (95% CI) ^b	reference	.74 (.69-.80)	1.19 (1.09-1.29)	1.78 (1.50-2.10)	1.16 (.96-1.40)
AOR (95% CI) ^c	reference	.85 (.78-.93)	1.22 (1.12-1.33)	1.55 (1.31-1.84)	1.20 (.99-1.46)
Gestational hypertension					
<i>n</i> (%)	1,360 (4.5)	422 (3.6)	252 (2.4)	47 (1.7)	27 (2.0)
OR (95% CI) ^b	reference	.94 (.82-1.07)	.69 (.59-.80)	.54 (.40-.73)	.75 (.50-1.11)
AOR (95% CI) ^c	reference	.80 (.69-.92)	.66 (.57-.78)	.61 (.45-.83)	.71 (.47-1.06)
Mild preeclampsia					
<i>n</i> (%)	989 (3.2)	551 (4.8)	361 (3.5)	53 (2.0)	41 (3.0)
OR (95% CI) ^b	reference	1.48 (1.30-1.68)	1.16 (1.01-1.33)	.55 (.41-.73)	.96 (.69-1.34)
AOR (95% CI) ^c	reference	1.26 (1.10-1.45)	1.10 (.95-1.26)	.62 (.46-.82)	.92 (.66-1.29)
Severe preeclampsia					
<i>n</i> (%)	498 (1.6)	404 (3.5)	238 (2.3)	37 (1.4)	40 (2.9)
OR (95% CI) ^{ba}	reference	1.59 (1.35-1.86)	.91 (.76-1.10)	.88 (.62-1.24)	1.01 (.71-1.43)
AOR (95% CI) ^c	reference	1.31 (1.10-1.57)	.83 (.69-1.01)	.96 (.68-1.35)	.92 (.64-1.31)
Eclampsia					
<i>n</i> (%)	13 (.1)	14 (.1)	8 (.1)	1 (.1)	1 (.1)
OR (95% CI) ^b	reference	1.45 (.55-3.87)	1.09 (.38-3.09)	.80 (.10-6.41)	1.28 (.15-11.28)
AOR (95% CI) ^c	reference	.81 (.27-2.42)	.73 (.24-2.20)	.96 (.12-7.73)	.89 (.10-8.24)
Chronic hypertension					
<i>n</i> (%)	238 (.8)	203 (1.8)	77 (.7)	9 (.3)	14 (1.0)
OR (95% CI) ^b	reference	1.32 (1.06-1.66)	.53 (.39-.71)	.40 (.20-.78)	.61 (.34-1.08)
AOR (95% CI) ^c	reference	1.43 (1.11-1.84)	.66 (.48-.90)	.49 (.25-.96)	.67 (.37-1.22)
Superimposed preeclampsia					
<i>n</i> (%)	100 (.3)	120 (1.0)	53 (.5)	4 (.2)	11 (.8)
OR (95% CI) ^b	reference	1.84 (1.34-2.52)	.89 (.60-1.31)	.44 (.16-1.19)	1.17 (.60-2.30)
AOR (95% CI) ^c	reference	1.98 (1.40-2.80)	1.08 (.72-1.62)	.53 (.19-1.45)	1.32 (.66-2.66)
Unspecified hypertension					
<i>n</i> (%)	92 (.3)	53 (.5)	50 (.5)	5 (.2)	8 (.6)
OR (95% CI) ^b	reference	1.03 (.68-1.56)	.94 (.61-1.44)	.64 (.26-1.60)	1.02 (.46-2.26)
AOR (95% CI) ^c	reference	.87 (.55-1.37)	.95 (.61-1.49)	.79 (.32-1.99)	.95 (.42-2.13)

^a Multivariable logistic regressions were performed to obtain the odds ratios.

^b Models were adjusted for site.

^c Models were adjusted for maternal age, prepregnancy maternal body mass index, insurance, smoking, marital status, and site.

remain normotensive during pregnancy, compared with non-Hispanic White women. The racial/ethnic variation in patterns of severe preeclampsia and superimposed preeclampsia mirrored cardiovascular disease risks later in life, where studies have generally found higher odds of cardiovascular diseases in non-Hispanic Black women and lower odds in Asian and Hispanic women.⁴

In general, our results are consistent with findings from prior studies, which likewise showed that non-Hispanic

Blacks have a higher risk of pregnancy-induced hypertension and Asians/Pacific Islanders a lower risk of preeclampsia compared with non-Hispanic White women.^{5-12,14} Our findings of lower rates of gestational hypertension for non-Hispanic Black and Hispanic women, and women with other race/ethnicity, compared with non-Hispanic White women, corroborates a prior study from a single state that used discharge data.²⁷ Likewise, the studies yielded consistent findings that non-Hispanic Black women were specifically

at a higher risk for severe preeclampsia and superimposed preeclampsia.²⁷

One previous study found no difference in severity of gestational hypertensive diseases by race, but they only distinguished between African American and others regarding race/ethnicity.⁷ This finding was not surprising as we found that risks varied significantly among non-Hispanic Whites, Hispanic women and Asian/Pacific Islanders. The results of our study also differed from those reported in that 1996 study, as the rates of hypertensive diseases among the

Table 3. Odds of pregnancy related hypertensive disease by maternal race/ethnicity stratified by gestational age at delivery^{ab}

Maternal Race	Delivery <34 weeks (n=1,878; 3.4%)			Delivery ≥34 weeks (n=53,910; 96.6%)		
	n (%)	OR (95% CI) ^c	AOR (95% CI) ^d	n (%)	OR (95% CI) ^c	AOR (95% CI) ^d
Non-Hispanic White	228 (28.5)	reference	reference	2,724 (9.3)	reference	reference
Non-Hispanic Black	154 (26.3)	.89 (.70–1.14)	.85 (.62–1.16)	1,290 (12.1)	1.34 (1.25–1.44)	1.10 (1.01–1.20)
Hispanic	81 (22.1)	.71 (.53–.95)	.61 (.43–.86)	828 (8.3)	.89 (.82–.96)	.85 (.77–.93)
Asian/Pacific Islander	9 (16.7)	.50 (.24–1.04)	.54 (.25–1.15)	134 (5.1)	.53 (.44–.63)	.68 (.57–.82)
Multiracial/other	11 (15.5)	.46 (.24–.89)	.40 (.19–.84)	106 (8.4)	.89 (.73–1.09)	.87 (.70–1.08)

^a Analysis limited to normotensive women at the beginning of pregnancy (ie, women with chronic hypertension or superimposed preeclampsia were excluded from this analysis).

^b Multivariable logistic regressions were performed to obtain the odds ratios.

^c Models were adjusted for site.

^d Models were adjusted for maternal age, prepregnancy maternal body mass index, insurance, smoking, marital status, and site.

non-Hispanic Black women in our study were considerably higher: 4.7% vs 1.8% experienced mild preeclampsia and 3.6% vs .7% severe preeclampsia.

Previous studies have found varying risk of preeclampsia for Hispanic women, when compared to non-Hispanic women.^{9,10,12,14,15,23} Studies investigating gestational hypertension separately from preeclampsia found that Hispanic women were at a lower risk of developing gestational hypertension when compared to non-Hispanic women.^{15,23} We found that Hispanic women were more likely to remain normotensive during pregnancy (ie, they had overall decreased risk of developing any hypertensive disease), with a decreased risk specifically

for developing gestational hypertension or having chronic hypertension. Our results may differ from previous findings on preeclampsia because Hispanic nationality is associated with a varying prevalence of preeclampsia.¹⁴

Of note, the pattern of racial/ethnic differences for developing gestational hypertensive diseases that we observed were consistent with those observed in the development of cardiovascular diseases later in life.^{28,29} In the US adult population, non-Hispanic Black women have a higher risk for cardiovascular disease, while Asian and Hispanic women have lower risk, relative to non-Hispanic Whites.⁴ The more severe hypertensive disorders of pregnancy observed in non-Hispanic Black women may be related to their higher risk of developing cardiovascular disease later in life. Future work is needed to determine whether these racial associations are due to genetic or social/environmental causes, in order to direct preventive interventions earlier in life.

Timing of delivery has been found to be a marker of differential risk for later onset cardiovascular disease; earlier delivery is associated with greater risk.³⁰ In one study, women who developed preeclampsia and delivered early preterm had an 8.1-fold increased risk of death from cardiovascular disease, compared to a 1.65-fold risk in women who developed preeclampsia and delivered at term.³⁰ We found that compared to non-Hispanic White women, Hispanic and Asian/Pacific Islander women may

have been less likely to develop hypertensive diseases associated with earlier delivery compared to later delivery, and non-Hispanic Black women had increased risk only associated with later delivery. Our sample size regarding earlier deliveries was small, but these findings suggest that race/ethnicity may be one shared pathway for preeclampsia and later onset cardiovascular disease, but the association has etiologies independent of race/ethnicity as well.

Though the overall cohort had a large sample size, one of the biggest to date, our study was limited by low numbers of women in certain categories, especially for eclampsia (n=45). Few studies have considered the risk of this very serious pregnancy complication separately from preeclampsia, so it remains important to establish risk factors specifically for eclampsia. Our study is further limited by lack of information on the timing of gestational hypertensive disease diagnosis, which would have been useful to more precisely categorize diseases into early- and late-onset and would have strengthened the argument for differing disease etiologic pathways. Minority women may have had less access to health care, which could have resulted in being less likely to be diagnosed with hypertensive diseases or being diagnosed with a more severe preeclampsia due to delay in diagnosis. However, we note that pregnancy care for lower income women has better coverage than primary care in the United States, so the potential for

In general, our results are consistent with findings from prior studies, which likewise showed that non-Hispanic Blacks have a higher risk of pregnancy-induced hypertension and Asians/Pacific Islanders a lower risk of preeclampsia compared with non-Hispanic White women.^{5–12,14}

missed diagnosis is lower. Additionally, our data and analyses are based on the delivery hospital records which may capture the rare cases that were missed earlier in gestation. We found that non-Hispanic Black women were more likely to begin pregnancy with chronic hypertension and to develop any form of preeclampsia regardless of severity (eg, mild, severe or superimposed preeclampsia), so if anything our findings may be conservative with respect to diagnosis. One of the major strengths of our study was the ability to adjust for a range of confounding factors given the detailed information available from the patient chart. Our results generally persisted after adjusting for these patient-specific factors. We also used a contemporary study population, which ensured that the diagnoses of hypertensive disorders were based on the current diagnostic criteria of hypertensive disorders in pregnancy.³¹

In conclusion, the prevalence of gestational hypertensive diseases varied by race/ethnicity with similar patterns as for development of cardiovascular disease outside of pregnancy. Non-Hispanic Black women had higher odds of developing gestational hypertensive diseases and Hispanic women and Asian/Pacific Islanders had an overall decreased risk of hypertension during pregnancy compared to non-Hispanic White women. Knowledge of these racial/ethnic susceptibilities to gestational hypertensive diseases may help guide research to target interventions to decrease risk of development of cardiovascular disease after pregnancy.

ACKNOWLEDGMENTS

The data included in this paper were obtained from the Consortium on Safe Labor. Institutions involved in the Consortium include (in alphabetical order): Baystate Medical Center, Springfield, Mass; Cedars-Sinai Medical Center Burns and Allen Research Center, Los Angeles, Calif; Christiana Care Health System, Newark, Del; Georgetown University Hospital, MedStar Health, Washington, DC; Indiana University Clarian Health,

Indianapolis, Ind; Intermountain Healthcare and the University of Utah, Salt Lake City, Utah; Maimonides Medical Center, Brooklyn, NY; MetroHealth Medical Center, Cleveland, Ohio; Summa Health System, Akron City Hospital, Akron, Ohio; The EMMES Corporation, Rockville Md (Data Coordinating Center); University of Illinois at Chicago, Chicago, Ill; University of Miami, Miami, Fla; and University of Texas Health Science Center at Houston, Houston, Tex. The named authors alone are responsible for the views expressed in this manuscript, which does not necessarily represent the decisions or the stated policy of the NICHD.

The Consortium on Safe Labor was supported by the Intramural Research Program of the Eunice Kennedy Shriver National Institute of Child Health and Human Development, National Institutes of Health, through Contract No. HHSN267200603425C.

This research was presented as a poster at the Annual Meeting of the Society for Pediatric and Perinatal Epidemiologic Research, Minneapolis, Minnesota, June 26, 2012.

REFERENCES

1. Smith GC, Pell JP, Walsh D. Pregnancy complications and maternal risk of ischaemic heart disease: a retrospective cohort study of 129,290 births. *Lancet*. 2001;357:2002–2006.
2. Williams D. Pregnancy: a stress test for life. *Curr Opin Obstet Gynecol*. 2003;15(6):465–471.
3. Männistö T, Mendola P, Väärämäki M, et al. Elevated blood pressure in pregnancy and subsequent chronic disease risk. *Circulation*. 2013;127:681–690.
4. Burt VL, Whelton P, Roccella EJ, et al. Prevalence of hypertension in the US adult population. Results from the Third National Health and Nutrition Examination Survey, 1988–1991. *Hypertension*. 1995;25:305–313.
5. Miranda ML, Swamy GK, Edwards S, Maxson P, Gelfand A, James S. Disparities in maternal hypertension and pregnancy outcomes: evidence from North Carolina, 1994–2003. *Public Health Rep*. 2010;125:579–587.
6. Shen JJ, Tymkow C, MacMullen N. Disparities in maternal outcomes among four ethnic populations. *Ethn Dis*. 2005;15:492–497.
7. Samadi AR, Mayberry RM, Zaidi AA, Pleasant JC, McGhee Jr N, Rice RJ. Maternal hypertension and associated pregnancy complications among African-American and other women in the United States. *Obstet Gynecol*. 1996;87:557–563.
8. Knuist M, Bonsel GJ, Zondervan HA, Treffers PE. Risk factors for preeclampsia in nulliparous women in distinct ethnic groups: a

prospective cohort study. *Obstet Gynecol*. 1998;92:174–178.

9. Caughey AB, Stotland NE, Washington AE, Escobar GJ. Maternal ethnicity, paternal ethnicity, and parental ethnic discordance: predictors of preeclampsia. *Obstet Gynecol*. 2005;106:156–161.
10. Brown HL, Chireau MV, Jallah Y, Howard D. The “Hispanic paradox” an investigation of racial disparity in pregnancy outcomes at a tertiary care medical center. *Am J Obstet Gynecol*. 2007;197:197e7–9.
11. Mittendorf R, Lain KY, Williams MA, Walker CK. Preeclampsia. A nested, case-control study of risk factors and their interactions. *J Reprod Med*. 1996;41:491–496.
12. Yeo S, Wells PJ, Kieffer EC, Nolan GH. Preeclampsia among Hispanic women in a Detroit health system. *Ethn Dis*. 2007;17:118–121.
13. Sibai BM, Ewell M, Levine RJ, et al. Risk factors associated with preeclampsia in healthy nulliparous women. The Calcium for Preeclampsia Prevention (CPEP) Study Group. *Am J Obstet Gynecol*. 1997;177:1003–1010.
14. Gong J, Savitz DA, Stein CR, Engel SM. Maternal ethnicity and pre-eclampsia in New York City, 1995–2003. *Paediatr Perinat Epidemiol*. 2012;26:45–52.
15. Wolf M, Shah A, Jimenez-Kimble R, Sauk J, Ecker JL, Thadhani R. Differential risk of hypertensive disorders of pregnancy among Hispanic women. *J Am Soc Nephrol*. 2004;15:1330–1338.
16. Grimes DA. Epidemiologic research using administrative databases: garbage in, garbage out. *Obstet Gynecol*. 2010;116:1018–1019.
17. Thadhani R, Mutter WP, Wolf M, et al. First trimester placental growth factor and soluble fms-like tyrosine kinase 1 and risk for preeclampsia. *J Clin Endocrinol Metab*. 2004;89:770–775.
18. Phillips JK, Janowiak M, Badger GJ, Bernstein IM. Evidence for distinct preterm and term phenotypes of preeclampsia. *J Matern Fetal Neonatal Med*. 2010;23:622–626.
19. von Dadelszen P, Magee LA, Roberts JM. Subclassification of preeclampsia. *Hypertens Pregnancy*. 2003;22:143–148.
20. Publications Committee, Society for Maternal-Fetal Medicine, Sibai BM. Evaluation and management of severe preeclampsia before 34 weeks’ gestation. *Am J Obstet Gynecol*. 2011;205:191–198.
21. Sibai BM. Management of late preterm and early-term pregnancies complicated by mild gestational hypertension/pre-eclampsia. *Semin Perinatol*. 2011;35:292–296.
22. Valensise H, Vasapollo B, Gagliardi G, Novelli GP. Early and late preeclampsia: two different maternal hemodynamic states in the latent phase of the disease. *Hypertension*. 2008;52:873–880.

23. Zhang J, Troendle J, Reddy UM, et al. Contemporary cesarean delivery practice in the United States. *Am J Obstet Gynecol*. 2010;203:326e1–10.
24. Interagency Committee for the Review of the Racial and Ethnic Standards. Review of the Racial and Ethnic Standards to the OMB Concerning Changes. 1997. whitehouse.gov/omb/fedreg_directive_15. Accessed March 13, 2014.
25. Simon E, Caille A, Perrotin F, Giraudeau B. Mixing nulliparous and multiparous women in randomised controlled trials of preeclampsia prevention is debatable: evidence from a systematic review. *PLoS One*. 2013;8:e66677.
26. Zhang J, Branch DW, Ramirez MM, et al. Oxytocin regimen for labor augmentation, labor progression, and perinatal outcomes. *Obstet Gynecol*. 2011;118:249–256.
27. Tanaka M, Jaamaa G, Kaiser M, et al. Racial disparity in hypertensive disorders of pregnancy in New York State: a 10-year longitudinal population-based study. *Am J Public Health*. 2007;97:163–170.
28. Kestenbaum B, Seliger SL, Easterling TR, et al. Cardiovascular and thromboembolic events following hypertensive pregnancy. *Am J Kidney Dis*. 2003;42:982–989.
29. Bellamy L, Casas JP, Hingorani AD, Williams DJ. Pre-eclampsia and risk of cardiovascular disease and cancer in later life: systematic review and meta-analysis. *BMJ*. 2007;335:974.
30. Irgens HU, Reisaeter L, Irgens LM, Lie RT. Long term mortality of mothers and fathers after pre-eclampsia: population based cohort study. *BMJ*. 2001;323:1213–1217.
31. National High Blood Pressure Education Program. Working Group Report on High Blood Pressure in Pregnancy. 2001. www.nhlbi.nih.gov/guidelines/archives/hbp_preg/. Accessed March 13, 2014.

AUTHOR CONTRIBUTIONS

Study design and concept: Ghosh, Grewal, Männistö, Xie, Laughon

Acquisition of data: Ghosh, Laughon

Data analysis and interpretation: Ghosh, Grewal, Männistö, Mendola, Chen, Xie, Laughon

Manuscript draft: Ghosh, Grewal, Männistö, Mendola, Chen, Laughon

Statistical expertise: Ghosh, Mendola, Chen, Xie, Laughon

Administrative: Mendola, Laughon

Supervision: Grewal, Laughon