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Impact of Pregnancy-Induced Hypertension on Stillbirth and Neonatal Mortality in First and Higher Order Births: A Population-Based Study

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Abstract

Background—Hypertensive disorders of pregnancy are more frequent in primiparous women, but may be more severe in multiparas. We examined trends in pregnancy-induced hypertension (PIH)-related stillbirth and neonatal mortality and explored whether mortality varied by parity and maternal race.

Methods—We carried out a population-based study of 57 million singleton live- and stillbirths (24-46 weeks) in the United States between 1990 and 2004. We estimated rates and adjusted odds ratio (OR) of stillbirth and neonatal death in relation to PIH, comparing births in 1990-91 with 2003-04.

Results—PIH increased from 3.0% in 1990 to 3.9% in 2004. In both 1990-91 and 2003-04 periods, PIH was associated with an increased risk of stillbirth and neonatal death. We explored this in more detail in 2003-04, and observed that the increased risk of stillbirth was higher in women having their second or higher order births (OR=2.24, 95% confidence interval (CI)=2.11-2.37) compared with women having their first birth (OR=1.52, 95% CI=1.40-1.64). Patterns were similar for neonatal death (OR=1.30, 95% CI=1.18-1.43 in first and OR=1.64, 95% CI=1.51-1.78 in second or higher order births). Among multiparas, the association between PIH and stillbirth was stronger in Blacks (OR=2.93, 95% CI=2.66-3.22) than Whites (OR=1.98, 95% CI=1.83-2.14).

Conclusions—A substantial burden of stillbirth and neonatal mortality is associated with PIH, especially among multiparas women, which may be due to more severe disease women, or to a higher burden of underlying disease.

Hypertensive disorders of pregnancy complicate 5-8% of pregnancies and are associated with increased risks of perinatal morbidity and mortality,[1-3] and maternal morbidity.[4] Preeclampsia, part of the spectrum of pregnancy-induced hypertension (PIH), is typically a disease of the first pregnancy, with a reduction in incidence among multiparas.[5] The occurrence of PIH in one pregnancy is a strong predictor of recurrence in the next pregnancy,[6-9] and recurrent hypertensive disorders is associated with substantially higher risks of adverse perinatal outcomes.[10]

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A study of first births in Norway between 1967-03 showed that the risk of stillbirth in relation to preeclampsia declined substantially between the periods 1967-78 (odds ratio 4.4) and 1991-03 (odds ratio 1.4).[11] The rate of births at <32 weeks among preeclamptic mothers tripled during the study period (from 1.6% in 1967-78 to 5.0% in 1991-03), but the decline in stillbirth was not paralleled by a substantial increase in post-natal death.11 Whether similar patterns are evident among multiparous women remains unexplored.

Rates of both preeclampsia and gestational hypertension have increased in the United States, [12] which underscores the importance of evaluating the burden of perinatal mortality associated with these conditions. Among women who had had preeclampsia in a previous pregnancy, severe gestational hypertension without proteinuria in the next pregnancy was associated with increased risk of preterm birth and small for gestational babies than among women who developed recurrent mild preeclampsia.[13] Hauth et al.[14] reported higher rates of infant and maternal morbidities in healthy nulliparas who developed severe hypertension or preeclampsia. These studies were, however, relatively small, and mortality could not be properly assessed.

Because limited attention has been devoted to pregnancy outcomes in multiparous women with hypertensive disorders of pregnancy, we investigated fetal and neonatal mortality in first and higher order singleton births in the United States. We compared births in 1990-91 to those in 2003-04, among Black and White women. These data, however, do not allow a distinction between preeclampsia and hypertension without proteinuria, and we thus used pregnancy-induced hypertension, comprising hypertension with and without proteinuria, as the entity of interest.

METHODS

Data source and composition of the analytic sample

We used the United States linked natality and infant mortality data for the period 1990-2004.[15] These data are based on birth and infant death certificates, and include data on maternal characteristics, medical and obstetrical complications, and fetal and infant outcomes. Infant deaths were not linked to the corresponding live births in the 1992-94 periods. To provide the most recent estimates, we focused our analysis on the period 2003-04, using 1990-91 as comparison.

Pregnancy-induced hypertension and perinatal mortality

The data include a check-box diagnoses of hypertension with or without proteinuria (i.e., PIH), as well as diagnoses of chronic hypertension and eclampsia, but a separate diagnosis of preeclampsia was unavailable. The 0.2% of women with a diagnosis of eclampsia was combined with the PIH group. Classification of preeclampsia diagnosis was modified in 1996 by the American College of Obstetricians and Gynecologists. The revision required that all cases meet criteria for hypertension in the presence of proteinuria after the 20th week of gestation.[16]

As PIH is rare in early pregnancy, we considered as stillbirths all registered fetal deaths with a gestational age of 24 weeks or higher. Neonatal deaths were defined as live births (from week 24 of gestation and onward) registered as having died within the first 28 days of life. Gestational age was based on the date of last menstrual period. If only the day of the menstrual period was missing, gestational age was statistically imputed by the National Center for Health Statistics prior to releasing the data.[17]

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Data exclusions

We restricted this analysis to singleton births. We excluded 1.1% (n=651,522) pregnancies with missing gestational age (i.e., missing month or year of menstrual date.) We also excluded 1.3% (n=773,935) of births with a gestational age <24 weeks or ≥47 weeks (n=104,724, 0.2%), and an additional 1.8% (n=972,718) of births with missing PIH status. After all exclusions, 56,987,638 singleton live births and 221,321 stillbirths remained.

Statistical analysis

We used logistic regression models to estimate odds ratio (OR) of stillbirth and neonatal death in relation to PIH, adjusted for geographic region, maternal age, education, race, and marital status, chronic hypertension and diabetes (categorized as shown in table 1.) To evaluate the recent impact of PIH on stillbirth and neonatal deaths, we restricted the majority of the analyses to the 2003-04 period. We performed all analyses separately for first and higher order births. In most analyses, we further categorized the latter group as second, third or fourth of higher order births. For 2003-04, we additionally estimated the OR of mortality following PIH in White and Black women.

Since maternal smoking, associated with a lower risk of preeclampsia[18-20] and higher risk of perinatal mortality,[21] is not reported in the California birth certificates, we performed a sub-analysis excluding California for the 2003-04 period, , to assess whether further adjustment for smoking altered the associations between PIH and mortality.

RESULTS

Women with PIH were more frequently younger (<20 years) or older (≥35 years) than women without PIH (Table 1). Rates of smoking were lower among women diagnosed with PIH, and a slightly higher proportion of black women than white women were diagnosed with PIH. PIH was reported in 5.3% and 2.8% of primiparous and multiparous women, respectively.

Differences in rates of stillbirth and neonatal mortality between women with and without PIH were greater in 1990-91 than in 2003-04 among both first and higher-order births (Table 2.) There was a higher rate of obstetrical interventions at preterm gestations in women diagnosed with PIH than in women without such diagnosis.

PIH was associated with a higher risk of stillbirth, especially among second and higher order births, both in the 1990-91 and 2003-04 periods (Table 3.) When birth order was more finely stratified, the risk of stillbirth in relation to PIH was fairly similar among women with their second, third or fourth or higher-order births. The risk of neonatal death following PIH was also elevated, although the difference between first and higher order births was less marked (Table 3.)

Blacks had consistently higher mortality rates, and multiparous Blacks had a higher OR of stillbirth following PIH compared with multiparous Whites (Table 4.) In 2003-04, rates of preterm obstetrical interventions among primiparous White and Black women without PIH were 3.6% and 5.4%, respectively, and 4.3% and 6.7% among multiparous Whites and Blacks, respectively. These rates were 4 to 5-fold higher among women diagnosed with PIH.

Trends in PIH and risks of stillbirth and neonatal mortality between 1990 and 2004 are shown in Figure 1 (appendix). When stratified by PIH status (Fig 2, appendix), the patterns of declining stillbirths and neonatal deaths were similar, although stillbirth showed a steeper decline among women with PIH.

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DISCUSSION

In this study, pregnancy-induced hypertension was associated with higher risks of stillbirth and neonatal mortality, especially in second and higher order births. Among normotensive births, on the other hand, rates of both stillbirth and neonatal death were similar between first and higher order births although, as expected, the risk was lowest in second births and highest in fourth or higher order births. Although absolute mortality rates have declined since 1990-91, odds ratios for mortality in relation to PIH changed little. Black women had higher absolute rates of stillbirth and neonatal death compared with Whites, but similar odds ratios of neonatal death. Among multiparas, Blacks had a higher odds ratio for stillbirth than Whites.

The substantial difference in PIH-associated mortality between first and later births may be due to more severe disease in multiparas, or to underlying maternal characteristics, including chronic diseases and, importantly, obesity.[22-25] For instance, Catov and colleagues[26] reported that these conditions, plus multiple pregnancy, obesity, and having had preeclampsia in the previous pregnancy accounted for over 50% of preeclampsia among multiparas.

It has been estimated that about 25% of preeclampsia cases in multiparous women occur in those with a prior preeclampsia,[27,28] and this proportion may be as high as 38% among women who experienced severe preeclampsia in a previous pregnancy.26 Overweight and obesity contribute significantly to preeclampsia,26,29-32 and prepregnancy body-mass index (BMI) is associated with increased blood pressure throughout pregnancy.[33] High BMI is also a risk factor for perinatal death,31,34,35 and women with the highest risk of stillbirth may thus not be those with a previous diagnosis of PIH but, instead, women with other risk factors, or with chronic diseases that were not evident in the first pregnancy. Type 2 diabetes, a risk factor for hypertensive disorders in pregnancy[36,37] and stillbirth38 may have also contributed to our results. Findings from a randomized trial showed that untreated gestational diabetes was associated with increased preeclampsia risk, but the association with perinatal death did not reach statistical significance.39 Obesity, diabetes, and subclinical maternal disease may be more common among multiparous women and could thus result in a different etiology and severity of preeclampsia in multiparas compared with primiparas.[40] In this analysis, despite adjustments for pre-existing hypertension and diabetes, the increased risk of stillbirth among multiparas with PIH persisted. It is possible that these conditions are not well registered in the US birth certificates, rendering adjustment ineffectual. In an analysis restricted to women with PIH, we also examined whether the increased mortality among multiparas could be explained by gestational age and birth weight; adjustment for these factors did not explain the findings (not shown).

Strengths and limitations of the data and consistency of associations

This analysis includes virtually all singleton births in the US, and we were able to adjust for a number of covariates. Although we only used smoking in a sub-analysis (due to sizeable missing data), the consistency in associations between the full analysis and those restricted to states that reported smoking data is reassuring. When we included a missing indicator for smoking, the results was again essentially unchanged (not shown.)

The main limitation of this analysis is that the diagnosis of PIH in our data did not permit distinctions between preeclampsia and gestational hypertension. The overall rates of PIH in our analysis were lower than those reported in a recent study,[12] suggesting the possibility of some under-reporting of PIH. A diagnosis of PIH is more likely to be accurate for babies who died than for babies who survived, and this may have biased the estimates away from the null. However, this mechanism is unlikely to explain the differences we observed between primiparas and multiparas. Furthermore, among primiparas, the estimated ORs were lower than those seen in Norway.[11] This may be because while the Norwegian study was based on

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preeclampsia, ours included women diagnosed with either preeclampsia or non-proteinuric hypertension. Thus, some attenuation of the association between PIH and mortality, given that PIH is likely a less severe disease than preeclampsia, is likely in our study.. If misclassification of PIH were non-differential, even a moderately low specificity would bias estimates towards the null, while low sensitivity would have little effect in case of a rare disease such as PIH. If severity of PIH influences the likelihood that a complication is recorded, the diagnosis we used may more closely reflect preeclampsia than gestational hypertension.

Errors in menstrual estimate of gestational age could have affected our comparisons to some extent.[41,42] However, we used gestational age for descriptive purposes, except when trying to assess whether it mediated the risk among strata of parity. The main analyses were not adjusted for gestational age, as it is an intermediate variable in the association between PIH and mortality.[11]

Deaths are generally accurately reported, although a small proportion remained unlinked in these data (<2%), and the distinction between stillbirth and (early) neonatal death may not always be clear-cut. However, these mechanisms are unlikely to have any substantial impact on our estimates. Residual confounding due to unmeasured factors may also have influenced our estimates to some extent.

Conclusions

Our findings suggest that women with second and higher order births following PIH have a higher risk of stillbirth than first-order births following PIH, particularly among Blacks. The elevated risk of mortality in multiparous women may be due to more severe disease or to the underlying characteristics of multiparas, and attempts should be made to explore this in studies where these predictors are available.

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Appendix

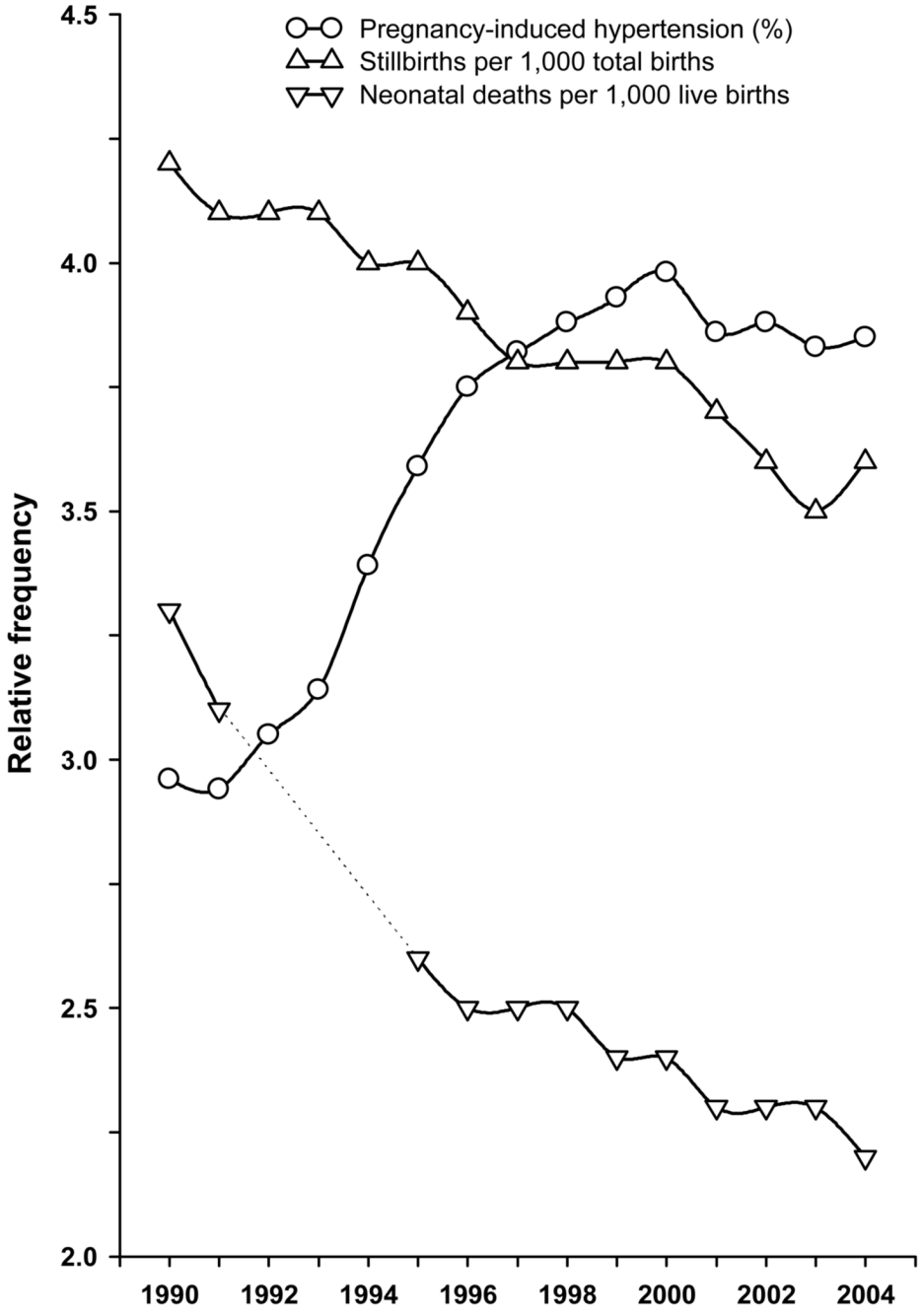

Figure 1.
Temporal trend in rates of pregnancy-induced hypertension, stillbirth, and neonatal mortality in the United States, 1990 to 2004 (neonatal deaths were not linked to the corresponding live births data file in 1992-94, and so are represented by dashed lines.)

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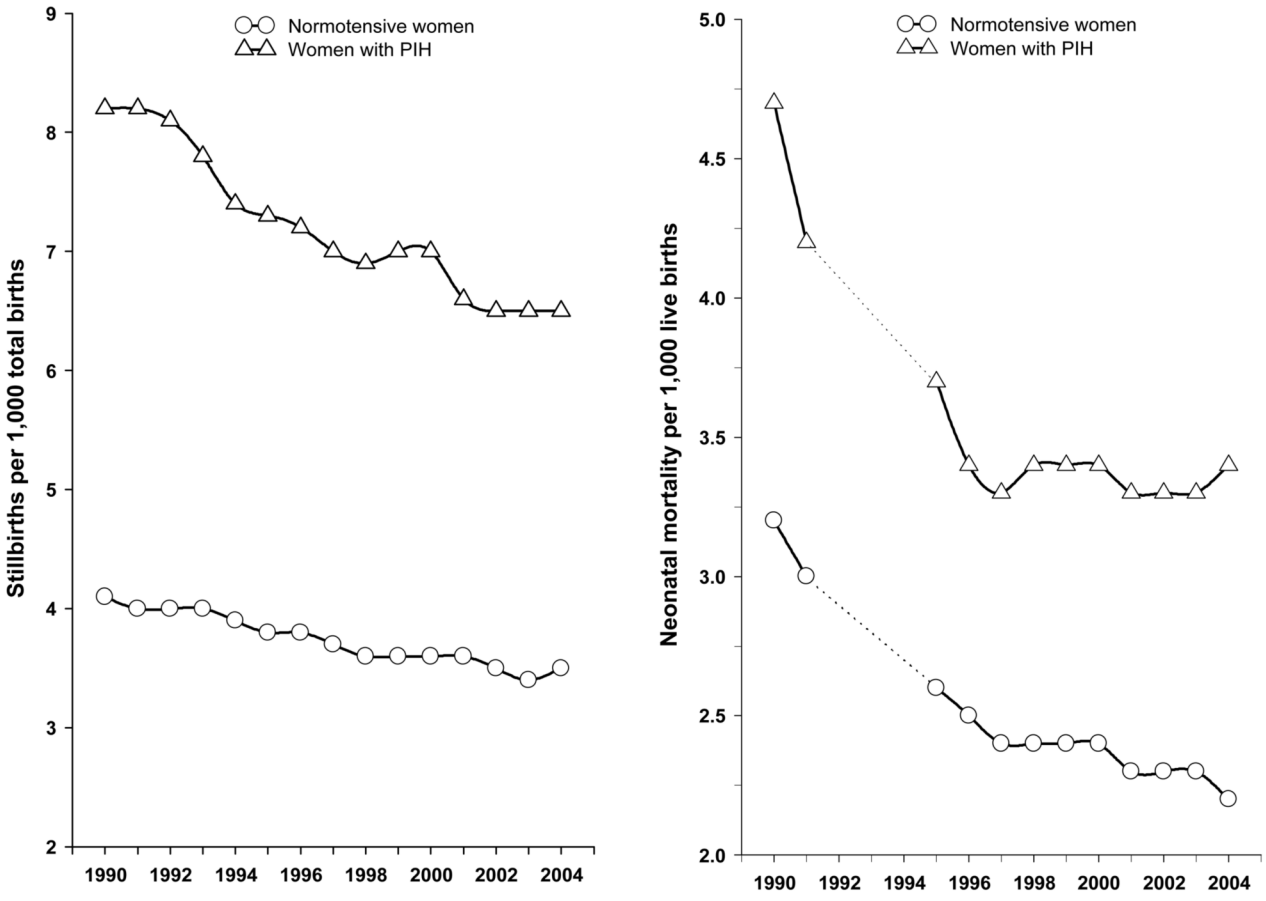

Figure 2.

Temporal trends in stillbirth (left panel) and neonatal mortality (right panel) in relation to pregnancy-induced hypertension status in the United States, 1990 to 2004 (neonatal deaths were not linked to the corresponding live births data file in 1992-94, and so are represented by dashed lines.)

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Table 1

Maternal socio-demographic characteristics and pregnancy-induced hypertension (PIH): United States 1990 to 2004

Maternal characteristics	Total population	Preg-induced hypertension	Normotensive Women	Missing data on PIH
Number of births	57,208,959	2,053,497	55,155,462	972,718
Geographic region				
Northeast	17.0	15.7	17.1	21.7
Midwest	22.7	23.9	22.7	10.8
South	35.9	41.1	35.7	53.3
West	24.4	19.4	24.6	14.2
Maternal age (years)				
<20	12.3	14.6	12.2	12.9
20-24	25.5	25.8	25.5	25.5
25-29	27.7	26.6	27.8	28.0
30-34	22.6	20.5	22.7	22.4
35-39	10.0	10.0	10.0	9.5
≥40	2.0	2.5	1.9	1.8
Education (years)				
<8	6.1	4.2	6.2	5.7
8-12	49.9	50.0	49.9	51.9
13-15	35.6	37.9	35.5	34.4
≥16	8.4	7.9	8.4	8.1
Primiparity	33.6	49.1	33.0	33.6
Single marital status	32.4	33.4	32.4	29.7
Maternal smoking†	13.7	11.0	13.8	15.3
Chronic hypertension	0.7	0.7	0.7	0.7
Diabetes	2.8	6.7	2.6	2.5
Maternal race				
Whites	79.0	79.2	79.0	78.4
Blacks	15.6	17.0	15.5	15.3
Other races	5.5	3.8	5.5	6.3

†Data from the state of California excluded since this state did not report smoking data Geographic regions were classified as Northeast (Maine, New Hampshire, Vermont, Rhode Island, Massachusetts, Connecticut, New York, New Jersey, and Pennsylvania), Midwest (Michigan, Ohio, Illinois, Indiana, Wisconsin, Minnesota, Iowa, Missouri, North Dakota, South Dakota, Nebraska, and Kansas), South (Delaware, Maryland, District of Columbia, Virginia, West Virginia, North Carolina, South Carolina, Georgia, Florida, Kentucky, Tennessee, Alabama, Mississippi, Arkansas, Louisiana, Oklahoma, and Texas) and West (Montana, Idaho, Wyoming, Colorado, New Mexico, Arizona, Utah, Nevada, Washington, Oregon, California, Hawaii, and Alaska.)

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Table 2

Pregnancy outcomes by pregnancy-induced hypertension (PIH) status: United States 1990-91 and 2003-04

	First births		Second or higher-order births	
	PIH	**Non-PIH**	**PIH**	**Non-PIH**
Total number of births				
1990-91	116,555	2,464,328	110,617	4,994,079
2003-04	142,342	2,483,383	158,094	5,031,247
Stillbirths per 1,000 total births				
1990-91	6.1	3.9	10.2	4.0
2003-04	4.8	3.2	7.8	3.4
Number of live births				
1990-91	115,840	2,454,658	109,493	4,974,191
2003-04	141,663	2,475,488	156,859	5,014,180
Neonatal deaths per 1,000 live births				
1990-91	4.2	3.1	4.7	3.1
2003-04	3.0	2.3	3.7	2.2
Birthweight (*g*), mean (standard dev)†				
1990-91	3,152 (729)	3,324 (556)	3,196 (768)	3,391 (577)
2003-04	3,034 (728)	3,284 (552)	3,083 (759)	3,356 (555)
Live births delivered at <37 wks (%)				
1990-91	17.0	9.4	18.0	9.6
2003-04	22.4	9.8	24.6	10.4
All births delivered at <37 wks (%)				
1990-91	17.3	9.6	18.5	9.8
2003-04	22.6	10.0	25.0	10.6
Live births delivered at <32 wks (%)				
1990-91	3.2	1.6	3.5	1.6
2003-04	4.0	1.5	4.3	1.4
All births delivered at <32 wks (%)				
1990-91	3.4	1.7	3.9	1.8
2003-04	4.2	1.6	4.6	1.5
Cesarean delivery (%)‡				
1990-91	42.5	22.5	38.1	20.6
2003-04	43.0	26.1	40.3	26.3
Labor induction (%)‡				
1990-91	36.4	10.3	30.0	9.1
2003-04	52.2	22.4	41.6	19.5
Preterm obstetrical intervention (%)*				
1990-91	12.4	2.5	12.8	2.9
2003-04	18.1	3.8	19.6	4.6

‡Cesarean delivery and labor induction includes interventions at all gestational ages

*Preterm obstetrical intervention includes labor induction, cesarean or both at preterm gestational ages

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Table 3

Association between pregnancy-induced hypertension and risks of stillbirth and neonatal death by birth order and period: United States 1990-91 and 2003-04

	Stillbirth per 1,000 total births			Neonatal deaths per 1,000 live births		
	PIH	**No PIH**	**OR (95% CI)**	**PIH**	**No PIH**	**OR (95% CI)**
			1990-91 period			
Number of births	n=227,172	n=7,458,407		n=225,333	n=7,428,849	
First births	6.1	3.9	1.37 (1.24-1.50)	4.2	3.1	1.32 (1.20-1.44)
Second or higher order births	10.2	4.0	2.20 (2.03-2.37)	4.7	3.1	1.46 (1.43-1.59)
Second births	8.6	3.3	2.19 (1.95-2.46)	4.2	2.7	1.49 (1.31-1.71)
Third births	9.8	3.7	2.31 (1.98-2.68)	4.5	3.0	1.46 (1.22-1.74)
Fourth or higher-order births	13.2	5.2	2.20 (1.19-2.51)	5.8	3.8	1.47 (1.25-1.71)
P-value for linear trend	<0.001	0.006		0.249	<0.001	
			2003-04 period			
Number of births	n=300,436	n=7,514,630		n=298,522	n=7,489,668	
First births	4.8	3.2	1.52 (1.40-1.64)	3.0	2.3	1.30 (1.18-1.43)
Second or higher order births	7.8	3.4	2.24 (2.11-2.37)	3.7	2.2	1.64 (1.51-1.78)
Second births	6.1	2.7	2.26 (2.05-2.49)	3.3	1.9	1.68 (1.47-1.91)
Third births	7.4	3.2	2.29 (2.04-2.58)	3.3	2.0	1.56 (1.31-1.85)
Fourth or higher-order births	12.7	5.4	2.30 (2.11-2.51)	5.0	2.9	1.68 (1.46-1.92)
P-value for linear trend	<0.001	<0.001		0.020	<0.001	

PIH, pregnancy-induced hypertension; OR, odds ratio; CI, confidence interval

Odds ratios are adjusted for geographic region, maternal age, maternal education, maternal race, marital status, diabetes, and chronic hypertension

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Table 4

Odds ratios of stillbirth and neonatal death following pregnancy-induced hypertension by birth order and maternal race: United States, 2003-04

	Stillbirth per 1,000 total births			Neonatal deaths per 1,000 live births		
	PIH	**No PIH**	**OR (95% CI)**	**PIH**	**No PIH**	**OR (95% CI)**
			White women			
Number of births	n=235,629	n=5,873,271		n=234,513	n=5,856,238	
First births	3.7	2.8	1.35 (1.22-1.49)	2.7	2.2	1.32 (1.18-1.48)
Second or higher order births	5.7	2.9	1.98 (1.83-2.14)	3.2	2.0	1.62 (1.46-1.80)
Second births	4.5	2.3	1.98 (1.75-2.24)	2.8	1.7	1.61 (1.38-1.89)
Third births	5.6	2.8	2.02 (1.74-2.35)	2.9	1.8	1.63 (1.32-2.01)
Fourth or higher-order births	9.1	4.6	1.94 (1.72-2.19)	4.1	2.5	1.63 (1.36-1.94)
P-value for linear trend	<0.001	<0.001		<0.001	0.099	
			Black women			
Number of births	n=50,484	n=1,101,405		n=49,779	n=1,095,283	
First births	10.9	5.3	2.11 (1.83-2.41)	4.5	3.7	1.20 (0.98-1.48)
Second or higher order births	16.1	5.7	2.93 (2.66-3.22)	6.1	3.6	1.64 (1.41-1.91)
Second births	13.0	4.8	2.68 (2.26-3.19)	5.6	3.3	1.67 (1.29-2.17)
Third births	15.5	5.2	3.14 (2.59-3.80)	5.2	3.4	1.48 (1.07-2.05)
Fourth or higher-order births	23.0	7.9	2.95 (2.58-3.37)	7.4	4.3	1.74 (1.39-2.19)
P-value for linear trend	<0.001	<0.001		<0.001	0.125	

PIH, pregnancy-induced hypertension; OR, odds ratio; CI, confidence interval

Odds ratios are adjusted for geographic region, maternal age, maternal education, marital status, diabetes, and chronic hypertension