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Differences in Hypertension Prevalence Among U.S. Black and White Women of Childbearing Age

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Synopsis

Hypertension and its sequelae complicate pregnancy and can result in poor perinatal outcomes. Overall, U.S. blacks are more likely to be hypertensive than whites, but the degree to which this is true among women of childbearing age (including teenagers) is unknown. Using data from the second National Health and Nutrition Examination Survey (NHANES II), the authors describe hypertension prevalence rates for 422 black and 2,700 white reproductive-age women.

The authors present observed data and also predicted prevalence rates derived by modeling the odds of hypertension using logistic regression statistical techniques. They find that black-white differences in hypertension prevalence are negligible among teenagers, but they are pronounced in the

older reproductive ages. They estimate that twice the proportion of black women relative to white are hypertensive during pregnancy.

Their results suggest that differential rates of hypertension between black and white women may

contribute to the persistent excess infant mortality among blacks, but conclusive results cannot be determined from these data. These data are also valuable for the design and evaluation of screening, intervention, and followup programs for hypertensive disease among young women.

HYPERTENSION and its sequelae complicate pregnancy and can lead to preterm labor and delivery, low birth weight, intrauterine growth retardation, abruptio placentae, and perinatal mortality (1-3). Hypertension prevalence is known to be greater among U.S. blacks relative to whites (4). Researchers and clinicians have been most interested in the differences that are apparent by middle age when hypertension leads to premature death and is noted as a risk factor for cardiovascular disease (5,6). However, the relative rates of hypertension for black and white teenage and young adult women have not been the explicit focus of research. In terms of the risks associated with childbearing, these are the ages of special interest. Improving our knowledge of the age patterns of hypertension risk among black and white young women may have implications for larger public health issues. For example, black-white differences in hypertension prevalence may be related to the large and persistent black-white differences in rates of low birth weight and infant mortality (7,8).

Existing estimates of hypertension prevalence for women 18 years of age and older show that black women exhibit higher rates than white, even at young adult ages (4). Yet, there is no analysis of the hypertension experience of reproductive age women that uses age breakdowns finer than 10-year divisions or that covers the teenage years, a period when many low-income and minority women begin childbearing (9).

Another unanswered question is whether the magnitude of the racial differential in hypertension prevalence is constant throughout the reproductive lifespan. There is suggestive evidence that it may increase in size over the childbearing ages (10). If this increase is indeed true, then the contribution of maternal hypertensive disease to the black-white infant mortality gap would depend not only on their relative hypertension prevalence rates, but also on the maternal age distribution of births for blacks relative to whites. In this study, we describe changes in hypertension prevalence rates among U.S. black and white menstruating women, ages 15 through 44, and discuss their potential impact on

pregnancy outcomes by estimating the proportion of black versus white women who would be expected to be hypertensive while pregnant.

Material and Methods

This study employed data from the National Health and Nutrition Examination Survey (NHANES II), 1976-80. This survey included a household interview and a medical examination for a sample of civilian, noninstitutionalized persons 6 months to 74 years of age (11). This data set has several advantages germane to our purposes: (a) it includes clinical examination data, laboratory test data, and survey data, providing us with clinical measures of hypertension as well as self-reports that can be linked to demographic and other background factors; (b) it is a large, nationally representative data set with an oversampling of blacks, allowing us to make national estimates of the prevalence of hypertension and to stratify these estimates; and (c) it provides information on teenagers as well as adults, enabling us to look at hypertension prevalence throughout the childbearing years.

The study sample included all currently menstruating 15-44-year-old women who were not pregnant at the time of the NHANES II survey and were racially identified as black or white. The racial identification of respondents was made by the NHANES II interviewers. Currently pregnant women were excluded from the sample we analyzed. We found there to be too few currently pregnant women to analyze (102 white, 18 black). We were also concerned about making comparisons in hypertension rates between pregnant and nonpregnant women or between pregnant women at different stages of gestation, given that pregnancy itself has effects on blood pressure that change during the course of pregnancy (12). The small number of pregnant women in the NHANES II sample suggests that their exclusion from the analysis is unlikely to have biased our results.

Women with missing data on current medicine usage or missing data on history of high blood

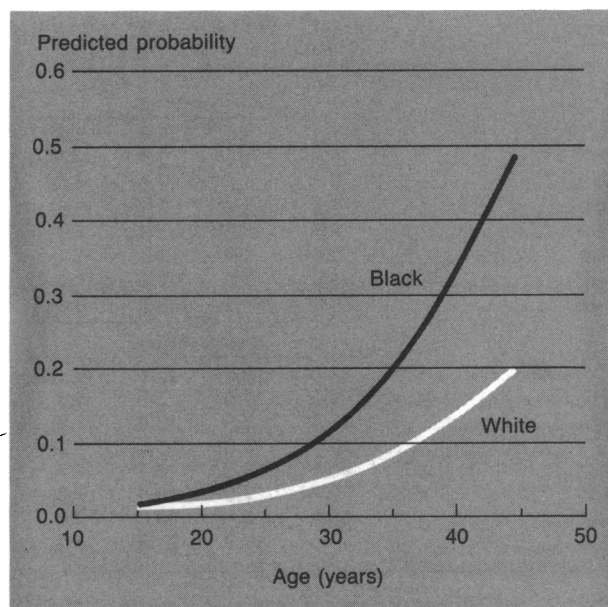
pressure were also deleted. A respondent had to have at least one valid systolic and diastolic reading to remain in the sample. Only 18 cases were lost to these restrictions. Missing data on parity were allowed with a variable indicating the presence of missing data included in prediction equations. After exclusions, the sample consisted of 2,700 white and 422 black women.

For NHANES II, blood pressure measurements were obtained using procedures recommended by the American Heart Association. Three readings were taken (sitting, recumbent, sitting). Averages of the available readings were used to determine clinical high blood pressure. This procedure deviates somewhat from those generally used to diagnose hypertension, since it is based on the average of several readings obtained on one occasion. The Joint National Committee on Detection, Evaluation, and Treatment of High Blood Pressure (JNC IV) recommends a diagnosis of clinical high blood pressure when blood pressure readings are elevated on two or more subsequent occasions (6). It is likely that not all persons with elevated readings on the occasion of the NHANES II examination would have a subsequent elevated reading (4). While this may lead to somewhat inflated measures of hypertension prevalence, there is no reason to believe that such inflation operates differentially between blacks and whites.

We constructed two definitions of hypertension prevalence for our analyses. The first, which we will henceforth refer to as our "narrow definition," included women who at the NHANES II examination exhibited clinical high blood pressure [defined as mean arterial pressure (MAP) greater than 105, where $\text{MAP} = (\text{systolic} + 2 \times \text{diastolic}) \div 3$] (13) or were current users of antihypertensive medication. Combining a clinical measure with medicine usage is the standard and recommended way to estimate hypertension prevalence (4).

However, this standard approach may underestimate the risk of hypertension as a pregnancy complication, our primary interest, because it does not account for past episodes of hypertension, including pregnancy-induced hypertension (PIH). In an attempt to address this shortcoming, we constructed an "expanded definition" of hypertension. The expanded definition includes all women identified as hypertensive by the narrow definition plus any other women who report a positive history of hypertension. The rationale for this approach is that a woman who experienced PIH in an earlier pregnancy would report a history of hypertension although she currently neither registers an elevated

Figure 1. Predicted probability of being hypertensive—narrow definition



blood pressure reading nor is undergoing antihypertensive therapy. We tested this possibility in our analyses by estimating the association between parity (defined as a history of any previous birth) and the two definitions of hypertension, narrow and expanded.

We report our major results for both definitions. We report those for the narrow definition because it is the standard approach, while use of the expanded definition is a bit unorthodox. Caution must be exercised in the use of the self-reported hypertension history data, which may be subject to recall bias or to nonrandom differences between women based on their access to health services. Some women may have had the opportunity to have their blood pressure measured, while others have not. It is unclear in which direction recall bias would affect our estimates, and the resulting error may be random. The issue of noncomparable access to health services is likely to lead to the underestimation of excess hypertension among black relative to white women, as blacks are more likely to suffer medical underservice than whites (14,15).

We calculated the observed hypertension prevalence for the sample by race and by fine age gradations estimating standard errors, following the procedures suggested by the National Center for Health Statistics (NCHS) to account for the complex sample design. (NCHS, Division of Health Examination Statistics: "Analytic Guidelines and Reliability Criteria for Analyzing HANES Data, May 1989." Unpublished.)

Table 1. Observed percent prevalence of hypertension for U.S. reproductive age women by race and age¹

Age range (years)	Black women	Standard error	White women	Standard error
<i>Narrow definition</i>				
Total ²	11.3	2.0	4.8	0.6
15-17	0.0	...	1.9	0.7
18-19	0.0	...	1.5	0.8
20-24	5.8	3.2	1.1	0.6
25-29	8.7	4.2	3.4	1.0
30-34	9.8	5.5	6.4	1.1
35-39	29.0	9.2	10.2	1.6
40-44	36.0	11.1	14.2	3.6
<i>Expanded definition</i>				
Total ²	22.5	2.5	13.5	0.9
15-17	1.0	1.5	3.8	0.9
18-19	12.0	5.9	6.8	1.6
20-24	20.4	5.3	8.0	1.3
25-29	20.1	5.5	13.9	1.7
30-34	20.2	7.0	16.4	2.5
35-39	48.6	9.4	23.5	3.6
40-44	43.0	10.7	27.9	4.3

¹ Standard errors calculated taking account of the complex sample design. See text for details.

² Standardized to the white age distribution.

SOURCE: Authors' calculations based on data contained in NHANES II, 1976-80, public use data files from NCHS.

Table 2. Predicted odds of hypertension for black and white women by age with black to white odds ratios

Age (years)	Black women	White women	Black-white ratio	t statistic	P
<i>Narrow definition</i>					
15	0.015	0.010	1.54	0.64	.52
25	0.060	0.029	2.08	1.98	.05
35	0.239	0.085	2.82	3.92	.001
45	0.950	0.250	3.80	2.98	.003
<i>Expanded definition</i>					
15	0.084	0.051	1.63	1.40	.16
25	0.209	0.114	1.83	3.08	.002
35	0.522	0.255	2.05	3.53	.002
45	1.306	0.567	2.30	2.30	.02

SOURCE: Authors' calculations based on NHANES II, 1976-80, public use data files from NCHS.

NOTE: t-statistics were calculated assuming design effects of 1.31 for the narrow definition and 1.22 for the expanded definition.

For the tabulations based on white women, we used Taylor series methods (16-20). To calculate standard errors for the tabulations involving black women, we adjusted standard errors (calculated under the assumption of simple random sampling) using the design effects calculated for white women averaged across age groups. For the narrow definition, this procedure amounted to multiplying standard errors calculated under the assumption of random sampling by 1.31. For the expanded defini-

tion, standard errors were multiplied by 1.22. We chose age breakdowns that were consistent with those used in published vital statistics data on natality to facilitate comparisons of changes in hypertension prevalence by age and the maternal age distributions of births in the United States.

One problem with using fine age breakdowns is that it introduces instability in our estimates by reducing the number of women on which each estimate is based. Thus, we also fit logistic regression models to the data to predict the relationship of age to hypertension prevalence within each race and to test the relationship of parity to the two measures of hypertension. For the logistic regression models, age was measured as a continuous variable from age 15 through 44. Parity was a dichotomous variable defined as a history of any previous birth (yes or no). Using the results of the logistic regressions, we estimated odds ratios to assess age and race differences in the risk of hypertension.

Finally, to obtain an initial, crude assessment of the relative proportions of black or white women who would be expected to be hypertensive while pregnant, we multiplied the observed prevalence rates by age to the actual maternal age distributions for births to black or white women separately. Currently, no data set exists that would allow such an estimate to be made reliably in a more direct way. We obtained maternal age distributions by race from U.S. vital statistics data (9).

Results

Table 1 lists the percentage of sample members in each age group who are or have been hypertensive according to both the "narrow" definition (current hypertension or current antihypertensive treatment) and the "expanded" definition that includes those women who report a past history of hypertension even in the absence of current indicators of hypertension. In the observed data, for both definitions, the black-white differences, while unsubstantial in the early teens (or even with the disadvantage being to whites), clearly emerge by ages 20-24, with black women experiencing higher rates throughout the remainder of the childbearing years. By the oldest ages, somewhat more than one-third of the black women were currently hypertensive or under treatment for hypertension, with an additional 7 percent reporting a history of hypertension. For whites, only 14-28 percent were hypertensive at the oldest ages, depending on the definition used.

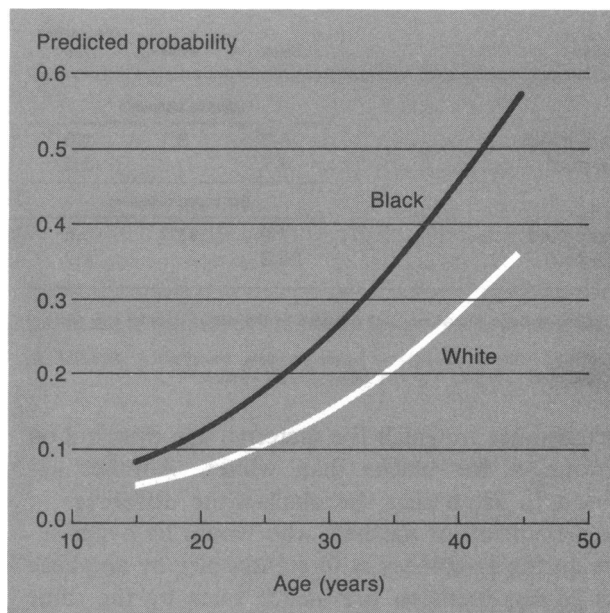
These results are summarized by estimating logistic regression models of the predicted odds of hypertension by age for each race. The results of fitting these models are depicted in figures 1 and 2, which graph the predicted prevalence of hypertension by age for blacks and whites using the narrow and expanded definitions. While the absolute levels of hypertension are higher for each group using the expanded definition, the age-race patterns are similar for the two measures. The general pattern is that hypertension prevalence increases with age for both races. However, the age gradient increase for blacks appears steeper; that is, the risk of hypertension appears to rise more rapidly with age for blacks compared with whites. The racial difference in the predicted odds of hypertension, thus, enlarges with age.

Using the logistic regression results, we report relative odds ratios (black to white) by age in table 2. These odds ratios also illustrate the enlargement of the black-white differences in hypertension prevalence with increasing age. While black-white differences were statistically insignificant among 15-year-olds, they increased in size and achieved statistical significance at conventional levels by age 25. By age 25, the black women were twice as likely as white women to be hypertensive or to have a reported history of hypertension. By the end of their childbearing years, black women were almost four times as likely as white women to suffer from objectively documented chronic hypertensive disease (the narrow definition).

Parity and Hypertension

To test the hypothesis that women who reported a history of hypertension, but were not currently hypertensive, included women with prior PIH, we modeled the odds of hypertension controlling for age and parity. Based on our hypothesis, we would expect the relationship between parity and hypertension to be stronger with the expanded definition of hypertension than with the narrow definition. In our logistic models, we found the effects estimates for parity were sensitive to the outcome measure used (narrow versus expanded). For both black and white women, the association between parity and the narrow definition of hypertension was small and insignificant. For black women, having borne a child raised the odds of being hypertensive by a statistically insignificant factor of 1.32 ($t=.47$, $P=.64$). For white women, having borne a child raised the odds of being hypertensive by a statistically insignificant factor of 1.26 ($t=.70$, $P=.48$).

Figure 2. Predicted probability of being hypertensive—expanded definition



However, there were strong associations for both groups and the expanded definition.

Both black and white women were more than twice as likely to report a history of hypertension in the absence of any indication of current hypertension if they had borne children than if they had not. For black women, the odds of reporting a history of hypertension was higher by a factor of 2.54 ($t=1.85$, $P=.08$) for those who had borne children compared with those who had not. For white women, the odds was higher by a factor of 2.74 ($t=4.21$, $P<.001$).

In table 3 we list the estimated proportion of pregnant women who were hypertensive by race and hypertensive measure (narrow or expanded) in 1980. Figures for the narrow definition are estimates of the proportion of women who would have started their pregnancies with preexisting chronic hypertensive disease. The expanded definition attempts to adjust for the fact that, using the narrow definition, we may have underestimated the proportion of women whose pregnancies would be complicated by hypertension, either chronic or pregnancy-induced. Using either measure, black mothers were far more likely to be hypertensive during pregnancy than white. Twice as many black as white women were estimated to begin their pregnancies with preexisting, chronic hypertension.

These numbers were estimated using the actual maternal age distributions of births to black and white women. As such, they represent the current

Table 3. Estimated percent prevalence of hypertension during pregnancy by race

Category	Black	White	Black-white ratio
<i>Narrow definition</i>			
Unadjusted	6.3	3.1	2.0
Adjusted ¹	7.7	...	2.5
<i>Expanded definition</i>			
Unadjusted	17.9	11.7	1.5
Adjusted ¹	20.3	...	1.7

¹ Estimated black prevalence rate adjusted to the white maternal age distribution.

SOURCE: Authors' calculations based on data contained in NHANES II, 1976-80, public use data files from NCHS and reference 9.

circumstance in which the maternal age distribution is younger for blacks than whites. Another approach to estimating the black-white difference in the proportion of mothers who would be hypertensive during pregnancy is to standardize by age both sets of hypertension prevalence rates by the same maternal age distribution. For this approach, we standardized to the white maternal age distribution of births. Estimating the proportion of black mothers who would have been hypertensive during pregnancy, under the assumption of the white maternal age distribution, led to small increases in this proportion and in the black-white difference in hypertension prevalence during pregnancy.

Comment

Our results indicate that racial differences in hypertension do in fact emerge among young women and intensify over the childbearing ages. In the United States, black women have higher predicted hypertension rates than white women at all adult ages, beginning in the teen years and leading to large differences during the predominant ages for childbearing. Although at age 15 there is essentially no black-white difference, by the middle of the childbearing ages black women are at least twice as likely as white women to be hypertensive.

The implications of hypertension for childbearing are clear. Both chronic hypertension and PIH are risk factors for poor pregnancy outcomes. Maternal hypertension is associated with increased rates of perinatal mortality, low birth weight, and prematurity (1-3). When we estimated the proportion of pregnant women who would be hypertensive, we found that although a small percentage of women would enter their pregnancies with preexisting hypertension, black women were twice as likely as white women to do so. When adjustments were

made to attempt to incorporate history of hypertension into our estimates, we found that more than 10 percent of white women and approaching 20 percent of black women would be likely to have some form of hypertension as a pregnancy risk factor. Standardizing hypertension prevalence rates by the white maternal age distribution led to modest increases in the estimates of the proportion of black mothers who would be hypertensive during pregnancy and in the black-white differential in estimated hypertension prevalence rates during pregnancy.

These estimates are probably conservative ones. The NHANES II data are a national sample of women, while women who are mothers (especially those who become mothers at young ages) are disproportionately members of low-income groups (21). As poverty and hypertension are known to be associated (22,23), at any given age, a larger proportion of pregnant women than all women may be hypertensive. Unfortunately, a data set that would permit more accurate estimation of hypertension prevalence during pregnancy (that is, one that includes a national sample of pregnant women and reliable data on their hypertension status) does not yet exist.

Racial differences in rates of low birth weight and infant mortality have been noted, and their magnitude of roughly 2 to 1 is equivalent to the size of the black-white differences in hypertension prevalence that we have demonstrated for reproductive-age women (7). Neonatal mortality among blacks has been observed to increase with increases in maternal age starting in the late teens (unlike whites, for whom large decreases have been observed between the teens and late twenties), and the black-white neonatal mortality differential has also been noted to increase with age (8). These maternal age patterns of neonatal mortality are similar to age patterns of hypertension prevalence among blacks and for blacks compared with whites, providing suggestive evidence that the hypertension experience and the infant mortality experience of blacks may be related.

One of us, ATG, has hypothesized (21) that black-white differences in maternal age patterns of neonatal mortality risk might, in part, be due to a more rapid deterioration of reproductive health status over the young adult ages among black women compared with white. This deterioration rate might reflect a "weathering" process; that is, that black women are exposed to the cumulative, physiological impact of social disadvantage as they age, and this exposure adversely affects their gen-

eral and reproductive health. The age patterns of hypertension prevalence that we report here support further consideration of the "weathering" hypothesis.

In a more indirect light, our results may have some application toward addressing the persistent question of whether PIH effects a woman's risk of future hypertension. Chesley and coworkers have reported that women who are eclamptic as primiparae do not have a higher risk of future death from hypertensive disease (24). Yet, Sibai and associates have noted an increased risk of chronic hypertension among women who have developed preeclampsia during pregnancy, suggesting that the disorders of PIH and later chronic hypertension may be linked (25).

Literature on the possible link between PIH and future development of chronic hypertension is limited by the lack of standard data for risk of hypertension by fine age gradations. For instance, followup studies of hypertension among preeclamptic and eclamptic women use broad age groupings for comparison (24,25). The data from this study provide a better mechanism to estimate baseline risk of hypertension by fine age gradations and permit a more accurate assessment of the significance of rates of hypertension in followup studies.

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