Demography, Vol. 27, No. 3, August 1990

Black/White Differences in Women's Reproductive-Related Health Status: Evidence From Vital Statistics

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Maternal-age–specific neonatal mortality risk differs by race, with the mid-20s risk low for whites but not blacks. This may be partially due to worsening health for black relative to white women. We analyzed deaths to young women in the aggregate and classified by causes that are also pregnancy risk factors. Over the predominant childbearing ages, mortality increases for blacks exceeded those for whites, usually by at least 25%. These indicators that black/white health differences widen as women progress through young adulthood suggest that such discrepancies may play a role in the black/white infant mortality differential, which merits further research.

Associations between maternal age and infant outcome have been thought to reflect an underlying age pattern of risk. Yet among first births in four states, Geronimus (1986, 1987) reported black/white differences in the distributions of neonatal mortality risk by maternal age. Not only were there differences in the absolute levels of neonatal mortality by race, but the age patterns of risk diverged. For whites the pattern clearly followed the familiar reverse-J-shaped curve with increasing maternal age that has often been found for the crude association between maternal age and poor infant outcome (Nortman 1974). The lowest risk points in this curve were observed in the mid- to late 20s. A different pattern described the black neonatal experience. Rather than following a smooth curvilinear function, above the age of 15, risk for blacks was almost uniform, with infants of mothers in their mid- to late 20s already exhibiting *higher* neonatal mortality rates than those with mothers in their late teens. In addition to questioning the importance of underlying maternal age patterns to the risk of infant morbidity and mortality, these findings provide possible clues for understanding black/white differences in infant mortality, and they imply that the magnitude of such differences increases with maternal age. If this is true, what are the mechanisms that drive it?

The persistence in the United States of black/white differences in infant mortality of roughly 2:1 remains an unsolved puzzle. Researchers have considered the role of socioeconomic background factors and access to prenatal care, but in so doing they have failed to account for the black/white differences satisfactorily. These remain at the level of the overall disparity even after controlling for typical sociodemographic and prenatal care variables (Geronimus 1986; Kleinman & Kessel 1987; Shiono, Klebanoff, Graubard, & Rhoads 1986). The divergent age patterns of risk suggest, however, that it is important to isolate the mechanisms that link sociodemographic background factors to infant outcome. As observed

by Mosely and Chen (1984), "social and economic determinants of child mortality necessarily operate through a common set of biological mechanisms, or proximate determinants, to exert an impact on mortality" (p. 25).

Given the observed age patterns of risk, an important focus for the discovery of such mechanisms is the changes by age in the health of women who become mothers or, more generally, in the health status of U.S. women of childbearing age. Many medical complications of pregnancy can have serious consequences for infant outcome (Burrow & Ferris 1988), and many of these complications are related to the mother's general health status and her experience with certain chronic and acute conditions and diseases *prior to* pregnancy. In the aggregate, the risk of many of these diseases is greater among the poor and blacks, and the etiology of many of these diseases includes social factors (for examples see the appendix, available from the authors). The treatment or management of these diseases before the onset of childbearing depends on access to and use of health services that, by definition, would predate prenatal care. Poor and minority American women suffer from disadvantages along many dimensions of access to health services, not only those directly related to pregnancy (Braveman, Oliva, Miller, Schaaf, & Reiter 1988). One set of proximate determinants of infant mortality that is theoretically worthy of inspection, then, is both the prevalence in the general population of women of chronic conditions and diseases that are also risk factors for poor pregnancy outcomes and any differences in access to those health services that if used could lead to cure or management of these diseases. Mortality from these diseases at teen and young adult ages provides at once an indicator of disease prevalance and medical underservice, since death so young is unusual and often preventable with appropriate medical intervention. In this article, by using mortality data, we consider U.S. black/white differences in both the absolute levels and age patterns of specific diseases among women that may have consequences for infant health. Specifically, this research is a first step toward testing the related hypotheses that (1) black/white differences in health status and medical underservice among women of childbearing age provide some of the mechanisms for racial differences in childbearing outcomes, and (2) black/white differences in health status increase rapidly within the childbearing ages, with the differential rate of declining health status evident by the mid- to late 20s.

Methods

For this initial test of the plausibility of our hypotheses, we chose as indicators of health status and medical underservice overall mortality among young adult women and mortality due to causes that are proximate determinants of poor infant outcome. Although, obviously, mortality statistics do not give us a direct measure of prevalence, we expect any excess mortality that we find for one race versus the other to be an indicator of higher prevalence rates. Furthermore, the alternative possibility that it reflects more extreme cases among one race versus the other or greater medical underservice is also consistent with the argument that one race suffers worse health status than the other in ways that could have implications for reproductive outcomes. In terms of medical underservice and the theoretical underpinnings of this investigation, mortality at young ages can be a marker for a curable, or at least manageable, disease gone undetected or left untreated and thus allowed to progress over time to a more pernicious form, a correlate of both aging and disadvantage.

For 1980–1985 (the most recent years for which data were available when we began this investigation), information was abstracted from national vital statistics data [National Center for Health Statistics (NCHS) 1985–1988] on deaths classified as due to these specific causes [numbers within parentheses are category numbers from the *International Classification of Diseases* (U.S. Dept. of Health and Human Services 1989)]: diabetes mellitus

(250), hypertension (401, 403), anemias (280–285), nephritis and other diseases of the urinary system (580–599), chronic liver disease and cirrhosis (571), viral hepatitis (070), pneumonia (480–486), diseases of the heart (390–398, 402, 404–429), chronic obstructive pulmonary diseases (490–496), and malignant neoplasms (140–208). These diseases were examined because each is a risk factor for pregnancy complications and can result in poor birth outcomes such as low birth weight (preterm birth or intrauterine growth retardation) and neonatal mortality. As a group, they cover the spectrum from chronic diseases to acute infections. They also include diseases (e.g., cirrhosis) that have behavioral roots (in this case alcohol consumption) and diseases that can result from the failure to detect or manage chronic diseases (such as heart disease, which can be a consequence of hypertension, or nephropathy, which can be a consequence of diabetes). The development of such diseases may indicate medical underservice. Given our interest in black/white differences, socioeconomic issues, and risks associated with increasing maternal age, we also picked diseases for which there is evidence of excessive prevalence among blacks or the poor or increasing prevalence or severity with age. [For the interested reader, we have written an appendix that reviews each death classification according to (1) its associated pregnancy complications and poor birth outcomes and (2) the salient social and behavioral issues it represents.]

Deaths in the aggregate (all causes) were also reviewed. The all causes category was considered in the aggregate as well as broken down into medical versus external causes. External causes represent primarily accidents and adverse effects (E800–E949) but also include suicide (E950–E959), homicide, legal intervention (E960–E978), and all other external causes (E980–E999). There were several reasons to look at these aggregate categories as well as to inspect deaths due to specific causes. First, we hypothesize a generally more rapid decline in health status among black compared with white women that reflects, in part, their relative medical underservice. This would be tested by using the all causes classification. Second, the specific reproductive-related causes include small numbers of deaths at the young ages of interest, lowering the reliability with which trends can be measured. This would not be the case for the all causes classification. The further breakdown of this category into medical versus external causes allowed us to observe the trends among physiological causes uncontaminated by clearly external causes and to note whether deaths due to physiological and external causes follow the same or different trends by race and age. If the trends are similar, this might suggest that any racial differences in deaths by medical cause may be related to social or behavioral differences by race. Finally, data on deaths classified as due to symptoms, signs, and ill-defined conditions (780–799) were also included as a check for evidence of any bias by age or race in reporting deaths by cause. The proportion of deaths classified to symptoms, signs, and ill-defined conditions is an index of the quality of medical certification of death (Rosenberg & Curtin 1986).

Death rates by cause were calculated for black and white women at the ages of 15–19 and 25–29 by using population estimates derived from the Current Population Reports (U.S. Dept. of Commerce 1987, ser. P-25). To calculate these 6-year rates, we divided the total number of deaths by the estimated total population at risk over 6 years in the relevant race/age category.[1] We estimated for each cause of death the relative odds of mortality for

1. black versus white women 15–19 years old,
2. black versus white women 25–29 years old,
3. black women 25–29 versus 15–19 years old, and
4. white women 25–29 versus 15–19 years old and
5. the relative rates of increase for black versus white women [the ratio of 3:4 (or 2:1)].

In each case 95% confidence intervals are also reported. The 95% confidence intervals for odds ratios and ratios of odds ratios were calculated by using formulas of the following general

form:

$$\exp\left[\hat{m} \pm 1.96 \sum_i \sum_j \frac{1}{D_{ij}(1 - r_{ij})}\right],$$

where $\hat{m}$ represents the relevant odds ratio or ratio of odds ratios, D_{ij} represents the number of deaths in the ith age group and jth racial group, and r_{ij} represents the mortality rate for the same group. In each case, summations occur only over the relevant groups.

Our first hypothesis—that black/white differences in health status among reproductive-aged women provide some of the mechanisms for black/white differences in childbearing outcomes—leads us to expect that mortality rates for black women will exceed those for white women by substantial amounts. Such differences would be noted through calculations 1 and 2. In particular, we expect there to be a difference when deaths associated with specific risk factors for poor pregnancy outcomes are considered. Our second hypothesis—that the gap widens with age (i.e., black/white differences in health status increase rapidly within the childbearing ages and are evident by the mid- to late 20s)—leads us to expect that the relative odds of dying rise more rapidly among black than white women (calculations 3–5). These data do not permit a direct test of our first hypothesis, as actual births are not included. The degree of consistency between our results and this hypothesis will suggest, however, whether this line of inquiry is worthy of continued pursuit.

Results

The results as summarized in the tables are consistent with the hypotheses. Table 1 reports death rates by race, age, and cause. Table 2 reports the relative odds of dying by cause for the relevant race/age categories. As shown in Table 1, at the ages of 15–19, death rates for most causes are higher for blacks than whites,[2] and at the ages of 25–29, death rates for all causes of death are higher for blacks than whites. Indeed, for several causes of death, the rates for 15- to 19-year-old blacks are higher than for 25- to 29-year-old whites. Almost all U.S. women bear children, white and nonwhite (Evans 1986). More than 75% of all births (including more than 87% of first births) in the United States occur to women between the ages of 15 and 29 (NCHS 1988). Taken together with our results, these facts suggest that racial differences in health status at these ages may be important factors explaining racial differences in pregnancy outcome. We also found that for both white and black women, roughly the same proportion (approximately 7%) of medical deaths are classified as arising due to ill-defined conditions. Thus it seems unlikely that any racial differentials in death rates can be accounted for by differentials in data quality.[3]

Table 1 suggests not only that death rates increase between these two young age groups but also that the increase with age is greater for blacks than whites, resulting in larger black/white differences at the ages of 25–29 than at the ages of 15–19.

The trends suggested by Table 1 are confirmed in Table 2. Although for some causes of death the pattern is more dramatic than for others, for every cause of death the trends in the odds ratios are consistent with the hypotheses. The odds ratio for 25–29/15–19 is greater for blacks than whites; the black/white odds ratio is greater at the ages of 25–29 compared with 15–19. The fifth column represents the relative black/white growth rate in the odds of dying. In most cases this ratio is well above 1, in terms of both its magnitude and its statistical significance, implying that death rates rise more rapidly for black than white women. A strong instance of this is seen for hypertension. White 25- to 29-year-olds have twice the rate of death by hypertension of white 15- to 19-year-olds, but for blacks the increase between these age groups is 14-fold. At the ages of 15–19, blacks and whites are basically equivalent in their odds of dying from hypertension, but by the ages of 25–29, the odds of dying from hypertension for blacks is almost 8 times that for whites. These results

Table 1. Death Rates for Women, 1980–1985, With Standard Errors

Causes of Death	White		Black	
	15–19	25–29	15–19	25–29
All causes	48.54	55.37	46.75	115.60
	(.32)	(.32)	(.73)	(1.19)
All medical	14.65	26.17	22.99	70.36
	(.17)	(.22)	(.51)	(.93)
External	33.89	29.19	23.76	45.24
	(.27)	(.24)	(.52)	(.74)
Diabetes	.20	.96	.32	1.66
	(.02)	(.04)	(.06)	(.14)
Hypertension	.02	.04	.02	.33
	(.01)	(.01)	(.01)	(.06)
Anemias	.12	.11	.83	1.74
	(.02)	(.01)	(.10)	(.15)
Urinary	.20	.42	.43	1.60
	(.02)	(.03)	(.07)	(.14)
Cirrhosis	.06	.69	.21	4.20
	(.01)	(.04)	(.05)	(.23)
Hepatitis	.10	.13	.20	.47
	(.01)	(.02)	(.05)	(.08)
Pneumonia	.48	.70	.83	2.45
	(.03)	(.04)	(.10)	(.17)
Heart disease	1.32	2.89	3.00	10.23
	(.05)	(.07)	(.19)	(.35)
Pulmonary disease	.26	.33	.83	1.31
	(.02)	(.02)	(.09)	(.13)
Malignancies	4.12	8.57	3.73	11.89
	(.09)	(.13)	(.21)	(.38)
Ill-defined	1.00	1.69	1.83	4.92
	(.05)	(.06)	(.15)	(.25)
% medical deaths classified as ill-defined	6.85	6.79	7.98	6.99

Note: Deaths per 100,000 population for selected cause. Standard errors are reported in parentheses.

are summarized by the ratios of the odds ratios, which show that the rate of increase for blacks over these ages is 7 times that for whites, as is the rate of the increase of the black/white difference in the odds of dying of hypertension. Other instances in which this pattern is marked include all causes and its subsets (all medical causes and external causes), anemias, pneumonia, heart disease, and ill-defined conditions. The weakest instances of this pattern are seen in the cases of diabetes and pulmonary disease.

In summary, the point estimates for 8 out of 10 specific causes of death (and for all aggregate categories) show increases in the odds of dying for black women that exceed those for white women by more than 50%, and 9 show increases for blacks that exceed those for whites by at least 25%. Only 1 of 10 causes—diabetes—shows rates of increase for black and white women of roughly comparable magnitudes. Even though the actual number of deaths observed for each of the 10 specific causes is quite small, the magnitude of the differences is large enough that for 6 out of 10 causes listed, the ratio of the odds ratios is sufficiently great that we can reject the null hypothesis that the growth in the black rate is no greater than the growth in the white rate at a 95% level. For 2 of the remaining 4 cases, one-tailed tests (which are appropriate given the hypothesis being tested) reject the null at

Table 2. Odds Ratios and 95% Confidence Intervals (CI) of Deaths, 1980–1985

Causes of death	25–29/15–19				Black/white				Relative b/w growth rate	
	White (1)	CI	Black (2)	CI	15–19 (1)	CI	25–29 (2)	CI	2/1	CI
All causes	1.14	(1.21, 1.16)	2.47	(2.38, 2.57)	.96	(.93, 1.00)	2.09	(2.04, 2.14)	2.17	(2.08, 2.26)
All medical	1.79	(1.73, 1.84)	3.06	(2.91, 3.22)	1.57	(1.50, 1.65)	2.69	(2.61, 2.77)	1.71	(1.62, 1.82)
External	.86	(.69, 1.08)	1.90	(1.81, 2.00)	.70	(.67, .73)	1.55	(1.50, 1.61)	2.21	(2.08, 2.34)
Diabetes	4.75	(3.83, 5.91)	5.15	(3.43, 7.74)	1.59	(1.05, 2.43)	1.73	(1.43, 2.09)	1.08	(.68, 1.72)
Hypertension	2.00	(.95, 4.22)	14.31	(3.40, 60.20)	1.10	(.24, 5.04)	7.90	(4.50, 13.85)	7.16	(1.42, 36.12)
Anemias	1.03	(.71, 1.48)	2.09	(1.57, 2.78)	7.09	(5.00, 10.05)	15.23	(11.26, 20.59)	2.15	(1.35, 3.40)
Urinary	2.06	(1.62, 2.62)	3.75	(2.61, 5.41)	2.11	(1.44, 3.07)	3.83	(3.09, 4.76)	1.82	(1.18, 2.82)
Cirrhosis	11.29	(7.74, 16.48)	20.21	(12.58, 32.47)	3.43	(1.90, 6.17)	6.13	(5.29, 7.10)	1.79	(.98, 3.28)
Hepatitis	1.28	(.88, 1.86)	2.37	(1.34, 4.20)	2.00	(1.15, 3.48)	3.70	(2.48, 5.52)	1.86	(.94, 3.68)
Pneumonia	1.47	(1.25, 1.73)	2.95	(2.25, 3.85)	1.74	(1.34, 2.27)	3.50	(2.94, 4.16)	2.01	(1.46, 2.75)
Heart disease	2.18	(1.99, 2.39)	3.41	(2.97, 3.92)	2.27	(1.96, 2.62)	3.54	(3.25, 3.85)	1.56	(1.32, 1.84)
Pulmonary disease	1.26	(1.00, 1.58)	1.56	(1.16, 2.10)	3.15	(2.36, 4.21)	3.96	(3.11, 5.03)	1.26	(.86, 1.83)
Malignancies	2.08	(1.97, 2.19)	3.19	(2.82, 3.62)	1.11	(.98, 1.24)	1.39	(1.30, 1.49)	1.54	(1.34, 1.76)
Ill-defined	1.77	(1.59, 1.98)	2.68	(2.23, 3.22)	1.83	(1.53, 2.19)	2.77	(2.46, 3.11)	1.51	(1.22, 1.88)

a 95% level. Equally important in supporting our hypothesis is the consistency of the results. The chances that at random all 10 of the causes of death would have shown greater increases for black as against white women is a trivial .001 ($.5^{10}$ = .00098).[4]

Discussion

These findings support our hypotheses and are noteworthy in several respects. First, that the trends by age and race for every cause of death considered are similar in direction, if not in magnitude, suggests that in the United States, black/white differences in health status widen rapidly during the childbearing years. That the trends in odds ratios by age and race follow similar patterns for both clearly medical causes of death and external causes of death may be an indication of the role of social factors even in the medical causes. Such findings suggest that the physiological mechanisms through which low socioeconomic status and medical underservice are translated into adverse pregnancy outcomes merit attention as investigators try to explain the excessive infant mortality rates among U.S. blacks. Identification of the mechanisms that link social factors to infant outcomes may be more fruitful than simply controlling for the usual socioeconomic indicators.

Our results are consistent with evidence that such diseases as hypertension and anemia may be important proximate determinants of the racial differences in birth outcomes. Each of these diseases has been identified as a risk factor for poor infant outcomes. Hypertension is associated with increased risk of pregnancy complications (preeclampsia, fetal death, and abruptio placenta) and with intrauterine growth retardation, preterm birth, and neonatal mortality (Ferris 1988; Lin, Lindheimer, River, & Moawad 1982; Sibai & Anderson 1986). Anemia is associated with low birth weight and preterm birth (Garn, Ridella, Petzold, & Falkner 1981; Klebanoff, Shiono, Berendes, & Rhoads 1989; Murphy, O'Riordan, Newcombe, Coles, & Pearson 1986). Hogue (1989) estimated that the attributable risk for preterm delivery among black infants of maternal anemia is 8.3%. Geronimus, Andersen, and Bound (1989) estimated that roughly twice as many black as white women enter their pregnancies with preexistent, chronic hypertension. Risk factors for these diseases include important social and behavioral components. Excessive hypertension prevalence is associated with low socioeconomic status (James, Strogatz, Wing, & Ramsey 1987; U.S. Dept. of Health and Human Services 1985) and a variety of behavioral factors, including poor diet (high cholesterol and high sodium), alcohol abuse, possibly smoking, and stress, both emotional and environmental (National Heart, Lung, and Blood Institute 1984). Prevalence rates of the predominant form of anemia in young women, iron deficiency anemia (Dallman, Yip, & Johnson 1984), are associated with poverty and are inversely related to educational level (Federation of American Societies for Experimental Biology 1985). In terms of behavior, poor diet (iron-deficient) is associated with increased risk. Death at young ages related to either of these diseases can be a marker of medical underservice. Blood pressure management has been shown to reduce drastically the risk of death from heart disease or stroke. Because the consequences of mild iron deficiency anemia are not readily apparent, detection and treatment are more likely if routine medical attention is available. If detected by simple laboratory testing, iron deficiency anemia is often easily treated with iron supplementation, averting the progression of the disease to more serious forms or death.

The two diseases that followed the hypothesized trends only weakly were chronic obstructive pulmonary disease (COPD) and diabetes. If one considers the course of these diseases in light of our theoretical framework, however, such findings are not surprising. The prevalence of the form of diabetes that is common at young adult ages—insulin-dependent diabetes (IDDM)—has not been demonstrated to be related to socioeconomic status (La Porte & Cruickshanks 1985; Wagenkneci, Roseman, & Alexander 1989), and the data relating socioeconomic status to the common form of COPD at young ages—asthma—

are conflicting (Evans et al. 1987; Higgins, Keller, & Metzner 1977; McWhorter, Polis, & Kaslow 1989). Forms of these diseases in which social and behavioral components have been more clearly implicated [non–insulin-dependent diabetes (NIDDM) and chronic bronchitis/emphysema] are more likely to be reflected in mortality statistics at later ages than we studied or than are relevant to childbearing. For example, most patients with NIDDM exhibit symptoms after the age of 40 (Amatruda 1987), and severe disability and death from chronic bronchitis/emphysema is most prevalent above the age of 55 (Welch 1982). Those diseases that more clearly followed the hypothesized trends than did diabetes or COPD were ones for which mortality at young adult ages has been more consistently linked to socioeconomic status, race, or other social and behavioral factors (for a review see the appendix, available from the authors).

Although our findings are highly suggestive, it is premature and incorrect to interpret them as demonstrating a measurable, direct link between the diseases we studied and black/white differences in infant outcomes. From a methodological standpoint, such demonstration awaits studies that use prevalence data and that estimate directly the effects of disease prevalence on infant outcome. Especially at the young ages of interest here, death by cause, even though an indicator of prevalence, is a conservative one. It most likely captures the most extreme cases, in terms of illness severity and medical underservice. Furthermore, an assumption is made here that trends in prevalence by age and race would follow trends in cause of death by age and race. This appears to be true in the case of hypertension, for which Geronimus et al. (1989) reported age/race patterns of hypertension prevalence among U.S. reproductive-aged women that are similar to those seen here in the mortality data. This assumption merits further testing on a broad array of conditions and diseases.

Finally, our findings are consistent with the theoretical view that aging, as it relates to reproductive outcome, may not be best thought of as only a process of biological maturation and deterioration but, rather, as an indicator of the length of exposure to life conditions that either undermine health (in the case of the disadvantaged) or promote it (in the case of the advantaged) (Geronimus 1987). We expect all women, black or white, to experience decreases in health status as they grow older, but we see indications here that the health of U.S. black women (who are more disadvantaged than white women as a group), as measured by their odds of death at very young adult ages, deteriorates more rapidly than that of white women. It appears a less plausible explanation that black women would, in their mid- to late 20s, already be aging in the biological developmental sense (i.e., experiencing senescence) than that their longer exposure to disadvantageous life conditions and medical underservice has impaired their health.

Notes

[1] This method of deriving death rates is the same as used in *Vital Statistics of the United States* (NCHS 1985–1988) when calculating single-year rates.

[2] The strongest exception to this is found among deaths due to external causes. Further inspection of the raw data reveals that black 15- to 19-year-olds are far less likely to die from accidents than white 15- to 19-year-olds and somewhat less likely to die from suicide than their white counterparts.

[3] Although reports on national data (Rosenberg & Curtin 1986) noted differences by race in data quality pertaining to the medical certification of deaths, we are not surprised that this is not the case here. Because our focus is limited to women who have died at very young ages, we assume that these women, whether black or white, fall at the low end of the health and socioeconomic status spectra and, therefore, may have uniformly poor medical certification. In addition, deaths at such young ages are rare and probably prompt careful determination of cause of death. Furthermore, young women are less likely than the elderly to suffer from multiple chronic conditions, and therefore, determination of their cause of death may be a relatively uncomplicated process. Thus there are several reasons to believe these data to be of good quality and any unreliability to pertain equally to blacks and whites.

[4] We can also infer from these results that the pattern demonstrated must hold for the residual (i.e., for the aggregate of the causes of death we did not study specifically). This fact is also consistent with the hypothesis that maternal health status deteriorates more rapidly among black than white women.

Acknowledgments

This work was done in the Department of Public Health Policy and Administration at the University of Michigan. Support was provided by National Institute of Child Health and Human Development Grant HD24122-01. An earlier version of this article was presented at the 1988 Annual Meeting of the American Public Health Association, Boston, Massachusetts. We would like to thank John Wilmouth and Barbara Anderson for helpful comments and Marianne Hillemeier for superb research assistance.

[*Received May 1989. Revised February 1990.*]

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