# RACE DIFFERENCES IN HYPERTENSION MORTALITY TRENDS

## DIFFERENTIAL DRUG EXPOSURE AS A THEORY

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### INTRODUCTION

Study after study has concluded that American Negroes have a greater incidence of hypertension and greater morbidity and mortality from hypertensive disease than American whites.[1]

Although there is much evidence to substantiate these differences, the causal factors have not yet been identified. Lennard and Glock advance four possible hypotheses: the genetic hypothesis, the physical exertion hypothesis, the psychosomatic hypothesis, and the associated disorder hypothesis.[2] None of these hypotheses suggests that differential medical care contributes directly to the higher rate of hypertension among Negroes. The absence of a differential medical care explanation is not surprising because until quite recently there was little medical science could offer the hypertensive patient to cure or arrest his illness. If a disease cannot be cured or effectively arrested, its relative incidence among Negroes and whites cannot be directly and appreciably influenced by inequality of medical care. Before 1948 this was probably true of hypertension. Since that year a whole battery of antihypertensive drugs has been introduced, and the contention is that these drugs can be quite effective in treating hypertension and reducing the mortality from it.[3] Hence, inequality

of medical care must now be considered a possible direct and important cause of differential rates of severe illness or mortality from hypertension.

The differential social and economic status of Negroes and whites in the United States almost certainly results in whites receiving the better medical care. Available data suggest that whites have had greater medical insurance coverage than nonwhites;[4] that white patients have made relatively greater use of hospitals than Negro patients;[5] and that whites have had access to more physicians in general and to more broad-certified specialists in particular than Negroes have had.[6]

Hypertensive white patients should be no exception to the contention that whites have received the better medical care, and, consequently, that they should have had greater exposure to the new antihypertensive drugs[7] than Negroes with hypertension.[8] If such a disparity in drug exposure has existed, and if the new drugs have been effective in reducing mortality from hypertensive diseases, it logically follows (other factors being equal) that the death rate from hypertensive disease should have declined since 1948, and the decline should have been greater for whites than Negroes—meaning that the Negro/white mortality ratio for this disease should have increased. The argument is that (regardless of the cause of any death-rate disparity between Negroes and whites in 1948) since 1948 differential exposure to antihypertensive drugs should have increased the Negro/white mortality ratio.[9] It is this differential exposure theory that led to the following study of mortality trends for hypertensive disease.

## METHODOLOGY

The National Office of Vital Statistics of the Public Health Service publishes annual death rates for selected causes by age, race, and sex for the United States. These death rates provided the raw data for the computations to be discussed. The years involved are 1949–1957 inclusive. This particular span of time was selected for theoretical and methodological reasons.

TABLE 1. NONWHITE/WHITE MORTALITY RATIOS

| Hypertension with Heart Disease | *Males*—*Age* 15–24 | 25–34 | 35–44 | 45–54 | 55–64 | 65–74 | 75–84 | 85+ |
|---|---|---|---|---|---|---|---|---|
| Year | | | | | | | | |
| 1949 | 2.92 | 7.88 | 7.39 | 5.16 | 3.50 | 2.42 | 1.52 | 1.12 |
| 1950 | 4.67 | 8.91 | 8.15 | 5.43 | 3.97 | 2.31 | 1.55 | 1.30 |
| 1951 | 3.00 | 9.17 | 7.91 | 5.13 | 4.00 | 2.42 | 1.59 | 1.17 |
| 1952 | 6.45 | 9.71 | 8.24 | 5.55 | 4.06 | 2.83 | 1.68 | 1.10 |
| 1953 | 6.00 | 11.67 | 8.97 | 5.75 | 3.96 | 2.73 | 1.62 | 1.20 |
| 1954 | 9.00 | 8.30 | 10.00 | 6.41 | 4.21 | 2.81 | 1.57 | .99 |
| 1955 | 8.00 | 12.14 | 10.75 | 7.04 | 4.46 | 3.06 | 1.66 | .99 |
| 1956 | 6.00 | 15.17 | 11.05 | 6.77 | 4.80 | 3.23 | 1.65 | 1.06 |
| 1957 | 10.00 | 14.33 | 11.45 | 6.30 | 4.86 | 3.32 | 1.74 | 1.09 |
| Hypertension without Mention of Heart Disease | | | | | | | | |
| Year | | | | | | | | |
| 1949 | 7.22 | 5.54 | 7.33 | 4.18 | 3.19 | 2.14 | 1.21 | 1.20 |
| 1950 | 3.50 | 7.75 | 6.63 | 4.36 | 3.26 | 2.06 | 1.29 | .68 |
| 1951 | 4.00 | 8.00 | 7.08 | 4.46 | 3.75 | 2.30 | 1.40 | 1.01 |
| 1952 | 3.19 | 11.25 | 7.37 | 4.49 | 3.69 | 2.41 | 1.50 | .97 |
| 1953 | 8.00 | 9.29 | 8.35 | 5.31 | 3.59 | 2.85 | 1.56 | .83 |
| 1954 | 6.00 | 9.00 | 10.28 | 5.86 | 3.50 | 2.62 | 1.29 | .89 |
| 1955 | 12.00 | 11.00 | 12.44 | 7.18 | 3.47 | 3.26 | 1.22 | .78 |
| 1956 | 5.00 | 14.00 | 12.40 | 6.19 | 4.72 | 2.89 | 1.34 | .52 |
| 1957 | 3.00 | 15.00 | 11.67 | 7.42 | 4.64 | 3.03 | 1.19 | .67 |

From a theoretical standpoint, it was necessary to focus on the years after the advent of the new antihypertensive (pharmacologic) agents. Veratrum, which began to be generally used for the treatment of essential hypertension in 1948, was apparently the first of these drugs to be introduced.[10] Hexamethonium and hydralazine were introduced a year or so later; rauwolfia (including reserpine) appeared about 1951; and many other antihypertensive agents have been added to the therapy spectrum since that time.[11]

From a methodological standpoint, it was impossible to study relevant trends for the years prior to 1949, and it would have been somewhat confusing to combine data for the 1949–1957 period and

TABLE 1. NONWHITE/WHITE MORTALITY RATIOS (CONTINUED)

| Hypertension with Heart Disease | *Females* 15–24 | 25–34 | 35–44 | 45–54 | 55–64 | 65–74 | 75–84 | *Age* 85+ |
|---|---|---|---|---|---|---|---|---|
| Year | | | | | | | | |
| 1949 | 8.47 | 11.46 | 10.61 | 6.55 | 4.28 | 2.39 | 1.31 | .89 |
| 1950 | 9.00 | 12.91 | 10.27 | 6.75 | 4.86 | 2.17 | 1.25 | 1.04 |
| 1951 | 5.33 | 11.92 | 11.19 | 7.05 | 4.82 | 2.29 | 1.36 | .96 |
| 1952 | 7.42 | 11.98 | 10.73 | 7.65 | 4.69 | 2.77 | 1.37 | .88 |
| 1953 | 12.00 | 10.22 | 13.22 | 7.47 | 4.87 | 2.65 | 1.46 | .92 |
| 1954 | 15.00 | 14.25 | 13.74 | 8.49 | 4.98 | 2.97 | 1.38 | .84 |
| 1955 | 7.00 | 21.00 | 14.65 | 9.01 | 5.42 | 3.20 | 1.35 | .79 |
| 1956 | 6.00 | 16.60 | 17.00 | 9.31 | 6.44 | 3.26 | 1.43 | .75 |
| 1957 | 6.00 | 17.60 | 18.17 | 10.30 | 6.51 | 3.43 | 1.45 | .75 |
| Hypertension without Mention of Heart Disease | | | | | | | | |
| Year | | | | | | | | |
| 1949 | 2.93 | 13.48 | 10.58 | 5.55 | 3.82 | 2.61 | 1.16 | .78 |
| 1950 | 3.50 | 9.14 | 9.29 | 6.65 | 5.70 | 2.00 | 1.15 | .86 |
| 1951 | 5.00 | 11.29 | 9.41 | 5.64 | 4.56 | 2.40 | 1.36 | .86 |
| 1952 | 12.60 | 12.96 | 9.29 | 6.58 | 4.52 | 2.95 | 1.35 | .74 |
| 1953 | 2.00 | 14.60 | 11.12 | 8.22 | 4.49 | 2.84 | 1.20 | .81 |
| 1954 | 8.00 | 16.00 | 14.08 | 9.97 | 4.01 | 3.21 | 1.41 | .92 |
| 1955 | 13.85 | 19.33 | 17.70 | 9.52 | 5.33 | 3.28 | 1.23 | .62 |
| 1956 | 6.00 | 15.67 | 17.33 | 8.78 | 4.95 | 3.10 | 1.43 | .79 |
| 1957 | 6.00 | 18.33 | 22.25 | 8.96 | 5.77 | 3.21 | 1.37 | .60 |

later years. The years 1949–1957 were covered by the Sixth Revision of the International Lists of Diseases and Causes of Death, under which hypertension with heart disease (categories 440–443) and hypertension without mention of heart disease (categories 444–447) were both specified as causes of death.[12] Before 1949 there was no grouping of causes comparable to hypertension with heart disease, and, hence, the Public Health Service claims that for this disease "it is not feasible to attempt to obtain trend figures prior to 1949."[13] For hypertension without mention of heart disease, the break in continuity was also great; and this disease accounted for so few deaths that even if Fifth Revision-Sixth Revision compar-

ability ratios were available by age-race-sex groupings, they would probably be unreliable. After 1957 the terms of the Seventh Revision apply, and one cannot compare 1957 and post-1957 death rates for hypertension without taking into account the relevant comparability ratios.[14] To avoid confusion, it was decided to treat the years 1949–1957 as a self-contained unit.[15]

The data gathering and computation procedure was as follows:

1. Death rates (per 100,000 population) for the United States as a whole for hypertension with heart disease and hypertension without mention of heart disease were collected or derived for white males, white females, nonwhite males, and nonwhite females[16] for eight age intervals.[17]

2. To obtain a comparative index for the death rates, a nonwhite/white death rate ratio was computed for each disease-sex-age-year subcategory.[18] A total of 288 ratios was computed (2 diseases × 2 sexes × 8 age categories × 9 years). One has only to scan these mortality ratios to see that in most instances there is a definite tendency for them to increase in magnitude over time (Table 1).

3. To summarize the trends in mortality ratios, a straight line of the form: log (nonwhite/white mortality ratio) = $\alpha + \beta t$ (where t is the year diminished by 1948) was fit by least squares for each disease-sex-age category. On the basis of the logs of the nine ratios,[19] three statistics were derived for each category: (a) an estimate of alpha ($\hat{\alpha}$), (b) an estimate of beta ($\hat{\beta}$), and (c) the estimated standard error of $\hat{\beta}$.

Both $\hat{\alpha}$s and $\hat{\beta}$s were used to plot the regression lines, but once the correctness of the linear model was established,[20] the $\hat{\beta}$s (i.e., the slopes) alone became the focus of concern because they are the measures of change over time in the nonwhite/white mortality ratios —the measures of direction of change and magnitude of change.

4. To interpret the trends in the mortality ratios, it was necessary to summarize the trends in the death rates themselves.[21] Three approaches were used. For each of the 48 disease-age-race-sex groupings $\hat{\beta}$s (i.e., slopes) were computed from the logs of the death rates for the nine years; Kendall rank correlation coefficients were com-

puted on the basis of the unlogged death rates for the nine years; and the death rates for the first and last years were compared according to the formula: (1957 death rate)/(1949 death rate). All three approaches measured direction of change. In addition, the slopes and the 1957/1949 data indexed magnitude of change,[22] and the Kendall taus indexed regularity of change.

## RESULTS OF THE DATA ANALYSIS

### *The Mortality Ratios*

There are 32 independent estimates of change over the period 1949–1957 in the nonwhite/white ratio of mortality from hypertensive disease.[23] These slopes (with their $t$ test indications of statistical significance) are set forth in Table 2. Of the 32 slopes, 26

TABLE 2. ESTIMATES OF CHANGE IN THE NONWHITE/WHITE RATIO OF MORTALITY FROM HYPERTENSIVE DISEASE FOR THE PERIOD 1949–1957 (THE SLOPES AND THEIR SIGNIFICANCE LEVELS)*

| | *Hypertension with Heart Disease* | | | | *Hypertension without Mention of Heart Disease* | | | |
|---|---|---|---|---|---|---|---|---|
| | *Male* | | *Female* | | *Male* | | *Female* | |
| Age | *Slope* | *Two-Tailed t Test Probability* | *Slope* | *Two-Tailed t Test Probability* | *Slope* | *Two-Tailed t Test Probability* | *Slope* | *Two-Tailed t Test Probability* |
| 15–24 | +.058 | <.01 | −.010 | >.20 | +.003 | >.20 | +.044 | >.20 |
| 25–34 | +.032 | <.01 | +.027 | <.05 | +.045 | <.001 | +.030 | <.01 |
| 35–44 | +.025 | <.001 | +.032 | <.001 | +.038 | <.001 | +.047 | <.001 |
| 45–54 | +.016 | <.01 | +.024 | <.001 | +.033 | <.001 | +.030 | <.01 |
| 55–64 | +.015 | <.001 | +.020 | <.001 | +.017 | <.02 | +.010 | >.20 |
| 65–74 | +.020 | <.001 | +.025 | <.001 | +.023 | <.001 | +.021 | <.02 |
| 75–84 | +.005 | <.02 | +.006 | <.05 | −.003 | >.20 | +.008 | <.10 |
| 85+ | −.008 | <.10 | −.015 | <.01 | −.027 | <.05 | −.013 | <.20 |

* The level of statistical significance used in this paper is the 5 per cent level.

are positive (22 significantly so) and six are negative (two significantly so). This definitely means that the trend was toward an increase in the nonwhite/white mortality ratios.

The two age categories at the extremes seem less reliable than the others: category 15–24 because it showed few deaths from hypertensive disease and category 85+ because population estimates for it disagree considerably.[24] Thus, the author has more confidence in data concerning the age range 25–84, and the discussion to follow will be limited to this range. Of the 24 slopes involved,[25] 23 are positive (21 significantly so) and one is negative (not significantly so). So in the more reliable age range the time trend was decisively toward an increase in the nonwhite/white mortality ratios.

The slope picture for the two hypertensive diseases and for the two sexes is essentially the same. Each disease and each sex has about the same number of significantly positive slopes, and there is no statistically significant difference (by a *t* test) between the diseases or sexes with respect to relative slope positiveness.[26] For example, in five of the 12 comparisons, the male slope is more positive than the female slope; in the other seven cases the reverse is true.

There is a definite tendency for the slopes to become less positive as age increases—meaning that as age increases there is less of an increase in the nonwhite/white mortality ratios. Kendall rank correlation coefficients (taus) were computed for each sex-disease grouping, as an index of the direction and regularity of the relationship between the six age ranks and the six slope ranks. All four taus are negative (i.e., as age increases, the slopes become less positive, their average being – .70, an average which is significantly different from zero at the .001 level).

*The Death Rates*

Over the period 1949–1957 there was a downward trend in the death rate from hypertensive disease. This pattern characterized every observation of whites and all but a few of nonwhites, regardless of the index used.[27] The death rate trends (as measured by each of the three approaches) are set forth in Table 3.

In 23 of the 24 white-nonwhite comparisons the whites show the

TABLE 3. MEASURES OF CHANGE IN THE DEATH RATES OVER THE PERIOD 1949–1957 SUBCLASSIFIED BY DISEASE, AGE, RACE, AND SEX

| Color, Sex and Age | *Hypertension with Heart Disease* | | | *Hypertension without Mention of Heart Disease* | | |
|---|---|---|---|---|---|---|
| | *Slope* | *Kendall tau* | *1957 (Death Rate)/ (1949 Death Rate)* | *Slope* | *Kendall tau* | *(1957 Death Rate)/ (1949 Death Rate)* |
| White male | | | | | | |
| 25–34 | −.101* | −.83* | .46 | −.085* | −.69* | .57 |
| 35–44 | −.087* | −.89* | .57 | −.083* | −.67* | .63 |
| 45–54 | −.085* | −.94* | .58 | −.080* | −.59* | .59 |
| 55–64 | −.071* | −1.00* | .61 | −.072* | −.92* | .58 |
| 65–74 | −.054* | −1.00* | .69 | −.040* | −.72* | .81 |
| 75–84 | −.052* | −.94* | .68 | −.039* | −.72* | .78 |
| White female | | | | | | |
| 25–34 | −.138* | −.78* | .42 | −.110* | −.67* | .60 |
| 35–44 | −.127* | −.92* | .43 | −.134* | −.76* | .42 |
| 45–54 | −.111* | −1.00* | .47 | −.115* | −.70* | .48 |
| 55–64 | −.081* | −1.00* | .56 | −.069* | −.89* | .58 |
| 65–74 | −.054* | −1.00* | .68 | −.051* | −1.00* | .71 |
| 75–84 | −.044* | −1.00* | .72 | −.052* | −.94* | .69 |
| Nonwhite male | | | | | | |
| 25–34 | −.028* | −.39 | .84 | +.017 | −.06 | 1.54 |
| 35–44 | −.029* | −.55 | .88 | +.003 | +.22 | .99 |
| 45–54 | −.048* | −.89* | .70 | −.004 | −.25 | 1.05 |
| 55–64 | −.036* | −.83* | .84 | −.032 | −.44 | .85 |
| 65–74 | −.008 | −.33 | .95 | +.013 | +.50 | 1.14 |
| 75–84 | −.040* | −.72* | .77 | −.045* | −.50 | .77 |
| Nonwhite female | | | | | | |
| 25–34 | −.073* | −.67* | .66 | −.038 | −.33 | .85 |
| 35–44 | −.053* | −.83* | .74 | −.024* | −.78* | .91 |
| 45–54 | −.055* | −.78* | .73 | −.045* | −.33 | .77 |
| 55–64 | −.034* | −.67* | .86 | −.045* | −.61* | .88 |
| 65–74 | +.003 | +.06 | .97 | −.004 | −.28 | .87 |
| 75–84 | −.031* | −.61* | .80 | −.033* | −.65* | .81 |

* Where there is an asterisk the change is statistically significant by the .05 standard. The two-tailed probabilities for the slopes were derived by a *t* test; those for the Kendall tau's were taken from Siegel;[28] no test of significance was applied to the 1957/1949 data.

more regular decline in the death rate;[29] also in 23 cases they show the greater decline in the death rate[30] ($p = < .001$).[31]

In 21 of the 24 interdisease comparisons hypertension with heart disease shows the more regular death rate fall ($p = < .001$); however, disease has no significant effect on degree of the death rate fall.

Sex has no significant impact on the regularity of decline when both diseases are considered together; but for hypertension without mention of heart disease alone, the females have the more negative tau in 10 of the 12 sex comparisons ($p = .038$). In 18 of the 24 comparisons the females show the greater drop in the death rate ($p = .022$). The pattern is again significant for hypertension without mention of heart disease alone, where the female fall is greater 10 out of 12 times ($p = .038$).

When age comparisons are made in 30-year steps,[32] age has no significant effect on regularity of death rate decline for both diseases considered together; however, for hypertension without mention of heart disease alone, the older age category has the more negative tau 10 out of 12 times ($p = .038$). It is the younger age categories that show the greater decline in the death rate. This is true in 20 of the 24 younger-older comparisons ($p = .002$). For hypertension with heart disease alone, the younger category shows the greater fall 11 out of 12 times ($p = .006$). [In sum, the death rate decline seems to have been more regular for the older age categories but more profound for the younger age categories.]

With respect to the 21 instances in which the nonwhite/white mortality ratio shows a statistically significant increase over time, the slope pattern for the death rates per se reveals that in 17 cases the death rate fell for both whites and nonwhites but more so for the whites.

In four cases the death rate fell for the whites and increased for the nonwhites.

## SUMMARY OF THE DATA

From the data at hand one can make the following summary statements:

1. Over the period 1949–1957 the nonwhite/white ratio of mortality from hypertensive disease *increased*. This trend applies to males and females and both forms of the disease, over the whole span of the age range from 25–84. There is no doubt of its statistical significance.
   a. The increase in the nonwhite/white ratio of mortality from hypertensive disease becomes less pronounced as age increases. This trend, too, is statistically significant.

2. Over the period 1949–1957 the death rate from hypertensive disease *decreased*. This trend applies to whites and nonwhites, males and females, and both forms of the disease, over the whole span of the age range from 25–84. There is no doubt of its statistical significance.
   a. In a statistically significant majority of sex comparisons, the females show a more profound decline of the death rate than the males.
   b. In a statistically significant majority of age comparisons, the younger age categories show a more profound decline of the death rate than do the older age categories.

## INTERPRETATION OF THE FINDINGS

This discussion focuses on two hypertensive disease trends—the decrease in the white and nonwhite death rates and the increase in the nonwhite/white mortality ratios.[33]

The theory that led to this research was set forth in the introduction to this paper. It argues that the hypotensive agents introduced since 1948 have been effective in reducing mortality from hypertensive disease and that whites have had greater exposure to these drugs than nonwhites. The white-nonwhite data discussed in this paper do not prove or disprove this theory, but they are definitely consistent with it. If it be assumed that the hypotensive agents introduced since 1948 have been effective in reducing mortality from hypertensive disease, and if it be assumed that white hypertensives have had greater exposure to these drugs than nonwhite hypertensives, then—other factors being equal[34]—it follows that:

1. The white death rate from hypertensive disease would have fallen since 1948.

2. The nonwhite death rate from hypertensive disease would have fallen since 1948,[35] but the relative decline would have been less than that for whites.

3. The nonwhite/white ratio of mortality from hypertensive disease would have increased since 1948.

All three of these trends are observed in the data for the 1949–1957 period discussed above. Since it was impossible to study the pre-1949 mortality trends for the hypertensive diseases, it is impossible to compare trends before and after the advent of antihypertensive drugs. One can only say that the post-1948 picture is consistent with the drug exposure theory—that the observed trends are necessary conditions for proving the theory, not sufficient conditions.[36]

The author is not the first to postulate that the antihypertensive drugs have reduced mortality from hypertensive disease;[37] however, the idea of differential (white-nonwhite) exposure is presumed to be an independent contribution. The contention that white patients with hypertension have benefited more from the availability of antihypertensive drugs than have nonwhites with the same disease is based on four interrelated propositions: That, on the average, whites with hypertensive disease of a given severity and type have been more likely to 1. visit a physician (or physicians) and utilize diagnostic tests for detecting their disease; 2. have a thorough and competent diagnosis and supervision of therapy; 3. have therapeutic drugs prescribed and be able to purchase them—particularly the most effective drugs; 4. follow the physician's orders in taking drugs —in continuing therapy and taking the prescribed dosage—than have been equivalent nonwhites.

These propositions were derived from a series of facts relevant to differential medical care. The argument is that the white-nonwhite differences referred to in the propositions led to the white-nonwhite mortality trend differences discussed in this article.[38] Several other interpretations of the mortality data have been considered, but none appears as tenable as the differential exposure theory. The author is currently examining these alternative hypotheses in more detail. Each is stated below and commented on briefly.

1. That over the period 1949–1957 there was a change in death certificate designations of cause of death (with respect to hypertensive disease) and that this change in diagnosis varied by race.

The argument here is that hypertension as a specified cause of death changed independently of hypertension as an actual cause of death. In light of the large northern and urban migration of nonwhites over this time period (a migration that could well have resulted in better medical care and diagnoses) and in light of a different migration pattern for whites, one has to acknowledge the possibility of a differential change in diagnosis. Such a factor may have contributed to the increase in the nonwhite/white mortality ratios to the extent that a higher proportion of nonwhites actually dying from hypertensive disease were so diagnosed than before[39]—but this could hardly explain the *decline* in the nonwhite death rate, let alone the *decline* in the white death rate.

To shed further light on this hypothesis, the author is examining the nonwhite/white mortality trends for other components of the cardiovascular disease complex.

2. That over the period 1949–1957 there was a differential (white-nonwhite) change in the incidence of hypertensive disease and, consequently, a differential change in the death rates.

The argument here would have to be that the incidence of hypertension decreased for both races but more so for the whites or that an increase in nonwhite incidence countered a drug-induced decrease in nonwhite mortality. Although this general idea is being considered, it seems more reasonable to assume that the incidence of hypertension among the two races remained constant over this period.

3. That over the period 1949–1957 there was a differential change in the accuracy of the population estimates for whites and nonwhites and that the death rates for the two races were accordingly affected.

It has been suggested to the author that nonwhite population estimates have improved with increasing urbanization and that this may have affected the nonwhite death rates. The fact is, however, that the white death rates changed more markedly than the nonwhite death rates; and so the author assumes that the change in mortality rates was not a result of changes in the accuracy of the population estimates.

4. That over the period 1949–1957 the antihypertensive drugs were more effective in the treatment of whites than nonwhites.

The idea here is that drugs accounted for the decline in mortality but that the race difference was caused by differential effectiveness rather than differential exposure. To shed light on this hypothesis, the author has been inspecting the literature dealing with trials of antihypertensive drugs on whites and nonwhites. The articles reviewed so far suggest that, given equality of illness, equality of use of drugs, etc., the drugs are equally effective in the treatment of whites and nonwhites.[40] Thus, it appears more valid to assume equality of drug effect than inequality of drug effect.

## SUMMARY

The author believes that the differential exposure-to-drugs theory is the best explanation of the differential (white-nonwhite) decline in hypertensive disease death rates over the 1949–1957 period. However, other possible interpretations are being considered.

## REFERENCES

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[2] Lennard, Henry L., and Glock, Charles Y., *op. cit.,* pp. 190–94.

[3] Dr. James V. Warren, past-president of the American Heart Association, has stated that " 'great improvement' in the control of hypertension through drugs" is a factor behind the sharp drops in deaths from strokes and hypertension since 1950 (Death Rate Drops in Stroke Cases, *The New York Times* (Western Edition), February 15, 1963, p. 5). On the general question of antihypertensive drugs and reduced mortality, *see* Dustan, Harriet P., et al., The Effectiveness of Long-term Treatment of Malignant Hypertension, *Circulation,* 18, 644–51, October, 1958; Kinsey, Dera, Sise, Herbert S., and Whitelaw, George P., Changes in Mortality Rates of Treated Hypertensive Patients in a Decade, *Geriatrics,* 16, 397–406, August, 1961; Perry, H. Mitchell, Jr., and Schroeder, Henry A., The Effect of Treatment on Mortality Rates in Severe Hypertension: A Comparison of Medical and Surgical Regimens, *Archives of Internal Medicine,* 102, 418–25, September, 1958; Smirk, F. H., Recent Developments in Hypertensive Therapy, *American Heart Journal,* 61, 272–81, February, 1961; Moyer, John H., and Brest, Albert N., Response to Blood Pressure Control in Patients with Hypertensive Vascular Disease, *The Nebraska State Medical Journal,* 47, 105–18, March, 1962; Smirk, F. H., Drug Therapy in Hypertension, *Clinical Pharmacology and Therapeutics,* 2, 110–20, January-February, 1961. Note also that the World Health Organization strongly recommends drug therapy in treating Stage 2 and Stage 3 of essential hypertension (Expert Committee on Arterial Hypertension and Ischaemic Heart Disease, Arterial Hypertension and Ischaemic Heart Disease: Preventive Aspects, WORLD HEALTH ORGANIZATION TECHNICAL REPORT SERIES, No. 231, p. 12, 1962).

[4] *See* Health Information Foundation, The People without Health Insurance, *Progress in Health Services,* 10, 1–2, October, 1961; Serbein, Oscar N., Jr., PAYING FOR MEDICAL CARE IN THE UNITED STATES, New York, Columbia University Press, 1953, p. 314.

[5] Cunningham, Robert M., Jr., Discriminatory Patterns, in Committee on Medical Care Teaching of the Association of Teachers of Preventive Medicine (editors), READINGS IN MEDICAL CARE, Chapel Hill, University of North Carolina Press, 1958, pp. 179–80.

[6] *See* Reitzes, Dietrich C., NEGROES AND MEDICINE, Cambridge, Mass., The Commonwealth Fund and Harvard University Press, 1958, pp. xxvii and 3; Simpson, George E., and Yinger, J. Milton, RACIAL AND CULTURAL MINORITIES: AN ANALYSIS OF PREJUDICE AND DISCRIMINATION, New York, Harper and Brothers, 1958, p. 422.

[7] The concept of differential exposure to drugs encompasses differential access and differential use.

[8] At least one research team has independently advanced a similar idea: "Despite the lack of good evidence, it is tempting to relate the decreased incidence of malignant hypertension to potent antihypertensive agents. The absence of any decrease among Negroes might favor this hypothesis, since Caucasoid patients would presumably be affected first by a new and relatively expensive therapy" (Perry, H. Mitchell, Jr., Caloyeropoulos, Angelos, and Moore-Jones, Dominic, Therapy of Severe Hypertension Complicated by Renal Failure, *The American Journal of Cardiology,* 9, 910, June, 1962).

[9] How long this mortality ratio might continue to increase is a question contingent on a whole host of variables including the relative effectiveness of newer drugs, possible changes in drug cost, the trickling down of therapeutic techniques to general practitioners treating Negroes, etc. The hypothesis guiding this inquiry was that the ratio would still be increasing as late as 1960 rather than leveling off or decreasing.

[10] Kinsey, Dera, et al., *op. cit.,* p. 401.

[11] *Ibid.;* Smirk, F. H., Recent Developments in Hypertensive Therapy, *op. cit.,* pp. 274–79; Grollman, Arthur, and Furness, Franklin N. (editors), New Diuretics and Antihypertensive Agents, *Annals of the New York Academy of Sciences,* 88, 771–1020, October 11, 1960.

[12] Many more deaths were allocated to the first disease than the second.

[13] United States Department of Health, Education, and Welfare: Public Health Service: National Center for Health Statistics, personal communication to the author, July 29, 1964.

[14] United States Department of Health, Education, and Welfare: Public Health Service: National Vital Statistics Division, Comparability Ratios for Selected Causes by Age, Color, and Sex, Based on Deaths from a 10-Percent Sample Assigned According to the Sixth and Seventh Revisions of the International Lists: United States, 1958, unpublished Vital Statistics, personal communication to the author, October 5, 1962.

[15] The data for 1958 on will be discussed in a separate paper and, where possible, the findings will be related to those for the 1949–1957 period.

[16] For the years involved it was impossible to separate death rates for Negroes and death rates for other nonwhites (Japanese, Chinese, Indians, Filipinos, and others). However, almost all nonwhites in the United States are Negroes (in 1950 the figure was 95 per cent); hence, the use of the category "nonwhites" in place of "Negroes" should not have distorted the results appreciably.

[17] Age intervals: 15–24, 25–34, 35–44, 45–54, 55–64, 65–74, 75–84, and 85 +.

The sources of the death rates for the years 1950–1951 and 1953–1957 were the volumes of *Vital Statistics* for these years. (United States Department of Health, Education, and Welfare: Public Health Service: National Office of Vital Statistics, *Vital Statistics of the United States, 1950,* Vol. I, Washington, D.C., U.S. Government Printing Office, p. 213; *ibid.,* 1951, Vol. I, p. XLIV; *ibid.,* 1953, Vol. I, p. LII; *ibid.,* 1954, Vol. I, p. LVI; *ibid.,* 1955, Vol. I, p. XCVII; *ibid.,* 1956, Vol. I, p. CV; *ibid.,* 1957, Vol. I, p. CXXV). Death rates for 1952 were computed for the above age intervals by using actual deaths from each of the two hypertensive diseases (*ibid.,* 1952, Vol. II, pp. 168–69) and the population for each subgroup (*ibid.,* Vol. I, p. XXVIII, Table K) as the basis for the computations. It was also necessary to compute the death rates for 1949. Actual deaths for appropriate disease-age-race-sex groupings were reported in VITAL STATISTICS for 1949 (Federal Security Agency, *ibid.,* 1949, Vol. II, pp. 256–57), but it was obvious that alternative population bases could be used (*ibid.,* Vol. I, p. XIII, Table F (or p. 38) and p. 37, Table XVIII). It was decided to use the latest population estimate available (*see* United States Department of Commerce: Bureau of the Census, Population Estimates, CURRENT POPULATION REPORTS, series P-25, no. 98, 7–11, August 13, 1954). A comparison of this estimate and that laid out by age, race, and sex in VITAL STATISTICS for 1949 (Federal Security Agency, *op. cit.,* Vol. I, p. 37, Table XVIII) revealed some rather large discrepancies. However, in the age range 25–84 (the age range on which this study focuses), the two estimates were quite close.

[18] It was decided that a ratio is better than a simple difference. Thus, a death rate of 10 versus a death rate of 20 compares to a death rate of 70 versus a death rate of 140, rather than a death rate of 70 versus a death rate of 80.

[19] Ratios for the nine years 1949–1957 inclusive.

[20] There was good linear regression and apparently homogeneous variance, ıdicating that the use of logs was a satisfactory measurement in these respects. lowever, there did appear to be a small (probably not important) cyclical pat-ern of variation around the regression lines, and further study is being made f this.

[21] It was obvious that any one of a number of white-nonwhite death rate end pairings could have produced the increase in the magnitude of the non-hite/white mortality ratios.

[22] The change expressed by the 1957/1949 comparison is more easily nderstood than that expressed by the slopes.

[23] 2 diseases × 2 sexes × 8 age categories = 32 observations.

[24] Category 85 + shows greater discrepancies between the two 1949 popu-ıtion estimates than does any other relevant age category (*see* United States )epartment of Commerce, *op. cit.*, and Federal Security Agency, *op. cit.*, Vol. I, . 37, Table XVIII).

[25] 2 diseases × 2 sexes × 6 age categories = 24 observations.

[26] Nor is there a significant difference between the sexes in this respect hen disease is held constant.

[27] All 24 white drops and the majority of the nonwhite drops are statis-ically significant. None of the upward trends is significant.

[28] Siegel, Sidney, NONPARAMETRIC STATISTICS FOR THE BEHAVIORAL CIENCES, New York, McGraw-Hill Book Co., Inc., 1956, p. 285.

[29] That is, they show the more negative tau.

[30] All these references to magnitude of death rate decline are references o the slope data rather than to the 1957/1949 data, because the slopes take ll nine years into account.

[31] The probabilities referred to here are probabilities associated with the ıarticular structure of the differences according to a two-tailed sign test.

[32] That is, age category 25–34 vs. age category 55–64, age category 35–44 s. age category 65–74, and age category 45–54 vs. age category 75–84.

[33] The age and sex patterns (*see* statements 1a, 2b, and 2a above) will not e interpreted here. Their explanation may hinge on certain assumptions about lifferential access to, use of, and effectiveness of the new antihypertensive gents. (For example, one could hypothesize that the younger age categories show he more profound death rate decline because a greater proportion of old than oung were beyond effective help when the drugs were introduced.) However, efore advancing a drug hypothesis to explain the age and sex patterns, the uthor wishes to study the data further and examine the relevant literature.

[34] The number of factors assumed to be equal is too extensive to be listed ere. Equality of disease incidence over time and equality of drug effect are wo of the important assumed equalities. They are discussed on page 212.

[35] Assuming that the nonwhites have had at least some exposure to the ew hypotensive agents.

[36] One could, of course, design a longitudinal study of white and nonwhite hypertensives that would provide a definitive test of the differential exposure-differential mortality theory.

[37] *See* reference 3.

[38] Since this article was written, the author has found that mortality trends for California are consistent with her theory. From 1950 to 1960 in California, hypertensive disease death rates declined for both whites and nonwhites. The drop for whites was consistently greater than that for Negroes, although often less than that for "other nonwhites." The general reduction in mortality has been explained by a drug theory; but the race differences in mortality trends have not been interpreted. (*See* Borhani, Nemat O., and Hechter, H. H., Recent Changes in CVR Disease Mortality in California, *Public Health Reports,* 79, 147–60, February, 1964.) These California data should be studied further; and, where it is possible, intrastate and interstate comparisons should be made.

[39] The thought is that hypertensive disease is not a wastebasket cause-of-death category as is ASHD and that, other factors being equal, hypertension is more likely to be a designated cause of death in areas having better and more available facilities for medical diagnoses.

[40] A summary and evaluation of this literature will be published in a later paper.

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