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ABSTRACT

Comparisons are used in this paper to identify improvements in mortality and morbidity experiences over time, to identify new environmental hazards, and to emphasize the potential for improvement. The comparisons are presented in the full belief that racial variations are fundamentally socioeconomic variations. Efforts are also made to identify areas of mortality and/or morbidity where available data suggest an interaction between environment and black identification. These are accompanied by descriptions of the types of information needed to interpret these phenomena and of actions necessary to reduce the added disease burden among blacks. In general, death rates within socioeconomic groups become more alike and those between groups more variable as infectious diseases are brought under control. Variations between socioeconomic groups are the compound result of a number of factors. Some of the most important are: exposure to the causes of chronic diseases, access to and use of medical services, personal health behavior, and individual resistance. The last is a physiological state that itself is the consequence of an interplay of environmental experiences, genetic endowment, and health behavior. Some effort is made to interpret the mortality statistics presented in terms of these contributing factors. A useful overall perspective is gained from summaries of the pattern of changes in major causes of death across all ages. (Author/AM)

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Morbidity and Mortality in American Blacks

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Epidemiology is based on the theory that disease is not a chance occurrence and variations between groups with high and low rates of disease, or between diseased and non-diseased individuals, are the result of different experiences, or different responses to similar experiences.

Successful investigations depend upon the degree of homogeneity achieved within, and the range of variation between groups chosen for comparison. Epidemiologists looking for environmental causes of disease need groups with low and high rates of disease or exposure to etiologic factors, but composed of individuals with uniformly high and low rates.

Comparison groups are established by one of two strategies: the identification of comparable ecologic units, or the identification of individuals with similar characteristics. The second provides better homogeneity but takes more time, effort, and money. Historically, this cost has been contained by use of vital records and routinely collected disease notifications. These documents, because they were designed for other purposes, record a limited number of observations that can be used for analytic studies. Chronological age has always been the single most useful characteristic to classify human beings into groups with relatively homogeneous "within" but very different "between" group death rates. Sex became more useful for this purpose as infectious diseases were brought under control.

Its discriminatory value may be reduced, however, as differences in male and female occupational/environmental exposures are reduced.

Life expectation of citizens living and working in different environments has been studied since the late eighteen hundreds. Census records of income, education, and housing have been used as variables for the necessary socioeconomic classifications.

The capacity to examine the mortality experiences of men in different occupations developed as epidemiologic interest focused on industrial hazards. In Britain, where the class system formalized life style and limited occupational opportunities, the readily available death certificate statement of occupation was used to construct social (life style) classes. These proved to be sufficiently homogeneous in disease experience to be accepted as a valuable tool in the investigation of disease. The tool increased in value as different life styles became more or less associated with environmental hazards, means for control of environmental hazards, and successful treatment of manifest disease.

This country did not institutionalize social structure and social roles in the British feudal format. Consequently, it has become more and more difficult to find indicators which identify population groups with relatively homogeneous social-environmental experiences.

DuBois made the point that skin color was a readily accessible general indicator of socioeconomic status in Philadelphia. (1) He went on to support his thesis that Black and white residents were similar when the two races had similar life styles.

The association between skin color and living conditions has been sufficiently strong throughout the years to make skin color the

most readily available general indicator of low socioeconomic living conditions. Its value for this purpose will be reduced as living conditions of Blacks and whites become more similar. In this context, it will presumably follow the pathway of "education." "Number of years of education" was a useful measure to separate adults of all ages into groups with different socioeconomic backgrounds. It has become of extremely limited value in studies of young adults whose educational experiences have been much more standardized.

The middle class exodus from the center of industrial cities in the 1960's has increased the similarity of the environments of Black and white city residents. It can be postulated that skin color will be of less and less value as a variable to classify urban dwellers into different socioeconomic groups. Some evidence to support this proposal appears in Table 1. Using the 1970 census data, large eastern seaboard cities were ranked by degree of similarity of the occupations of their Black and white residents. Age-adjusted death rates (in ten year intervals) were calculated for the working age population (15 - 64 years) in each city.* The Black and white death rates became more similar as the occupational distributions became more similar.

TABLE 1

Ratio $\frac{\text{Black}}{\text{White}}$ Age-Adjusted Death Rates (15-64 Years), 1970

CITIES	RATIO
Birmingham, Charlotte, Charleston	2.17
New Orleans, Richmond, Atlanta	2.05
Baltimore, Philadelphia, New York	1.94

This paper uses comparisons to identify improvements in mortality and morbidity experiences over time, to identify new environmental hazards, and to emphasize the potential for improvement. The comparisons are presented in the full belief that racial variations are fundamentally socioeconomic variations.

Efforts have also been made to identify areas of mortality and/or morbidity where available data suggest an inter-action between environment and Black identification. These are accompanied by descriptions of the types of information needed to interpret these phenomena and of actions necessary to reduce the added disease burden among Blacks.

In general, death rates within socioeconomic groups became more alike and those between groups more variable as infectious diseases were brought under control. Variations between socioeconomic groups are the compound result of a number of factors. Some of the most important are: exposure to the causes of chronic diseases, access to and use of medical services, personal health behavior, and individual resistance. The last is a physiologic state that itself is the consequence of an interplay of environmental experiences, genetic endowment and health behavior. Some effort has been made to interpret the mortality statistics presented in terms of these contributing factors.

Table 2 summarizes the improvement in death rates during this century. It emphasizes the dramatic improvement in survival of children and the decreasing gains from adolescence to old age. The statistics reflect the control of acute infectious diseases which victimized infants and children and the improved care of pregnant women and newborns. The greatest impact is on age groups with the highest rates of infection and on maternal and newborn complications.

This relative impact is illustrated in Table 3 which shows the ratios of Black and white age-specific death rates in 1900 and 1970.

The fall in the ratio describes relatively more improvement in Blacks under the age of 15 and over the age of 75. The increased ratios reflect more white than Black improvement between the ages of 25 and 74.

The gain due to infectious disease control accelerated in the 1940's and 1950's with the discovery and marketing of anti-microbial drugs. The last twenty years have seen the emergence of chronic diseases as major killers and evidence of inability to continue to make major improvements in life expectancy. The greater rate of improvement in those most victimized by infectious diseases continued up to the stabilization of control of pulmonary tuberculosis mortality in the late nineteen fifties.

Figure 1 shows similar trends in death rates in both races from the late fifties to the late sixties. The last ten years saw an increase followed by a relatively more rapid decrease in the Black death rate.

If environment determines variations in death rates, it does so either by causing differences in the incidence of fatal diseases or by mediating alterations in survival.

It is relatively easy to apply available knowledge and technology within the framework of diagnostic and treatment services, but much more difficult to apply them within the context of health education and behavior change in order to reduce the incidence of disease. Given differences in social class and, therefore, in Black and white death rates, it is assumed that they will be most rapidly reduced when deaths are due to variations in medical care rather than the incidence of disease.

A standard of reference was sought to pin-point disease specific death rates with promise of rapid improvement through the application of medical services. It was assumed that the white population in the state with the highest per capita income in 1970 would have the best medical care and the lowest death rates attainable through its general use. Connecticut had the highest per capita income. (2)

In making comparisons, more difference between U.S. Black and Connecticut white population death rates, than between U.S. Black and U.S. white death rates, has been accepted as evidence that socioeconomic variations exist among whites. To the extent the variations are caused by medical care, they offer potential opportunities for rapid improvement in Black rates.

Comparisons between national Black and white death rates and between national Black and Connecticut white death rates were made for the four age groups, 0 through 4 years, 5 through 44 years, 45 through 74 years, and 75 years and over.

In reviewing relative death rate ratios it has been assumed that two to three fold Black-white differences identify diseases where improvements are most likely to be possible. Whenever there is an additional Black-Connecticut white death rate ratio increment, it has been assumed to magnify the potential.

Table 4 shows the leading causes of death in the first five years of life in 1960 and 1970. A majority of deaths in Black children are associated with the reactions of newborns to both conditions surrounding birth and to infection. Infants of both races made similar prematurity death rate improvements in the decade. The calculated relative risks show that Connecticut whites had a slight advantage

over both Blacks and the rest of the whites in the United States.

Deaths assigned to "other diseases of early infancy" and to "birth injury" are also closely dependent on newborn stamina. These causes separately and together showed less improvement in the Blacks. In 1970 there were two-fold differences between U.S. Blacks and whites and almost three-fold differences between U.S. Blacks and Connecticut whites. These ratios suggest that a relatively small but real improvement could be made through concentrated efforts to provide optimum medical management during labor, following delivery, and in the immediate post-neonatal period. Since prematurity and related conditions account for a majority of deaths of Black neonates and contribute in a large way to overall Black/white mortality differences, the subject of infant mortality is explored in more detail later in this paper.

In this age group, 23 per cent of the 1960 and 14 per cent of the 1970 deaths were assigned to infections. There was a three and one-half fold difference in death rates from infections between the two races throughout the ten-year period. This three-fold difference increased to an almost seven-fold difference when U.S. Black infants were compared with Connecticut white infants. These figures suggest strong potential for rapid reduction in deaths from influenza and pneumonia, sepsis of the newborn, and other infectious diseases in Black newborns. These reductions could be achieved through early use of appropriate medical services and improved opportunities for maternal care at home.

Although the remaining listed causes of childhood deaths each accounted for relatively few deaths in terms of the total, some changes are worth notice. There was a large relative increase in reported

deaths from diseases of the heart among Black children. Greatest proportional increases were in reports of heart disease associated with murmurs and reports of non-specific acute cardiac damage. This observation together with the lower relative risk in comparison with Connecticut suggests that diagnosis and management of heart disease per se is probably not the major reason for the change. It is assumed that both improved survival of small infants and improved neonatal diagnosis made contributions.

Although rates of accidental death increased across the board, they increased more rapidly in Black children and the magnitude of the difference between comparisons with Connecticut and U.S. whites suggests that rapid inroads can be made. The emphasis here must again be on improved maternal care and immediate access to medical care.

Death rates from cancer decreased in white children but remained the same for Black children. The improved white experience was concentrated among deaths from leukemia and lymphoma. Children of both races had essentially the same experience with all other cancers. It is worth passing note that 60 per cent of white and 30 per cent of Black cancer deaths in this age group are assigned to leukemia and lymphoma. This is one instance where access to medical care and diagnostic facilities can be both an advantage and a disadvantage. There are two components to lower death rates, a lower incidence of disease and/or better treatment. While Blacks may have a lower incidence in cancer from lack of exposure to X-rays and pharmaceutical products, the more rapid decrease in the white death rates is probably explained by a postponement of death through treatment. Connecticut

whites have an added advantage which can be assumed to be due to some extent to the availability of high quality medical care.

Data for the leading causes of death in the 5 to 44 year olds are shown in Table 5. This group includes young Black adults whose relative experience has deteriorated in the last decade. In both 1960 and 1970 half their deaths were attributed to heart disease, homicide, accidents and cancer.

To facilitate discussion of causes of mortality in this age group, the specific causes listed in Table 5 were grouped into disease categories which are presented in Table 6. Diseases of the heart and cerebrovascular lesions, both associated with high blood pressure, make up the first category. Accidents and homicide are grouped with cirrhosis of the liver and suicide as conditions associated with personal and social behavior. The remaining major causes of death fall into the infectious disease category. It is important to note that although overall rates of death in this age group are low, they are of particular concern because they occur at the peak of an individual's earning capacity and because a majority are avoidable.

Rates of heart disease and cerebrovascular deaths were reduced in both races, with heart disease rates falling more rapidly in whites. The relative death rate ratio for cardiovascular diseases was 2.3 in 1960, 2.6 in 1970 and 2.9 compared with Connecticut. This sequence suggests that medical care could provide a small but immediate benefit among Blacks. Current knowledge suggests this benefit would be mediated through the control of high blood pressure.

All four behavior associated causes increased between 1960 and 1970, and three out of the four increased more rapidly in Blacks. The increased death rate from accidents was essentially due to a rise in

deaths from automobile accidents which increased by 50 per cent in Blacks. Death rates from cirrhosis more than doubled, and those from homicide nearly doubled. As a consequence, the death rate from social disorders increased in Blacks from 1 in 2,000 to 1 in 1,000.

Relative risk of death among Blacks from the four social diseases increased from 1.6 in 1960 to 2.3 in 1970 while there was a three-fold difference between U.S. Blacks and Connecticut whites in 1970. This comparison suggests that immediate attention should be directed toward control of these particular diseases in Blacks. Homicide has the highest relative risk with a nine-fold higher rate in U.S. Blacks than U.S. whites in both 1960 and 1970 and a fifteen-fold higher rate than 1970 Connecticut whites. Homicide will also be discussed in more detail later.

The death rate ratio comparison shows the steepest gradient for infectious diseases. The rate of death from infectious diseases was four times higher in Blacks in 1960; three and one-half times higher in 1970 and almost six times higher than Connecticut whites in 1970. The contributing diseases are influenza and pneumonia, tuberculosis, and syphilis. Prompt use of appropriate medical services can limit deaths from all these causes. Consideration must, of course, be given to such co-morbid conditions as alcoholism and drug addiction that affect decisions to use available services as well as to availability and accessibility of services.

A review of changes in cancer death rates show that most of the differences between Blacks and whites is explained by a slower rate of improvement in rate of death from cancer of the cervix and a more rapid increase in death rates from cancer of the lung. The

former can be easily remedied by use of appropriate medical services, the latter is amenable to control only through modification of cigarette smoking habits.

Table 7 illustrates the changes in death rates for ages 45 through 74 years. During the past decade this age group has shown greatest evidence of an increased incidence of chronic diseases. There was both a Black and white increase in death rates for cirrhosis, cancer, chronic obstructive lung disease and homicide. A more rapid increase in Black cancer death rates was explained by rapidly rising rates from lung cancer, breast cancer, and leukemia. In addition, Blacks showed an increase in gastro-intestinal cancer death rates, while whites showed a decrease, and improvements in death rates for cancer of the cervix were markedly lower in Blacks. The limited impact of any and all medical treatment on lung cancer, breast cancer, leukemia, and gastro-intestinal cancers leads to the assumption that this age group has had a real increase in incidence of cancer of these particular sites. This issue is explored in more detail later in the text. The only immediate action available to reduce the Black cancer death rate would be increased use of medical services for control of cancer of the cervix and intense efforts to reduce the habit of cigarette smoking.

Mention was made earlier of the belief that homicide and cirrhosis deaths are due largely to social and behavioral determinants. It would be reasonable to add chronic obstructive lung disease to this group of diseases because a majority of cases are associated with cigarette smoking. Although it is increasing in frequency as a cause

of middle and old age deaths in both races, chronic obstructive lung disease is one example of a disorder in which there seems to be a worse impact of environmental factors on whites than Blacks.

In this age group, death rates for diabetes mellitus, accidents, and heart disease increased in Blacks but decreased in whites during the decade. Adult diabetes deaths can be moderated by personal health care directed to diet and hygiene and by medical supervision. Intervention to reduce accidental deaths, the majority of which are due to motor vehicles, should be focused on alteration of behavior of both drivers and pedestrians.

The overall rates for heart disease deaths in Blacks summarize two trends: a more rapid increase in atherosclerotic heart disease, and a slower fall in hypertensive heart disease deaths. To the best of medical knowledge, the former is associated with changes in life style, particularly with changes in diet and smoking habits. The latter is associated with medical management or lack of medical management of elevated blood pressure levels and is open to immediate action. Atherosclerotic heart disease, however, is less easy to attack because it requires modification of behavior.

The relative risk of Blacks when compared with Connecticut is higher than that compared with U.S. whites for heart disease, cancer, cerebrovascular disease, influenza and pneumonia, diabetes and nephritis. The differences in the two ratios are, however, relatively small. Connecticut whites show small advantages over the entire U.S white population for the majority of the diseases in question. Taking into account the absolute rates of death and the 1970 relative risks, the greatest immediate impact should lie in a determined effort to control high blood pressure levels to prevent cerebrovascular lesions and

hypertensive heart disease, and in programs of health education and medical care to reduce death rates from diabetes mellitus, influenza, pneumonia and tuberculosis. An attack on health "behavior" should follow.

For some years there has been interest in reported lower death rates in Blacks over the age of 75 years. Two questions must always be considered. First, to what extent is age misclassified through lack of specific birth records, and second, to what extent has selective survival constructed an unusual older cohort.

The last decade has seen a closer approximation of the terminal experience of Blacks and whites lending credence to the belief that selective survival has affected vital statistics (Table 8). It is of interest to note that Blacks age 75 and over have rates more similar to Connecticut than to U.S. whites (i.e., their experience is more like that of the white population with the better mortality experience).

In 1970, rates were approximately the same for diabetes, twice as high for nephritis, and almost two and a half times as high for tuberculosis. For nephritis and tuberculosis the relative risk was greater in the Connecticut comparison suggesting that these particular problems would, in part, yield to an intensive program of care and education.

During the ten years in question, death rates among Blacks from heart disease, cerebrovascular lesions, cancer, diabetes, accidents and chronic obstructive lung disease drew closer to those of the white race as the result of more rapid increases in cancer, diabetes, and

chronic bronchitis and slower rates of reduction in deaths from cerebrovascular lesions, accidents, and cirrhosis. In addition, there was an increase in deaths from heart disease compared with a decrease for whites, this increase being confined to atherosclerotic heart disease.

Cross-sectional views of death rates at any point in time give a distorted impression if major environmental changes have had differential effects on the sequential birth cohorts included in the analysis. There have indeed been a number of major modifications in disease experience in the past 40 years, some of which are known to be, and others presumed to be, the result of changes in environment. First, there has been a reduction in the perinatal mortality rate, which has changed the composition of adolescent and young adult cohorts.

The second modification, control of acute infectious diseases, has had two or three different effects. First, it has changed the composition of the adolescent and young adult population. Secondly, it has changed the immune status and the immunological experience of children, adolescents and young adults. The full impact of this change has yet to be recognized, although the consequences on the manifestations of one or two diseases like poliomyelitis and hepatitis have been documented. An impact on chronic diseases has also been suggested by studies of multiple sclerosis in migrants, but the extent of the effect on diseases such as cancer and cirrhosis of the liver has yet to be investigated. The relationship between enteric infections in childhood and atherosclerosis, and between childhood respiratory infections and chronic lung disease are other avenues for exploration. In addition, the true nature of chronic renal disease has not been clarified, and the extent to which it is a consequence of infection

or lack of early infection has yet to be explored.

Control of acute infections and later of chronic infections has had the greatest effect on those with the highest rates of disease. Eradication, however, has taken longer in these same groups. As a consequence, the remnants of infectious disease were still in existence when chronic diseases began to emerge. For a period of time, people living in the most disadvantaged environmental circumstances were living in double jeopardy.

Many so-called chronic diseases are associated with life styles that reflect increases in disposable income. Automobiles, alcohol, cigarettes, diets high in fat, and refined sugars are for sale. Their increased availability in central cities and socially restricted environments was bound to magnify the rate of increase in deaths associated with their use. Chronic diseases are believed to develop in adolescence and young adult life. Medical services have become readily available for children but are not yet readily available to and/or frequently used by adolescents and young adults. Services should be provided for this age group to limit the progress of these chronic diseases.

The extent to which these environmental changes have changed and are changing the disease experiences of consecutive birth cohorts must be carefully observed and conclusions taken into consideration in planning for disease control.

A useful overall perspective is gained from summaries of the pattern of changes in major causes of death across all ages.

Infectious Diseases.

The past ten years have seen a more rapid white than Black reduction in deaths from infections in infancy and childhood. There is every reason to believe that remaining racial differences could be rapidly reduced by appropriate use of medical services. For example, one of the earliest and most marked effects of the National Maternal and Infant Care Program has been a reduction in post-neonatal deaths, many being deaths from infections.

Adult deaths from infection fall into two major categories; deaths from influenza and pneumonia and deaths from "other infections." The latter group includes tuberculosis, syphilis, and meningitis, but the majority of deaths are from tuberculosis. Many deaths from pneumonia and influenza can be prevented by prompt and appropriate medical management. This is particularly true in young adults free from other disabling diseases. As the frequency of cardiovascular diseases, alcoholism, and diabetes mellitus increase, personal health behavior interferes more and more with the ability to prevent deaths from infection. Most of the excess Black deaths from infection at ages 5 to 44 years are assigned to influenza and pneumonia. It is reasonable to believe that the effect of this socioeconomic gap could be reduced very quickly in this age group by prompt and appropriate medical attention. There should also be potential to close the gap in deaths from tuberculosis in this age group in spite of differences in incidence.

The increasing frequency of vascular disease, alcoholism, and diabetes as co-morbid conditions reduces the ability to lower death

from tuberculosis and from pneumonia and influenza in later years. Nevertheless, the high relative risk in comparison with whites and the even greater relative risk in Blacks as compared with the Connecticut white rates suggest that serious efforts to improve medical management would pay some dividends at ages 45 through 74.

The relatively high proportion of Blacks at risk of death from tuberculosis in this age group is a time-limited phenomenon. The baseline of infected individuals which determines the population at risk of pulmonary tuberculosis and deaths assigned to tuberculosis has completely changed in the past twenty years. Higher death rates currently associated with poor economic conditions at ages 45 onward are likely to disappear within a generation. This does not mean that more intensive medical care should not be directed to tuberculosis at older ages in the immediate future. While the most senior Black age group showed slightly more improvement (24 per cent) than their white counterparts (18 per cent) in death rates from influenza and pneumonia and death rates from nephritis, they showed less improvement in death rates from tuberculosis.

Social and Behavioral Disease.

A number of common diseases are associated with social behavior and personal habits. Accidents, cirrhosis of the liver, homicide, suicide, chronic obstructive lung disease, and lung cancer are among them. The last decade has seen overall increases in rates of death from cirrhosis, suicide, motor vehicle accidents, homicide, and chronic obstructive lung disease. Table 9 shows that motor vehicle accidents increased in both races at all ages except 75 years and over and that the increase was greater for Blacks of all ages. The rate of homicide increased in Blacks across the board and in whites at all ages under

75 years. In terms of total deaths from homicide there was a relatively greater increase in Blacks. Death from cirrhosis of the liver increased more rapidly in Blacks between the ages of 5 and 75 years. Black rates of suicide increased between the ages of 5 and 44 years and decreased from the age of 45 years onward. However, whether the rates were increasing or decreasing, the Black rate has moved closer to the originally higher white rate. In addition Black rates approached higher white rates for the two other diseases associated here with social behavior, chronic obstructive lung disease, and lung cancer.

Because of the nine to fifteen-fold difference in homicide death rates between Blacks and whites of different social classes, some particular attention should be paid to homicide and means for its control. The social environment of the sixties provoked and provided an opportunity for unusual rates of increase in Black homicide deaths. Furthermore, differences in Black and white homicide rates appear to be much greater than expected from social class variation alone. Table 10 shows homicide death rates by age and race for males in Baltimore City, in 1970. The rate in Black males, age 20 to 44 was three per 2,000, a rate 5 times that in white males of the same age.

TABLE 10

Homicide Death Rates per 100,000 by Age and Race

Baltimore City Males, 1970

Race	Age in Years			
	Under 20	20-44	45-64	65 and over
Black	28.6	154.6	66.9	19.2
White	5.6	29.7	22.7	25.8
Ratio: $\frac{\text{Black}}{\text{White}}$	5.1	5.2	2.9	0.7

A review of both Baltimore City and available national data suggest that the differences in the Black/white relative risks at older and younger ages reflect a cohort effect. If this interpretation is true, the rate in older adults will increase as the present generation grows older. If the white males retain approximately the same rates, Black rates at ages 45 to 64 will rise to 1.5 per 1,000 and rates at 65 and over will gradually increase to 1.3 per 1,000.

Guns were used in 75 per cent of homicide deaths in males, ages 20 to 44. The death rates from homicide are so high that these percentages mean that one in every thousand young black males is shot each year. The use of guns is certainly amenable to social control and the threat of a cohort effect should emphasize the need to reconsider present gun sanctions.

Cancer.

Very few cancers can be cured by treatment. Death rates from cancer of the cervix can be reduced and those from leukemia in children can be modified by medical management. In these two sites, medical care could do much to bring Black and white death rates closer together. Table 11 illustrates the changes in cancer mortality over time. The data shown in Table 12 confirm suspicions that much of the relative increase in the Black rates of death from cancer are due to increases in the incidence of cancer in some major organ sites where medical care has limited impact on survival.

As mentioned in the narrative description of Black mortality data, infant mortality and cardiovascular diseases warrant much more extensive discussion.

Infant Mortality and Prematurity.

Figure 2 shows a relative improvement in Black post-neonatal death rates in the latter part of the decade. It coincided with widespread implementation of the National Maternal and Infant Care Program which concentrated its efforts on infants at high risk of perinatal and post-neonatal mortality.

The relative gain was achieved by greater reductions in death rates from infectious diseases and ill-defined conditions. There was no parallel improvement in rates from accidents, congenital malformations and the remaining variety of causes. These trends are emphasized by changes in relative risk of cause specific deaths between 1963 and 1969 (Table 13). The impact is essentially one of maternal use of available pediatric services. The observation that maternal and infant care programs have not had the same impact on Black neonatal death rates is not surprising since the majority of deaths in the first month of life are more closely associated with the viability of newborn infants than with their domestic and medical environments. This does not mean, however, that existing excess Black neonatal deaths could not be reduced by judicious attention to the factors contributing to them. Neonatal death rates showed no meaningful changes in death between 1963 and 1969. Available analysis of national data collected to describe the experience of the 1963 birth cohort was used to investigate reasons for slow improvement in the overall neonatal death rate. The 1963 analysis was acceptable because the relationship between Black and white death rates for the major causes of neonatal deaths did not change between 1963 and 1969.

Although Black infants have had consistently lower birthweights, Table 14 shows that birthweight alone does not explain their mortality experience. They survive better than white infants at all weights below 3,000 grams. However, their relative survival deteriorates at higher weights (i.e., in "full term" not in "premature" infants). Approximately 38 per cent of Black and 44 per cent of white neonatal deaths were in full-size babies in 1963.

The introduction of intensive neonatal care nurseries has had a decided impact on the survival of infants with birthweights between 1,000 and 2,500 grams. Their introduction has resulted in an increased overall proportion of neonatal deaths occurring in full term infants. Graven in Wisconsin reported that 75 per cent of neonatal deaths in a large group of hospitals associated with an intensive care program were in infants weighing 2,500 grams or more at birth. (3) The higher Black death rate in heavy babies is, therefore, becoming more rather than less important over time.

The two questions to investigate, therefore, are causes of higher Black death rates in full term infants and reasons for the birth of excess numbers of small infants who, in spite of better survival, still contribute sufficient numbers of deaths to affect the overall neonatal rates.

The leading causes of neonatal death in infants weighing 3,000 to 4,000 grams at birth in 1963 and their Black/white relative risks are shown in Table 15. Excess Black risks are concentrated among infections and ill-defined conditions. Excess deaths occur between the 8th and 28th day after delivery. (4)

These observations suggest that emphasis should be placed on maternal education for infant care with particular directions about the prompt use of pediatric services for all infant ailments. The need for pediatric services accessible and available to all mothers of newborns goes without saying. However, in the absence of intensive education about their use, their provision has had limited benefit to data.

The major variables associated with birthweight distribution are pregnancy order, maternal height and weight, and maternal cigarette smoking habits. In second or later pregnancies, the outcome of the most recent pregnancy is important. One illustration is that the proportion of infants under 2,501 grams born to all Black pre-natal registrants in the University of Maryland Hospital between 1962 and 1967 was 17 per cent among women who smoked and 11.4 per cent among women who did not smoke during pregnancy. When the same women were classified by their weight and height, the proportions of low weight infants ranged from 19.8 among the relatively underweight, to 7.6 among the relatively heavy women. (5) In fact, maternal biological characteristics show such consistent relationships to infant birthweight it is difficult to detect an impact of pre-natal care on the birth weight distribution. (6)

Recognition of the importance of maternal physique in determination of infant birth weight led to a detailed analysis of this relationship in teenage mothers whose infants, as a group, have extraordinarily high neonatal and post-neonatal death rates.

There are many more Black than white teenage pregnancies, particularly early teenage pregnancies and the rate is increasing more

rapidly in Blacks. The consequences of teenage pregnancy and delivery are, therefore, of particular concern. An analysis of pregnancy outcomes in young Black primiparae showed no additional adverse effects of cigarette smoking on birth weight but an exaggerated effect of low maternal weight relative to height. (Figure 3) Girls under 15 years of age who were relatively underweight at conception had a major increase in births of very small infants. Table 16 shows an analysis of the gestational age of these babies. The results suggest an increased proportion of infants both under 36 weeks of age and under 2,500 grams in weight. (7) These infants face the dual hazards which accompany immaturity and small size for gestational age.

The problem of ill-advised teenage pregnancy is best attacked through sex and contraceptive education, family planning, and judicious use of abortion facilities. Girls should be counselled and taught to postpone pregnancy until physically and physiologically mature. It is important to note that the foregoing analyses show that maternal physical maturity more than chronological age appears to be the factor associated with the health of newborns and that biological maturity rather than age should trigger admission to programs for counselling and service.

Cardiovascular Diseases.

Although social class differences in cardiovascular mortality have been repeatedly confirmed in this country, racial differences have continued to outweigh them. There is good evidence to believe that environmental factors associated with the development of high

blood pressure have more impact on Blacks than whites, with a particular life style being a factor in the interaction. There is equally good evidence that the life style of the white population has led to atherosclerosis. An increase in Black atherosclerotic heart disease rates has now appeared when rates in whites are beginning to fall. The decrease in whites is generally believed to be associated with improved control of high blood pressure and the initial impact of reductions in smoking habits. The fact that Blacks lag behind whites in blood pressure control is emphasized by a slower rate of improvement in deaths from cerebrovascular lesions and hypertensive heart disease. Morbidity from hypertension and strokes show similar racial patterns as mortality. This observation is general throughout the country, but Baltimore City data have been chosen for illustration.

The follow-up study of the Chronic Illness Survey in Baltimore City twenty years ago reported higher all cause death rates in the lower socioeconomic groups in both races and both sexes. Differences between socioeconomic classes were, however, less than differences between races. Strokes had marked racial but no social class associations. (8)

A morbidity survey in Baltimore City in 1966-1968 showed a higher frequency of symptoms of transient cerebral ischemia, believed to precede strokes, in lower socioeconomic groups in both races (Table 17). (9) It also showed excess numbers of affected Blacks over the age of 55 years (Figure 4). In the same survey, the proportion of Blacks with histories of diagnosed strokes and hypertension was higher at every age (Table 18).

A more recent survey of the total prevalence of treated and untreated high blood pressure in the center of Baltimore City showed statistically significant associations between Black census tract prevalence rates and measures of socioeconomic status. (10) The scattergram shown as Figure 5 illustrates the association between age and sex adjusted prevalence rates of diastolic blood pressure levels over 94 mm mercury and individually collected measures of completed years of education. These social class differences in blood pressure levels among Blacks are believed to be the result of both variations in incidence and variations in medical care.

There is evidence to support the existence of variations in incidence between Blacks and whites in the same social classes. The preliminary data shown in Table 19 compares age-specific blood pressure levels in Black and white federal employees who volunteered for a health screening program. (11) Although the data have not been adjusted for occupational status within the agency, or self-selection bias, acceptance rates were sufficiently high and age specific work requirements are sufficiently similar, that observed differences are believed to reflect racial rather than concealed socioeconomic variations.

The current national series of investigations into the benefits of early versus later blood pressure management should provide tools to equalize morbidity in the absence of changes in incidence. (12) The very high prevalence rates, however, and the prospect of thirty to forty years of pharmaceutical management emphasize the urgent need for studies to identify those particular features of Black life style that predispose to rises in blood pressure in early adult life. The results of preliminary investigations into the relationships between

the stress of social incongruity (13) and changing social responsibilities (14) and the results of comparative epidemiologic studies of the effects of dietary sodium and calcium intakes (15,16), suggest these are promising areas for more extensive exploration.

In addition to a higher prevalence of elevated blood pressure and its complications, particularly stroke, there is fairly strong circumstantial evidence that hypertensive diseases have more rapid natural histories in Blacks. Differences between Black and white adult populations increase from prevalence (twofold) (17,18) through morbidity (threefold or more) (17,19) to mortality (sevenfold) (20).

Attention has been drawn to the rapidly increasing Black death rate from atherosclerotic heart disease. The early evidence suggests that the increased death rate is the consequence of both an increasing rate of new disease and an unusually high fatality associated with acute events.

The higher incidence of the disease is attributed to more heavy cigarette smoking and increased use of high fat diets. In spite of higher rates of obesity, Blacks have previously had lower levels of blood cholesterol. Some evidence that differences in Black and white blood cholesterol levels are disappearing comes from the preliminary results of cardiovascular screening in the group of federal employees previously mentioned (Table 20). (21) Age-specific blood cholesterol levels are virtually the same in both races in this more or less middle-class population.

Data from several sources support the idea that there is continued increase in cigarette smoking among Blacks while this habit is falling off, at least in white middle aged males. (22) The smoking habits of the same group of federal employees reflect these trends

(Table 21). (23)

An analysis of the records of all patients with confirmed myocardial infarction admitted to the University of Maryland Hospital since 1960 shows a steady increase in the number of afflicted Black patients. It is believed that the increase reflects more than changes in the population at risk of admission and/or admission policies. In addition, the findings of Shapiro, et al suggest that case-fatality rates are higher in Blacks who suffer myocardial infarctions. (24)

The extent to which increased death rates from diabetes mellitus reflect the same changes in Black life style and/or contribute to the increased rates of incidence and death from atherosclerotic heart disease is a matter for immediate investigation. However, an all-out attack on cigarette smoking, eating patterns, and compliance with hypotensive therapy cannot be delayed until more is known about the mechanisms of disease.

Use of Services

A note of encouragement towards provision of appropriate education and services for specific health problems comes from review of the use of both preventive and diagnostic services when there is equal opportunity for access. Tables 22 and 23 show measures of the use of prenatal and post-partum services by young Black and white registrants in 13 maternal and infant care project centers. (25) As evident in the tables, Blacks made better use of this preventive care.

Table 24 shows rates at which Blacks and whites in Baltimore accepted invitations to attend a special clinic for diagnostic

examinations. (26) Some invited participants had symptoms of transient cerebral ischemia and some had histories of strokes and hypertension. All were identified and interviewed in a household survey. More Black females than white accepted whether or not they had disease, symptoms or no symptoms. Black males accepted more often overall, but there was some variation among the responses of those with different numbers of symptoms or different disease histories.

In both projects the invitation, transportation assistance, and facilities were uniformly available to all potential users.

Health

Recent national measures of physical attributes and limitations in children and young people provide room for quiet optimism. As can be seen in Figure 6 Black children aged 6-11 are as tall as their white peers. (27) The slightly greater weight of Black females in this age group is probably evidence of more rapid sexual maturation. There is, however, a tendency to excess weight in adult Black females. Since adult obesity is associated with obesity in childhood, the problem should be addressed and corrected during childhood.

Evidence of better Black dental health has persisted in spite of presumed changes in dietary patterns. Black children have fewer carious primary and secondary teeth and the dental advantage continues into adult life. These gains are illustrated by differences in the results of adult dental examinations at the national level shown in Figure 7. (28)

Although differences are not as marked as those for dental health, Black children have better measures of hearing at upper and lower frequencies. (29) Their measures of visual acuity are the same as those for white children and slightly better in terms of less impaired

distance vision. (30)

Table 25 shows rejection rates of army enlistees in 1971, with specific causes for rejection by race. (31) Table 26 provides the same information for army draftees. (32) Although these data published by the armed services include known as well as unknown selection biases, they do not suggest any excess disabilities in the current Black adolescent and young adult populations.

The draftees probably provide the better basis for overall comparison as they can be presumed to be more representative of the population from which they are selected. The results of medical examination show distinct Black advantages. Black and white enlistees are considered to be more alike in terms of socioeconomic background. If anything, Black enlistees are likely to be from better educational backgrounds than whites. This assumption is based on general knowledge of employment opportunities open to young men of both races. It is, however, hard to believe that Black enlistees as a group have enough socioeconomic advantage to explain observed disability differences on this ground alone.

TABLE I

Ratio $\frac{\text{Black}}{\text{White}}$ Age-Adjusted Death Rates (Age 15-64 Years)

Selected Cities, 1970

Cities	Ratio
Birmingham, Charlotte, Charleston	2.17
New Orleans, Richmond, Atlanta	2.05
Baltimore, Philadelphia, New York	1.94

TABLE 2

Age-Specific Death Rates per 1,000

United States, 1900 and 1970

Age in Years	1900 ¹		1970 ²		Per Cent Decrease 1900-1970 ²	
	Black	White	Black	White	Black	White
All Ages	25.0	17.0	9.5	9.4	62.0	44.7
Under 1	333.9	159.4	31.8	18.0	90.5	88.7
1 - 4	43.5	19.4	1.3	0.7	97.0	96.4
5 - 14	9.0	3.8	0.5	0.4	94.4	89.5
15 - 24	11.5	5.7	2.1	1.1	81.7	80.7
25 - 34	12.1	8.1	3.8	1.3	68.6	84.0
35 - 44	14.8	10.1	6.8	2.7	54.1	73.3
45 - 54	24.3	14.8	13.1	6.6	46.1	55.4
55 - 64	42.1	27.0	26.1	15.7	38.0	41.9
65 - 74	68.9	56.2	53.8	35.2	21.9	37.4
75 - 84	120.9	123.3	71.3	78.2	41.0	36.6
85 & over	215.2	262.0	88.6	190.1	58.8	27.4

¹Death Registration States included 10 states and the District of Columbia

²Estimated

TABLE 3
 Ratio $\frac{\text{Black}}{\text{White}}$ Age-Specific Death Rates

United States, 1900 and 1970

Age in Years	1900	1970
Under 1	2.1	1.8
1 - 4	2.2	1.9
5 - 14	2.4	1.2
15 - 24	2.0	1.9
25 - 34	1.5	2.9
35 - 44	1.5	2.5
45 - 54	1.6	2.0
55 - 64	1.6	1.7
65 - 74	1.2	1.5
75 - 84	1.0	0.9
85 & over	0.8	0.5

TABLE 4

Death Rates per 10,000 by Cause and Race

Ages 0-4 Years

United States 1960, 1970; Connecticut, 1970

Cause	Year				Rate Black U.S. Rate White U.S.		Rate Black U.S. Rate White Connecticut	
	1960		1970		1960	1970	1970	
	Black	White	Black	White			Black	White
Prematurity	33.0	17.6	19.5	10.6	1.9	1.8	2.3	
Influenza & Pneumonia	16.6	4.4	11.2	3.2	3.8	3.5	6.6	
Congenital Malformations	7.9	8.7	7.1	6.6	0.9	1.1	1.1	
Other Diseases of Early Infancy	7.5	3.7	3.0	1.0	2.0	3.0	3.3	
Birth Injury	6.0	4.8	2.6	1.2	1.3	2.2	2.4	
Sepsis of the Newborn	5.6	1.8	3.5	1.0	3.1	3.5	5.8	
Accidents	1.6	1.1	2.1	1.2	1.5	1.8		
Motor Vehicle Accidents	1.0	0.9	1.6	1.1	1.1	1.5	3.2	
Cancer	0.7	1.1	0.7	0.7	0.6	1.0	1.4	
Other Infectious Diseases	0.6	0.3	0.4	0.2	2.0	2.0	10.0	
Diseases of the Heart	0.5	0.4	1.1	0.3	1.3	3.7	2.8	

TABLE 5

Death Rates per 10,000 by Cause and Race
Ages 5-44 Years

United States 1960, 1970; Connecticut, 1970

Cause	Year				Rate Black U.S. Rate White U.S.		Rate Black U.S. Rate White Connecticut	
	1960		1970		1960	1970	1970	
	Black	White	Black	White				
Diseases of the Heart	3.8	1.9	3.7	1.5	2.0	2.5	2.9	
Homicide	2.6	0.3	4.6	0.5	8.7	9.2	15.3	
Cancer	2.5	2.1	2.4	1.8	1.2	1.3	1.2	
Accidents	2.4	2.2	3.5	2.9	0.9	1.2		
Motor Vehicle Accidents	2.1	2.1	3.2	2.8	1.0	1.1	1.8	
Cardiovascular Lesions	1.4	0.4	1.2	0.4	3.5	3.0	3.0	
Influenza and pneumonia	1.2	0.4	0.9	0.3	3.0	3.0	4.5	
Other Infectious Diseases	0.8	0.1	0.3	0.04	8.0	7.5	7.5	
Cirrhosis	0.6	0.3	1.4	0.4	2.0	3.5	3.5	
Suicide	0.4	0.7	0.7	0.9	0.6	0.8	1.0	

TABLE 6

Death Rates per 100,000 by Disease Category and Race

Age 5-44 Years

United States 1960, 1970 and Connecticut, 1970

Disease Category	1960		1970	
	Black	White	Black	White
Cardiovascular	5.2	2.3	4.9	1.9
Social and Behavioral	5.6	3.5	10.2	4.5
Infectious	2.0	0.5	1.2	0.3
Cancer	2.5	2.1	2.4	1.8

Death Rate Ratios

Disease Category	$\frac{\text{Black U.S.}}{\text{White U.S.}}$		$\frac{\text{Black U.S.}}{\text{White Connecticut}}$
	1960	1970	1970
Cardiovascular	2.3	2.6	2.9
Social and Behavioral	1.6	2.3	3.2
Infectious	4.0	3.5	5.0
Cancer	1.2	1.3	1.2

TABLE 7
 Death Rates per 10,000 by Cause and Race
 Ages 45-74

United States 1960, 1970; Connecticut, 1970

Cause	Year		Rate Black U.S. Rate White U.S.		Rate Black U.S. Rate White Connecticut		
	1960	1970	1960	1970	1970	1970	
	Black	White	Black	White	Black	White	
Diseases of the Heart	92.4	75.4	99.5	70.5	1.2	1.4	1.6
Cancer	42.0	37.1	51.1	38.9	1.1	1.3	1.4
Cerebrovascular Lesions	38.1	16.0	32.8	12.9	2.4	2.5	2.9
Influenza and Pneumonia	10.4	4.4	8.4	3.7	2.4	2.3	2.9
Diabetes	6.7	3.7	8.7	3.5	1.8	2.5	3.1
Other Infectious Diseases	5.2	1.6	2.3	0.5	3.3	4.6	8.7
Accidents	4.5	3.8	5.5	3.7	1.2	1.5	
Motor Vehicle Accidents	3.1	2.4	4.1	2.7	1.3	1.5	3.3
Nephritis	3.6	1.0	2.8	0.7	3.6	4.0	4.7
Cirrhosis	2.8	3.2	5.8	4.2	0.9	1.4	1.2
Asthma, emphysema, bronchitis	2.1	2.6	2.7	3.8	0.8	0.7	1.0

TABLE 8

Death Rates per 10,000 by Cause and Race

Age 75 and over

United States 1960, 1970; Connecticut, 1970

Cause	Year		Rate Black U.S. Rate White U.S.		Rate Black U.S. Rate White Connecticut		
	1960	1970	1960	1970			
	Black	White	Black	White			
Disease of the Heart	367.6	505.9	387.9	457.8	0.7	0.8	0.9
Cerebrovascular Lesions	163.1	187.4	160.5	160.7	0.9	1.0	1.1
Cancer	89.4	120.3	115.7	120.6	0.7	1.0	0.9
Influenza and Pneumonia	46.5	47.2	35.1	38.3	1.0	0.9	0.8
Diabetes	14.1	16.9	21.4	19.5	0.8	1.1	1.1
Nephritis	13.2	16.2	7.4	3.4	2.1	2.2	3.4
Accidents	11.1	24.3	9.6	17.1	0.5	0.6	
Motor Vehicle Accidents	3.4	4.2	3.4	4.2	0.8	0.8	1.4
Other Infectious Diseases	9.1	4.0	4.7	1.7	2.3	2.8	2.5
Asthma, Emphysema, Bronchitis	3.9	7.5	7.3	13.2	0.5	0.6	0.6

TABLE 9

Death Rates Per 10,000 for Motor Vehicle Accidents by

Age and Race

United States, 1960 and 1970

Year	Age in Years									
	0-4		5-44		45-74		75 and over		All ages	
	Black	White	Black	White	Black	White	Black	White	Black	White
1960	1.0	0.9	2.1	2.1	3.1	2.4	3.4	4.2	2.2	2.1
1970	1.6	1.1	3.2	2.8	4.1	2.7	3.4	4.2	2.9	2.7

TABLE 10
 Homicide Deaths by Age and Race
 Rates per 100,000
 Baltimore City Males, 1970

Age in Years	Black	White	$\frac{\text{Black}}{\text{White}}$
Under 20	28.6	5.6	5.1
20-44	154.6	29.7	5.2
45-64	66.9	22.7	2.9
65 and over	19.2	25.8	0.7

TABLE 11

Age-Adjusted Death Rate per 100,000* for Selected Groups
of Cancer Sites by Race
United States, 1940-1970

Cause	Race	1940	1950	1960	1970
Digestive Organs and Peritoneum	Black	41.8	49.8	48.1	42.5
	White	56.1	47.3	40.3	35.2
Respiratory System	Black	4.3	10.6	21.1	31.5
	White	7.5	13.0	19.1	29.3
Breast	Black	9.2	9.7	10.9	10.9
	White	12.0	11.7	11.8	12.5
Genital Organs	Black	30.8	31.3	28.1	23.8
	White	22.8	19.7	17.0	15.0
All Malignant Neoplasms	Black	101.5	128.6	139.3	145.5
	White	121.3	124.7	124.2	127.6

*Adjusted to 1940 population

TABLE 12

Age-Adjusted Incidence Rates per 100,000* for Selected
Groups of Cancer Sites by Race
United States, 1947 and 1970.

Group	Race	1947	1970
Digestive Organs and Peritoneum†	Black	73.5	66.0
	White	87.1	60.4
Esophagus	Black	5.9	9.1
	White	4.9	2.5
Respiratory System	Black	18.0	49.9
	White	22.0	40.0
Breast	Black	27.0	30.9
	White	38.0	39.0
Genital Organs	Black	70.4	67.4
	White	55.1	47.5
All Sites	Black	272.0	292.8
	White	333.0	269.9

* Adjusted to 1950 population

+ (Exclusive of Esophagus)

TABLE 13

Relative Rates of Neonatal and Post-Neonatal Death* by
Cause and Race
United States, 1963 and 1969

Neonatal

Cause	1963	1969
Prematurity	2.1	2.3
Postnatal Asphyxia and Atelectasis	1.5	1.8
Ill-defined Conditions	1.5	2.4
Birth Injury	1.2	1.9
Congenital Malformation	0.7	0.8
All Other Causes	2.1	1.5

Post-Neonatal

Cause	1963	1969
Influenza and Pneumonia	3.6	3.2
Gastritis, Duodenitis, Enteritis and Colitis, except Diarrhea of the Newborn	5.9	3.0
Symptoms and Ill-defined Conditions	7.1	3.4
Accidents	2.1	2.0
Congenital Malformations	1.0	1.1
All Other Causes	2.6	2.6

* $\frac{\text{Rate Black U.S.}}{\text{Rate White U.S.}}$

TABLE 14

Neonatal Death Rates by Race and Birthweight
United States, 1963 Cohort

RACE	Total	Birth Weight							Rate per 1,000 Live Births	
		1,001 grams or less	1,001 to 1,500 grams	1,501 to 2,000 grams	2,001 to 2,500 grams	2,501 to 3,000 grams	3,001 to 3,500 grams	3,501 to 4,000 grams		4,001 to 4,500 grams
White	16.9	924	555	198	45.0	10.00	4.38	3.28	3.60	7.68
Black	26.7	884	434	130	30.7	9.42	6.42	6.61	10.1	16.30

Source: Henderson, M., Hebel R., and Boht, L. Dimensions of teenage pregnancy. Presented to the Society for Adolescent Medicine, Los Angeles, California, May 9-11, 1973.

TABLE 15

Major Causes of Neonatal Death by
Race. Birthweight 3000-4000 Grams
Rate per 100,000 Live Births
United States, 1963

Causes of Death	Ratio $\frac{\text{Black}}{\text{White}}$	
	Birthweight in Grams	
	3001 - 3500	3501 - 4000
Congenital Malformations	0.63	0.95
Birth Injuries	1.57	2.13
Postnatal asphyxia and atelectasis	1.67	1.77
Pneumonia of the newborn	2.70	3.44
Diarrhea of the newborn	4.96	8.73
Ill defined diseases of infancy	1.86	2.88
"Residual" conditions	2.46	3.30
All other	1.29	2.00

Per Cent of Deaths Due to Causes Specified

	<u>3001 - 3500</u>	<u>4001 - 4500</u>
White	87.6	89.5
Black	89.0	89.6

TABLE 16

Prematurity and Immaturity by Age and Stature

NIMDB 1959 - 1965

Black Primigravidas

Age	Percentage less than 2501 grams		Percentage less than 2501 grams and 36 weeks gestation	
	Small	Medium or Large	Small	Medium or Large
Under 15	26.1	11.6	10.4	2.9
15 - 19	16.8	13.8	6.9	5.6
20 - 24	16.0	13.2	5.8	5.1
25 - 34	22.4	15.3	4.0	5.2

TABLE 17

Per. Cent of Respondents with Symptoms of Cerebral Ischemia

by Education

Baltimore, Maryland

1966

Symptom	Education*	
	Low (1,284) %	High (3,303) %
Fainting	5.5	4.6
Dizziness	27.0	17.7
Falls	14.3	12.5
Loss of Vision	2.8	1.4
Double Vision	6.5	4.4
Nausea	20.1	16.0
Difficulty Speaking	2.4	1.5
Difficulty Swallowing	3.1	2.8
Numbness	31.7	26.3
Paralysis	0.9	0.4

* Low Less than 6 years
High 6 or more years

Source: Mules, Janet, et al. A population survey of symptoms suggestive of transient ischemic attacks. Stroke 2: 120, 1971.

TABLE 18

Per Cent of Respondents Reporting a History of Stroke
and/or Hypertension by Age, Race and Sex*

Baltimore, Maryland

1966

Disease	Race, Sex	Age in Years				All Age
		45-54	55-64	65-74	75 and over	
		(%)	(%)	(%)	(%)	(%)
STROKE	WM	1.7	1.6	5.0	5.8	4.3
	WF	1.1	2.6	2.9	3.4	2.8
	BM	2.3	5.2	3.8	4.8	3.7
	BF	2.0	3.8	5.3	3.6	3.4
	TOTAL	1.9	3.7	3.0	3.1	3.4
HYPERTENSION	WM	14.0	23.6	20.8	17.0	19.6
	WF	20.1	25.0	40.4	44.3	37.1
	BM	23.4	26.7	33.8	35.5	27.3
	BF	40.9	46.5	52.8	44.6	45.5
	TOTAL	28.5	32.7	26.6	26.5	32.2

* Number of individuals in each appears in Appendix B

Source: Mules, Janet, et al. A population survey of symptoms suggestive of transient ischemic attacks. Stroke 2: 122, 1971

TABLE 19
 Mean Diastolic Blood Pressure Level*
 by Age, Race, and Sex
 Social Security Administration
 Baltimore, Maryland
 1969 - 1974

AGE IN YEARS	MALE				FEMALE			
	BLACK		WHITE		BLACK		WHITE	
	Mean	s.d.	Mean	s.d.	Mean	s.d.	Mean	s.d.
20 - 24	69.3	10.4	72.7	10.4	66.0	10.3	66.8	9.4
25 - 29	70.6	11.5	72.6	10.7	68.0	25.9	67.4	9.3
30 - 34	75.5	11.7	75.3	10.0	70.4	11.0	69.2	10.9
35 - 39	80.1	12.8	76.9	10.2	76.0	12.1	73.4	10.6
40 - 44	77.8	12.5	79.2	10.8	78.1	13.6	74.2	11.0
45 - 49	83.4	11.6	80.6	11.5	79.3	13.1	75.4	12.1
50 - 54	86.6	12.2	81.8	11.4	82.3	12.3	77.7	11.2
55 - 59	84.5	11.6	82.9	11.5	84.4	14.2	77.0	11.0
60 - 64	92.0	16.1	82.1	11.6	82.3	13.0	78.7	11.9

* Number of individuals in each will appear in Appendix C

Source: Sherwin, R., et al. Personal communication, August, 1974.

TABLE 20

Mean Serum Cholesterol Level*

by Age, Race and Sex

Social Security Administration

Baltimore, Maryland

1969 -- 1974

AGE IN YEARS	MALE				FEMALE			
	BLACK		WHITE		BLACK		WHITE	
	mg/100 ml	s.d.	mg/100 ml	s.d.	mg/100 ml	s.d.	mg/100 ml	s.d.
20 - 24	168.4	30.4	169.4	30.9	182.8	38.0	178.3	37.6
25 - 29	174.8	38.8	180.2	36.3	187.5	41.1	184.3	37.4
30 - 34	193.4	39.2	194.1	39.5	190.7	42.5	187.3	36.6
35 - 39	215.7	45.0	203.4	40.9	200.0	48.9	201.2	43.4
40 - 44	212.0	71.7	214.3	43.5	208.7	41.1	204.2	41.3
45 - 49	218.3	40.9	215.0	41.6	212.4	46.8	212.8	38.2
50 - 54	220.9	44.1	217.6	40.1	223.5	47.3	229.1	45.1
55 - 59	217.7	48.2	217.1	38.4	230.1	53.3	240.2	45.3
60 - 64	221.3	61.7	219.8	44.3	239.2	47.7	252.5	47.5

* Number of individuals in each cell appear in Appendix D

Source: Sherwin, R., et al. Personal communication, August, 1974.

TABLE 21

Per Cent of Screenees Smoking Twenty or More
Cigarettes* per Week by Age, Race and Sex

Social Security Administration

Baltimore, Maryland

1969 - 1970

Age, in Years	MALE		FEMALE	
	BLACK (%)	WHITE (%)	BLACK (%)	WHITE (%)
20-24	43.0	45.0	40.0	33.0
25-29	43.0	32.0	40.0	35.0
30-34	39.0	29.0	40.0	30.0
35-39	42.0	22.0	40.0	27.0
40-44	36.0	26.0	42.0	35.0
45-49	46.0	18.0	46.0	29.0
50-54	44.0	15.0	37.0	26.0
55-59	33.0	15.0	40.0	26.0
60-64	14.0	21.0	33.0	27.0

* Number of individuals in each cell was not available but is comparable to those in Appendix C.

TABLE 22

Median Number of Prenatal Visits for
 Maternal and Infant Care Program Participants* Under 22
 Years of Age, 1969 - 1970

RACE	Age in Years			
	Under 16	16 - 17	18 - 19	20 - 21
Black	7.8	7.5	7.4	7.3
White	8.0	7.3	7.3	6.8

* Based on 11,458 Black and 4,061 white participants in 13 MIC centers.

Source: McDill, Mary S. Can prenatal care alter the morbidity of pregnancy? Presented to The Society for Adolescent Medicine, Los Angeles, California, May 9-11, 1973.

TABLE 23

Per Cent of Maternal and Infant Care Program Participants*
 Less than 22 Years of Age Who Made Post-Partum Visits†
 by Age and Race, 1969-1970

RACE	AGE IN YEARS			
	Under 16	16-17	18-19	20-21
Black	71.0	70.0	66.0	62.0
White	57.0	58.0	59.0	55.0

*: Based on 11,458 Black and 4,061 white registrants in 13 M.I.C. Centers.

† Within 8 weeks of delivery

Source: McDill, Mary S. Can prenatal care alter the morbidity of pregnancy?
 Presented to The Society for Adolescent Medicine,
 Los Angeles, California, May 9-11, 1973.

TABLE 24

Per Cent Accepting Physical Examination
by Race, Sex, and Risk Group*
Baltimore, Maryland
1967

Risk Group	Male		Female		Total %
	Black %	White %	Black %	White %	
Disease History					
Stroke	73.3	46.7	66.7	35.3	54.8
Heart Disease	48.6	60.9	64.9	43.8	54.5
High Blood Pressure	52.5	54.2	46.3	44.8	48.1
Symptoms of Cerebral Ischemia					
1 or more focal	62.5	56.3	53.7	52.6	55.9
3 or more focal	62.5	47.7	65.7	65.1	62.3
2 diffuse	45.7	46.7	60.6	50.0	52.4
No Symptoms of Cerebral Ischemia	54.0	48.1	47.2	30.1	43.6
Total	57.4	50.4	56.5	45.8	52.4

Numbers of individuals in each cell appear in Appendix E

Source: Tonascia, Susan. Personal communication, August, 1974.

TABLE 25

Army Medical Examination Rejection Rate per 1,000 by
Cause and Race*
Enlistees, 1971

Cause	Black	White
Musculoskeletal Defects	17	20
Circulatory System Diseases	15	16
Other Specified Defects	15	15
Eye Diseases and Defects	13	10
Overweight	13	19
Symptoms and Ill-Defined Defects	10	9
Ear Diseases and Defects	9	13
Underweight	8	8
Digestive System Diseases	7	12
Congenital Malformations	5	7
Drug Abuse	5	3
Mental Disorders	5	11
Diseases of Skin and Cellular Tissue	4	10
All Causes	140	177

*Based on 79,000 examinations of Blacks and 67,000 examinations of Whites

Source: Col. William Faulk, Office of the Surgeon General, personal letter, May, 1973

TABLE 26

Army Medical Examination Rejection Rate per 1,000 by
Cause and Race*
Draftees, 1971

Cause	Black	White
Musculoskeletal Defects	43	69
Circulatory System Diseases	38	58
Overweight	33	44
Ear Disease and Defects	26	25
Other Specified Defects	26	45
Eye Diseases and Defects	21	18
Symptoms and Ill-Defined Defects	18	16
Mental Disorders	15	32
Drug Abuse	13	8
Digestive System Diseases	11	22
Congenital Malformations	10	19
Underweight	10	11
Diseases of Skin and Cellular Tissue	6	20
All Causes	286	414

* Based on 67,000 examinations of Blacks and 471,000 examinations of whites

Source: Col. William Faulk, Office of the Surgeon General, personal letter, May, 1973.

FIGURE 1

AGE-ADJUSTED DEATH RATES FOR ALL CAUSES BY
RACE: UNITED STATES, 1930-72

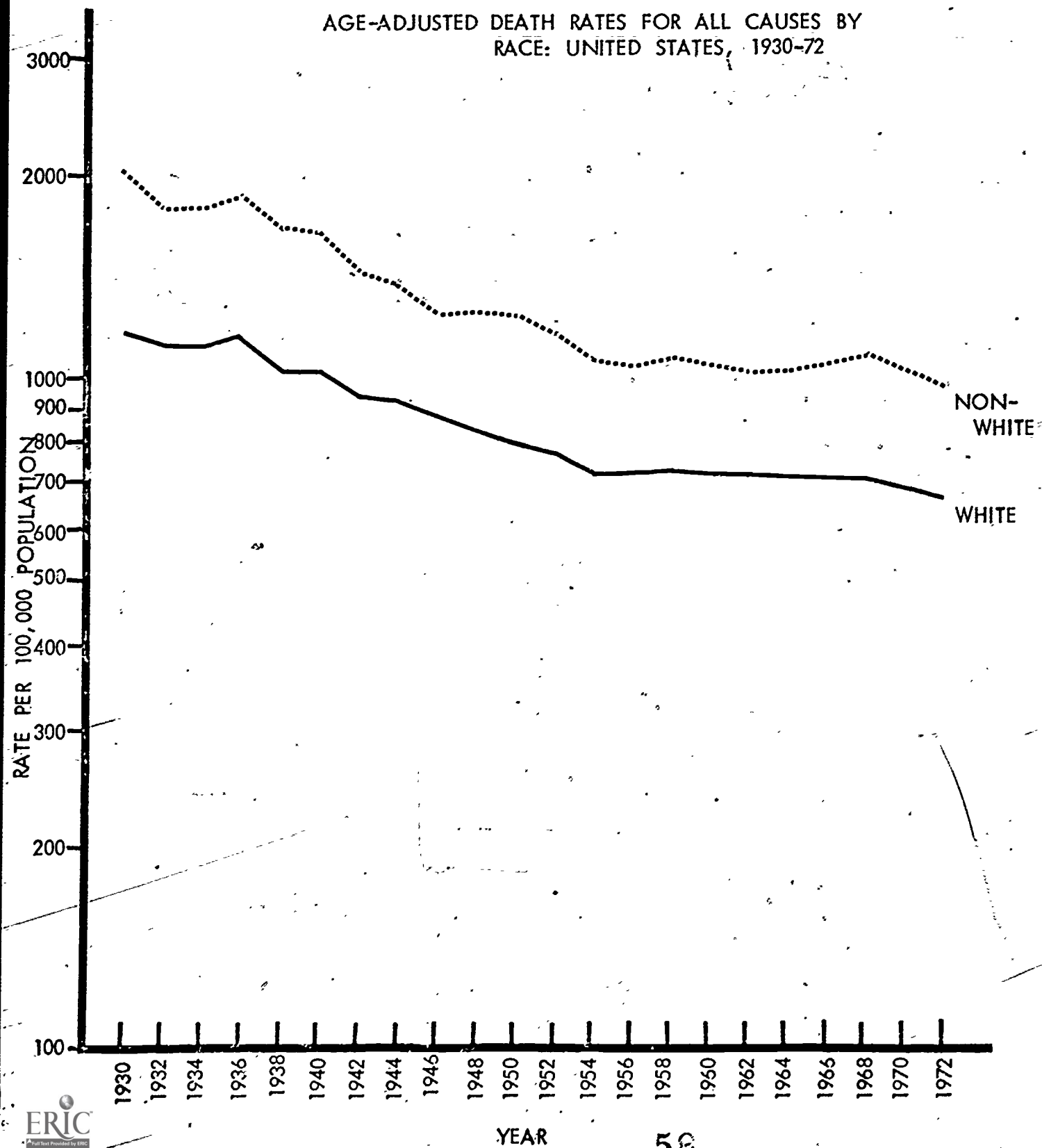


FIGURE 2

NEONATAL AND POST NEONATAL DEATH RATES
BY COLOR, PER 1,000 LIVE BIRTHS
UNITED STATES 1940-1970

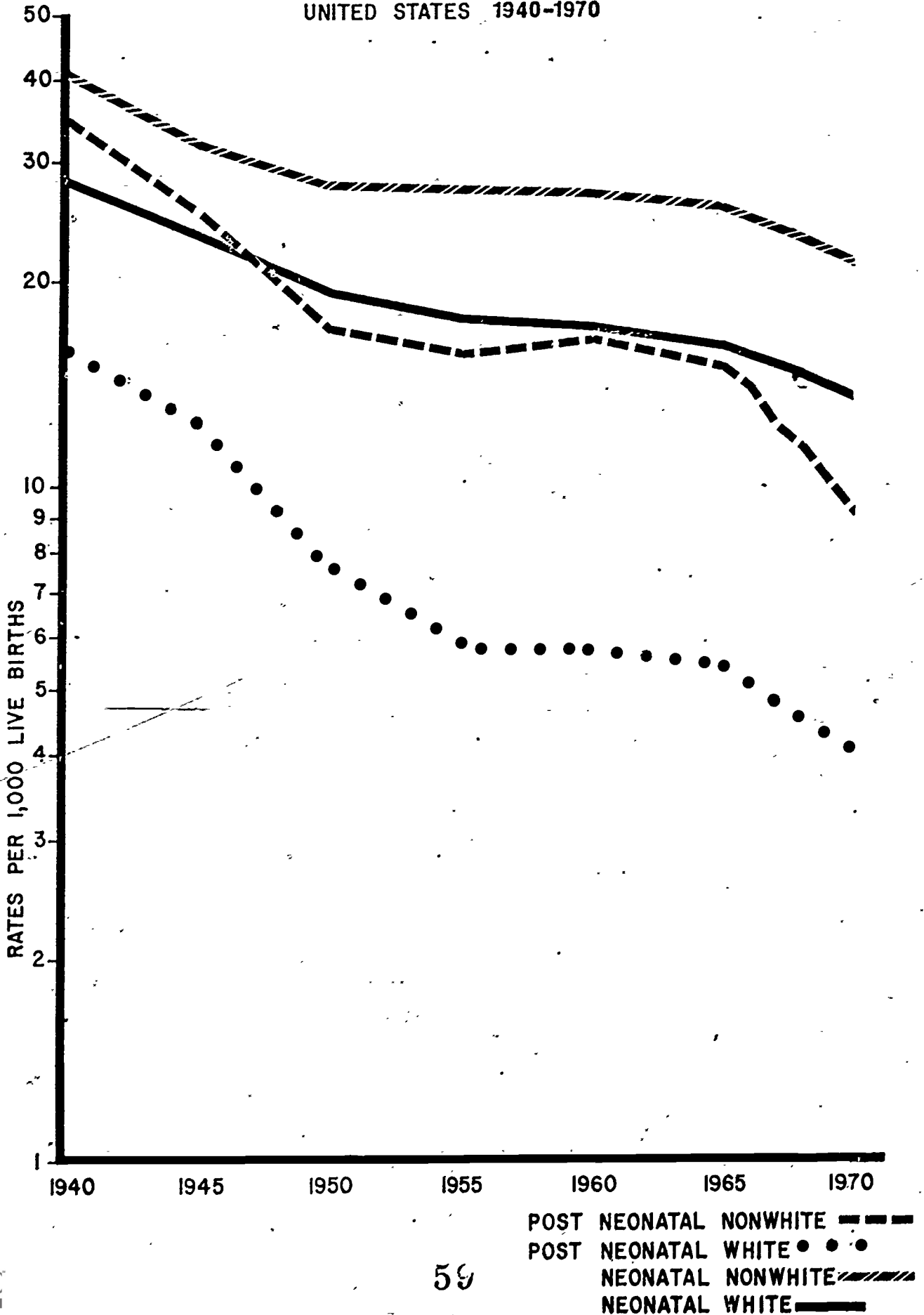
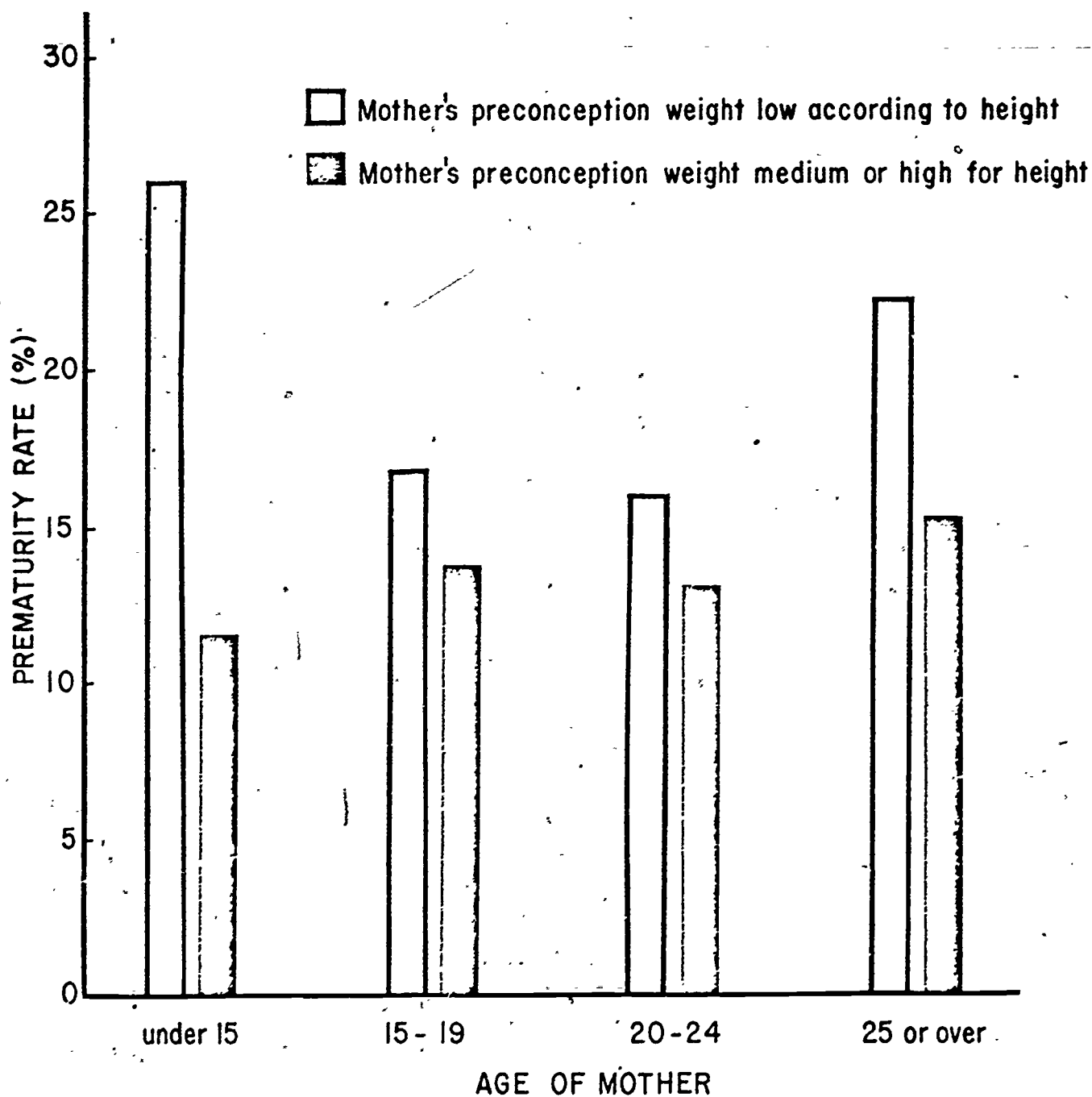


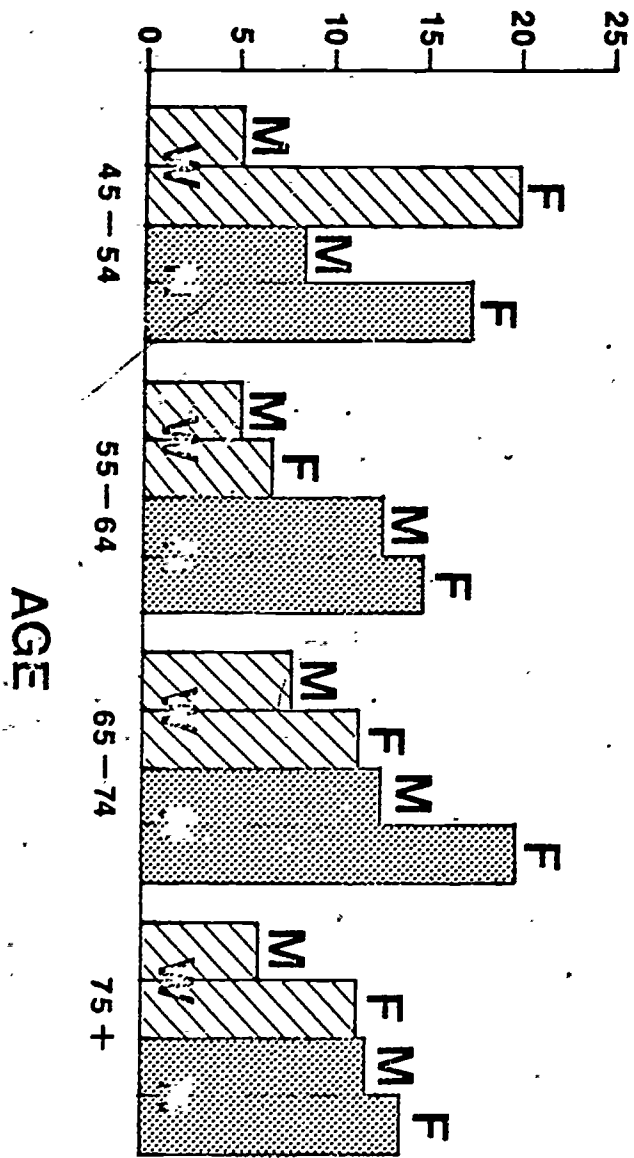
FIGURE 3

PREMATURITY RATES BY MOTHER'S AGE AND STATURE
NINDB COLLABORATIVE PERINATAL STUDY, 1959-1965
BLACK PRIMIGRAVIDAS



Source: Henderson, M., Hebel, R. and Roht, L. Dimensions of teenage pregnancy. Presented to the Society for Adolescent Medicine, Los Angeles, California, May 9-11, 1973.

PERCENT WITH THREE OR MORE SYMPTOMS



**Percent With three Or More Symptoms
by Age, Race and Sex
Baltimore, Maryland, 1967**

FIGURE 4

Source: Mules, Janet, et al. A population survey of symptoms suggestive of transient ischemic attacks. *Stroke* 2:119, 1971.

Age - Sex Adjusted Census Tract Prevalence
of High Blood Pressure by Median Education
Baltimore City, 1971 - 1972

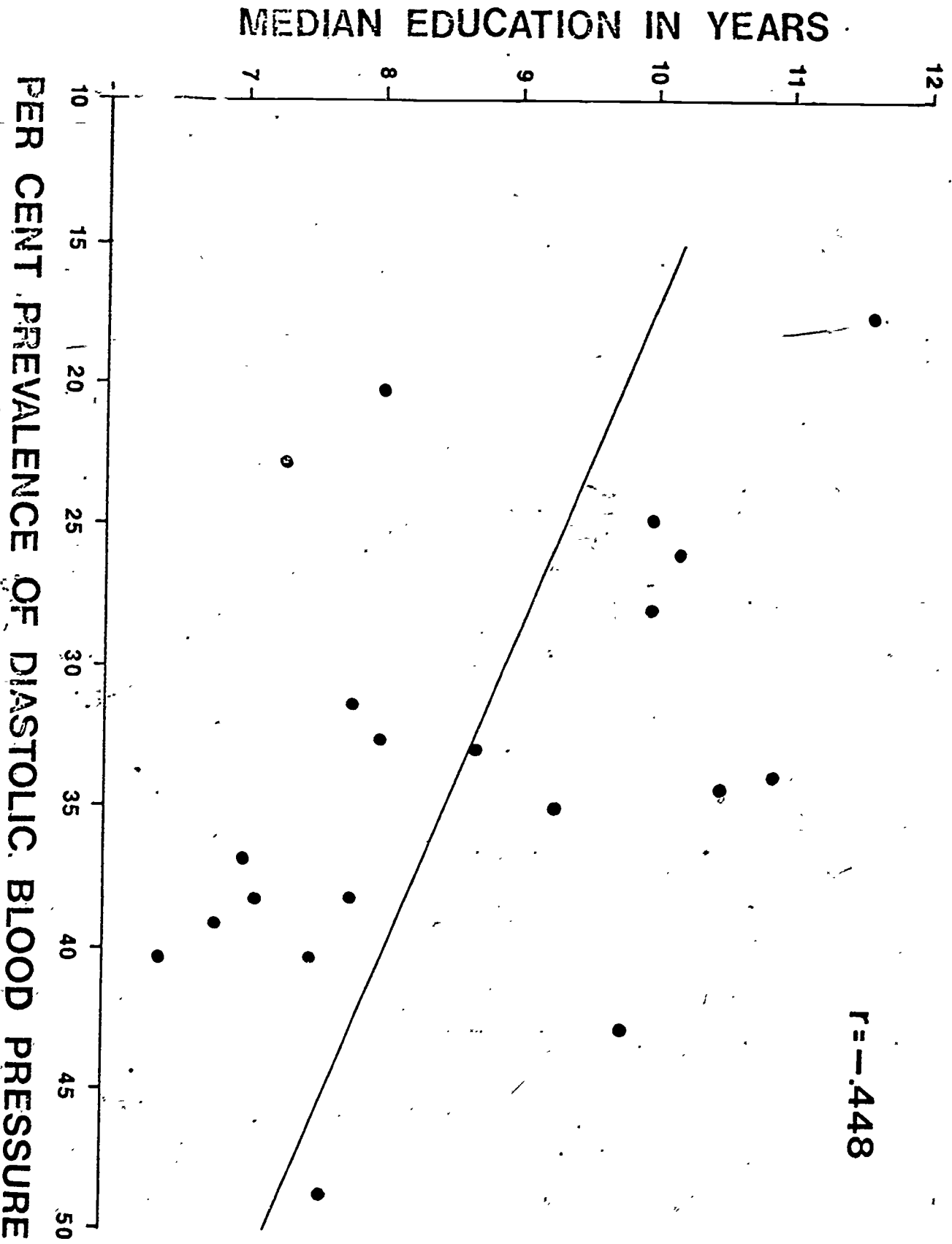
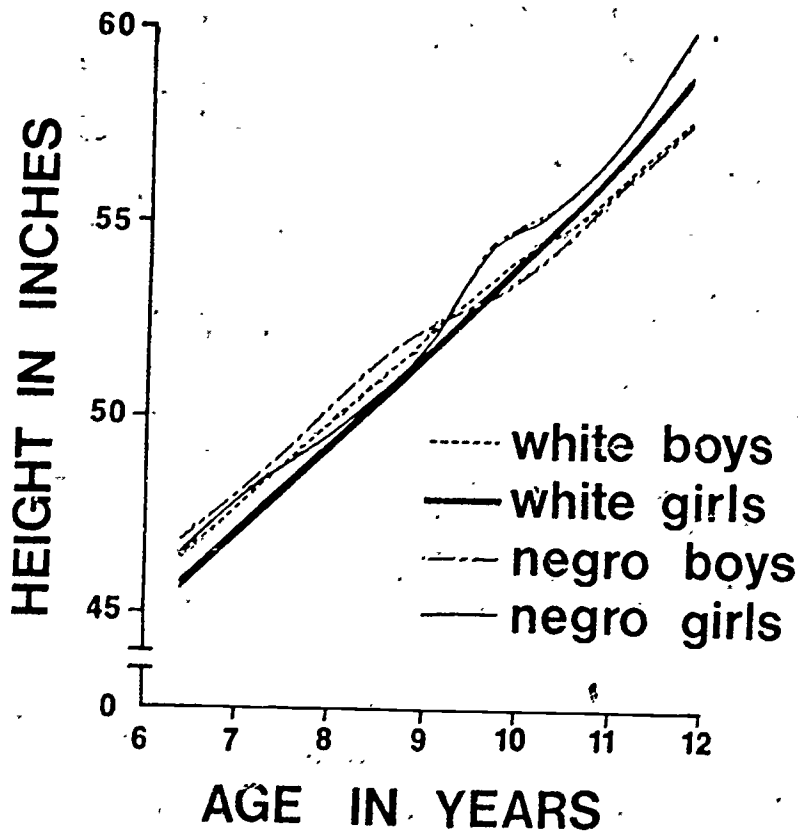


FIGURE 6

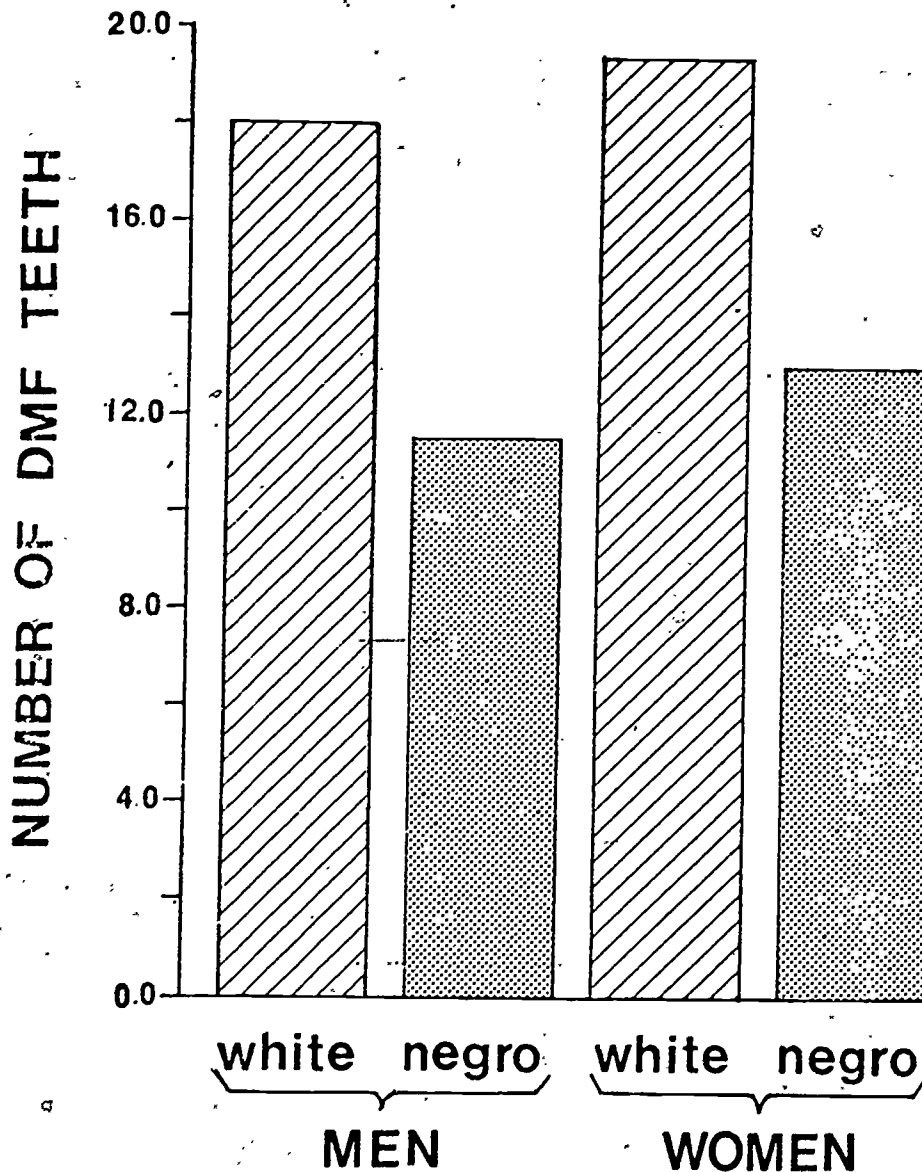
Average Height in Inches of Children
Aged 6-11 by Age, Race and Sex
United States, 1963-1965



Source: National Center for Health Statistics. Height and weight of children: socioeconomic status, United States, Series 11, No. 119, Washington, D.C., 1972.

FIGURE 7

Mean Number of Decayed, Missing,
and Filled Teeth Among Dentulous
Adults by Race and Sex
United States, 1962



Source: National Center for Health Statistics. Decayed missing and filled teeth in adults: United States, 1960-1962, Series 11, No. 23, Washington, D.C., 1967.

Appendix A

Age-Adjustment Computation Procedures Used for Table 1

The ratios of death rates (Black:white) for the selected cities shown in Table 1 were computed by the direct method of adjustment. Considering one city at a time, the age-specific death rates for each ten year interval between the ages of 15 and 64 for each race were applied to the total 1970 United States population in the corresponding age interval. Summing the deaths for each race separately yielded the expected number of deaths between the ages of 15 and 64 for Blacks and for whites. The same procedure was used for each of the nine selected cities. The cities were then grouped in threes from least racial comparability in terms of employment categories (Birmingham, Charlotte and Charleston) to most racial comparability (Baltimore, Philadelphia and New York). The expected deaths for each race were then summed for each of the three city sets. A ratio of the expected deaths was obtained by dividing the age-adjusted death rate for Blacks by the age-adjusted death rate for whites.

APPENDIX B

Number of Respondents in Transient Ischemic
Symptom Survey by Age, Race and Sex
Baltimore, Maryland

1966

Disease	Race, Sex	AGE IN YEARS				ALL AGES
		45-54	55-64	65-74	75 and over.	
STROKE	WM	121	127	542	206	996
	WF	189	196	797	409	1591*
	BM	427	345	213	62	1047
	BF	447	346	246	83	1122*
	TOTAL	1184	1014	1798	760	4756*
HYPERTENSION	WM	121	127	542	206	996
	WF	189	196	797	409	1591*
	BM	427	345	213	62	1047
	BF	447	346	246	83	1122*
	TOTAL	1184	1014	1798	760	4756*

* Excludes age unknown

Source: Mules, Janet, et al. A population survey of symptoms suggestive of transient ischemic attacks. Stroke 2: 122, 1971.

APPENDIX C

Number of Individuals for Whom Diastolic Blood Pressures were
 Measured by Age, Race and Sex
 Social Security Administration
 Baltimore, Maryland
 1969-1974

Age in Years	Male		Female	
	Black	White	Black	White
20-24	62	170	393	688
25-29	135	444	896	834
30-34	67	602	508	339
35-39	61	552	379	197
40-44	92	450	333	213
45-49	124	432	275	426
50-54	107	395	226	563
55-59	55	324	90	449
60-64	31	177	47	220

Source: Sherwin, R., et al. Personal communication, August, 1974.

APPENDIX E

Number of Individuals Invited for Physical Examination
by Race, Sex and Risk Group
Baltimore, Maryland
1967

Risk Group	Male		Female		Total
	Black	White	Black	White	
Disease History					
Stroke	45	45	45	51	186
Heart Disease	37	23	57	48	165
High Blood Pressure	40	24	67	58	189
Symptoms of Cerebral Ischemia					
1 or more focal	32	32	41	38	143
3 or more focal	64	44	99	109	316
2 diffuse	35	30	66	60	191
No symptoms of Cerebral Ischemia	87	54	142	123	406
TOTAL	340	252	517	487	1596

Source: Tonascia, Susan. Personal communication, August, 1974.

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