THE UNIVERSITY OF OKLAHOMA

GRADUATE COLLEGE

AN EPIDEMIOLOGICAL STUDY OF HYPERTENSIVE DISEASE MORTALITY IN OKLAHOMA, 1958-1962

A DISSERTATION

SUBMITTED TO THE GRADUATE FACULTY

in partial fulfillment of the requirements for the

degree of

DOCTOR OF PUBLIC HEALTH

BY

CORNELIUS ASKEW, JR.

Oklahoma City, Oklahoma

1969

AN EPIDEMIOLOGICAL STUDY OF HYPERTENSIVE DISEASE MORTALITY IN OKLAHOMA, 1958-1962

APPROVED BY

Robert D. Ludeman

Wilson A. Steen

Thomas D. Lynn

Claude M. Blass

Albert C. Duncan

DISSERTATION COMMITTEE

ACKNOWLEDGMENTS

The writer wishes to express his deepest appreciation and indebtedness to the following persons: Dr. Robert Lindeman, for his continued and very helpful guidance as an advisor, his assistance and direction throughout this research, and critical evaluation of the entire study. The writer is also greatly indebted to Dr. Wilson D. Steen, Dr. Thomas Lynn, Dr. Robert Duncan, Dr. Claude Bloss and Dr. Paul S. Anderson, Jr. for their valuable assistance throughout his graduate study.

Appreciation is also extended to Dr. Kelly West and the Oklahoma Regional Medical Program for making funds available for the preparation of this manuscript, and to the Department of Biostatistics and Epidemiology for making available the use of the computer facilities throughout this research.

TABLE OF CONTENTS

LIST OF TABLES

LIST OF TABLES--Continued

LIST OF ILLUSTRATIONS

LIST OF ILLUSTRATIONS--Continued

AN EPIDEMIOLOGICAL STUDY OF HYPERTENSIVE DISEASE MORTALITY IN OKLAHOMA, 1958-1962

CHAPTER I

INTRODUCTION AND REVIEW OF LITERATURE

Mortality Statistics

Hypertension may be defined as the disease process characterized by sustained blood pressure elevation, involving diastolic and (in most cases) systolic pressures, cause known (secondary hypertension), or unknown (primary or essential hypertension), with or without clinically demonstrable involvement of blood vessels and their organs (1).

Hypertensive disease is a derious problem because of its complications, principally coronary heart disease, congestive heart failure, cerebral hemorrhage and thrombosis, and renal failure. Mortality data on hypertensive disease tend to underestimate its significance because of rules for coding deaths to single causes, according to the International Statistical Classification of Diseases and Cause of Death i.e., deaths of hypertensive patients are frequently attributed to arteriosclerotic heart disease or cerebrovascular disease (2).

This might possibly be one of the explanations for the current reported downward trend in the death rates from hypertensive disease, however, improved medical management is also an important consideration

in these trends (3). Despite the significant limitations regarding coding, mortality statistics from hypertensive disease give a partial measure of the scope of the problem, and some indication of the major-sex-race patterns described by several investigators (4).

The data further demonstrate the marked racial difference between Negroes and whites in susceptibility to hypertensive disease. Little or no sex differential is recorded in mortality rates for hypertensive disease (5). For whites the mortality rate is slightly lower for women at ages 45 to 54 and 55 to 64 than for men, despite the fact that hypertensive disease is undoubtedly more frequent among women than men from age 45 up (6). The sex differential mentioned earlier is more pronounced in the non-whites than whites. Although the age patterns are similar to those of whites, all reports to date show that Negro females experience higher age-adjusted rates after age 35. Rates as much as five times those experienced by the Negro male have been reported (7). The Negro male rate, although lower than the female rate, is considerably higher than that of the white male. The Negro female in some sections of the country has experienced rates as high as ten times that of her white counterpart (8).

Primary hypertension is widespread in the adult American population. While exact statistics on its prevalence are not available, surveys among selected groups suggest that between 5 and 25 percent of the adult population is affected. Variation in the clinical and laboratory procedures employed in the diagnostic examinations, as well as in the criteria for classification, make it impractical to compare the results of various studies and surveys. However, the results of the

Health Examination Survey for hypertensive heart disease indicate the magnitude of the problem. Definite hypertensive heart disease is estimated to be present in 9.5 percent of American adults, and suspected hypertensive heart disease in an additional 4.3 percent (9).

Definite hypertensive heart disease occurs in 11 percent of adult women and 8 percent of adult men; it is found in 21 percent of adult Negro Americans compared with 8 percent of adult White Americans (10). The prevalence increases sharply with age from about 1 percent at age 30 to about 10 percent twenty years later, with even higher rates above age 65 (11).

Primary hypertension is more common among women, but more severe in men. The onset is usually during the third or fourth decades of life; very few cases occur after the age of 50, although this point has been disputed (12). Even though the use of mortality data for hypertension is hazardous, it appears that this disease may be more frequently a cause of death in urban than rural areas of the United States.

Geographical Distribution

The geographical distribution of age-adjusted death rates for hypertensive disease (ISC 440-447) for whites range widely from 32 per 100,000 population in South Dakota up to 95 per 100,000 in Maryland, approximately a three-fold difference (13). The geographical pattern is more consistent for hypertensive disease than for arteriosclerotic heart disease (14). The lowest rates are confined to a large area in the central part of the country, mainly agricultural states but including both mountain and plain states. The highest rates are all found on the southeastern coast running eastward from Louisiana up to New York and

Massachusetts, with Maryland and the District of Columbia showing the maximum rates. Within the high rate areas, there is considerable variation, and within the states in these and other areas even further variation by counties (15). South Carolina, for example, has a rate that is over 60 percent higher than that for West Virginia and one of the highest in the country (16).

Taking the country as a whole, there has been a significant correlation reported between urbanization and mortality from hypertensive disease (17). The association, however, is far from consistent. For example, it has been reported that hypertensive disease mortality rates for California and Arizona are almost identical, yet there are ten-fold variations in population density. One finds a similar situation when observing mortality rates from the East. For example, New Jersey and Georgia have shown similar mortality rates although their population densities are vastly different (18). Also, it has been reported from the District of Columbia, an entirely urban area, age-adjusted death rates similar to those from rural parts of Maryland. This seems to suggest that factors which determine the mortality rates for hypertensive disease are in many individual instances independent of factors associated with population density.

The distribution of mortality among non-white persons has been reported by several investigators (19). It must be remembered with regard to states in the mountains and plains regions, of which Oklahoma is located, that "non-whites" include other races in addition to Negro. Furthermore, among Negroes there has been a great deal of migration, so that the place of death may often be remote from the place in which most

of life has been spent. This is particularly true for the Northeast central and Pacific regions (20). In California, for example, 56 percent of the Negro population residents at the time of 1950 census had migrated there during the last ten years. Furthermore, in the 1960 census it has been estimated that 10 percent of the Negro population in the country was not included in the census (21).

In general the geographical patterns of mortality are very similar to those observed for whites, although both the rates themselves and the magnitude of variation are at a much higher level. The highest rate was (445 per 100,000) population in South Carolina, 1960 and the lowest rate (74 per 100,000) in Arizona for the same period, a six-fold difference (22). Thus the lowest rates for non-white persons are a little lower than the highest rates for the white population. Evidently the racial differences in mortality are not determined wholly by the genetic make-up of the population.

The mortality patterns of the non-white population does not seem to correspond in any obvious way with economic development (23). Arkansas, where the Negro's status is low, has reported low rates; New York, where the Negro status is higher, has shown moderately high rates; but South Carolina, with a low economic status has the highest of all (24).

Environmental Factors

Several environmental factors have been reported as having an effect on mortality from hypertension and related cardiovascular diseases, however, water hardness and socio-economic status have been the two most reported in relation to mortality from cardiovascular disease. Other

factors will be discussed in connection with disease prevalence.

Water Hardness

Several studies suggest that the softer the water the higher the cardiovascular death rate. Low cardiovascular disease death rates have been associated with hard water. The Japanese drew attention to the remarkable high death rates from cerebral hemorrhage in areas where the river waters tended to be acid, and where the sulphate-bicarbonate ratio was higher than 0.3 (25). The data showed a fairly high correlation between death from apoplexy and acidity of river water and between death rates from heart disease and water composition (26). On the other hand significant negative correlations have been reported between water hardness of several local water supplies and cardiovascular disease death rates in the United States and the county boroughs in England and Wales (27, 28). A similar study of local water supplies in Oklahoma failed to yield similar results (29).

Three possibilities have been suggested as accounting for the low cardiovascular mortality associated with hard water (30): (1) there is something beneficial in hard water; (2) there is some adverse factors in soft waters; (3) soft waters and acid waters attack pipes through which they run, taking up zinc, cadmium, copper and lead depending upon pipes and solders used. Cadmium or lead might be suspected as both have been shown by several workers to accumulate in human tissue (31). These possibilities require further investigation before definite conclusions can be drawn.

Attempts to find what factors in water hardness might be associated with lower death rates were successful mainly in exclusion of

several factors. The influence on cardiovascular mortality appeared not to depend on the amounts of iron, manganese, carbonate and nitrate, nor was there apparent dependence of death rates on the sodium and potassium content of water (32). However, significant correlations were found for magnesium, calcium, bicarbonate and sulphate, fluoride, dissolved solids and pH. In all cases, the correlations were negative in that soft water was associated with higher death rates (33).

Socio-Economic Status

Hypertensive disease mortality in different socio-economic and occupational groups in the United States have been published recently by several investigators (34, 35). As was noted in the cited patpers and elsewhere, many problems exist in the validation and interpretation of these data. For white males, no occupation group had a standardized mortality ratio sizeably different from that of all white males. For Negro males exhibiting the expected high death rates from hypertensive disease, a considerable range of standardized mortality ratios were registered among the several occupational groups. The laborer, and professional-technical groups tended to have ratios considerably above that for all non-whites; the managerial-professional-proprietary, the clerical, the sales, and operative groups had ratios below those for all non-whites. These findings have been validated by studies in the living population (36).

Income also has been used as a socio-economic index in observing mortality from hypertensive disease. Several studies have been reported in which death rates were similar for all income strata of a given sex color group, except for a higher rate in non-white females with a median

income of $3,000 - 3,900 (37). For the age group 55-64, the mortality rates for hypertensive disease tended to be lower in the higher income whites; for non-whites, they were lowest in the lowest income group. Overall, as expected, rates for non-whites (Negroes) far exceeded those for whites. Whether the differences among income classes of specific sex-color groups were artificial or real cannot be determined from mortality studies, but in depth studies among representative samples of the living population have revealed findings similar to those described in the mortality studies (38).

The Society of Actuaries and the Metropolitan Life Insurance Companies have sponsored several studies on "Blood Pressure and Mortality" and these studies have been of particular value to medicine because they have provided the largest body of data ever assembled bearing on the prognosis of hypertension in its pre-clinical stages. These studies show that men with casual blood pressures of 140mm. systolic and 90mm. diastolic, who are free of other impairments, are subject to about 50 percent extra mortality as compared with the average of insured lives cver a period of about 20 years (39). Men with casual blood pressures of 145mm. systolic and 95mm. diastolic are similarly subject to about 100 percent extra mortality, while those who have blood pressures of 160mm. systolic and 100 mm. diastolic experience 200 percent extra mortality.

Death Certificate Data

In a death certificate study of this type it seems appropriate to briefly discuss some of the problems associated with the use of death certificate data. Several attempts to determine the accuracy of the underlying causes of death on the death certificate have been made and

might be divided into three categories (40).

1. A query of physicians who signed the death certificate to determine the criteria of diagnosis, followed by a comparison between the underlying cause reported on the death certificate and a diagnosis made by another physician using information provided by the physician signing the certificate at the time of death.
2. An evaluation of the cause of the death statement by reviewing available medical information and comparing the cause of death as determined independently by two or more physicians. This method differs from the first only in that records are reviewed rather than using information supplied by the physician who certified the cause of death.
3. A comparison of the pathology observed at autopsy with the underlying cause of death. This type of study is limited by the selectiveness, according to the circumstances of death, with which autopsies are performed and by the lack of correlation between certain clinical diagnosis and pathological observations.

All attempts to measure the accuracy of arteriosclerotic heart disease, hypertensive disease, and other cardiovascular diseases as the underlying cause of death are difficult for several reasons (41).

1. A high proportion of deaths especially attributed to arteriosclerotic heart disease are sudden and unexpected, and frequently both the clinical and pathological information are meager;
2. Because of the association of several cardiovascular diseases in the same person, the choice of single underlying causes of death is frequently arbitrary;

3. A high proportion of deaths attributed to arteriosclerotic heart diseases and other cardiovascular diseases are not certified by the patient's personal physician but by the medical examiner or coroner. Changing patterns as well as variations in certification by medical examiners among communities may influence both geographic difference in rates and trends in mortality attributed to cardiovascular diseases.

It also has been suggested that inaccuracies in cause of death data may occur because the physician does not list the available material correctly on the death certificate. Another problem in regard to these statements concerns the insufficient clinical and laboratory data before autopsy to enable the physician to determine the cause of death correctly. In some cases where all information, including the autopsy data, is available, it is still difficult to decide on one underlying cause of death. In using death certificates to study mortality from hypertension and hypertensive heart disease (ISC 440-447), one is not only faced with the problem just recited, but with an additional problem of having deaths from hypertension attributed to the other major cardiovascular-renal diseases as previously mentioned.

Prevalence and Incidence Data

Several factors have been related to the prevalence and incidence of hypertension. Community studies and clinical observations have suggested an association of primary hypertension with a number of factors including diet (particularly salt intake), occupation, psychological and social characteristics, smoking and heredity (42). These topics can be summarized very briefly, since there is a lack of data to identify clearly

the etiological importance of any of these variables.

Salt

Salt has been incriminated as one of the factors in the causation of hypertension. In 1940, Ambard and Benjard observed that restriction of sodium chloride might ameliorate human hypertension (43). Since that time a substantial body of animal experimentation and human observational data have been accumulated. It is now well established that diastolic hypertension can be induced in animals by the continued administration of large and excessive amounts of salt. Several investigators in carefully designed experiments in rats found that by adding measured amounts of salt over a ten month period, hypertension was well established in rats who were ingesting higher levels of salt (44). To confirm the above and similar experiments, various epidemiological studies on human beings have been carried out. An association with salt ingestion has been reported by Dahl; observations in other human studies have been contradictory (45).

Diet

Diet may affect blood pressure, indirectly through its effect on body weight, and directly through the effect of one or several of its nutrients. Weight seems to carry a positive relationship with blood pressure levels as attested by numerous statistical studies carried out by Huber, Symond, Masters, and others (46). These studies have conclusively shown that blood pressure is significantly higher in obese persons than in those who are average weight or under weight. Boynton and Todd (47) mentioned that overweight appears to be a factor in the

causation and the increased prevalence of hypertension. A careful study of Dawber indicated that one half of the hypertensive patients he studied were obese, while only three percent were below normal weight (48). The association of overweight and hypertension has been noted in several studies (49) and found to be most severe among women when there was definite obesity (50).

Occupation

Several recent studies conducted in the Soviet Union suggested the importance of occupation in the etiology of primary hypertension (51), and presented evidence indicating that the course of the disease may be modified by changes in occupation or living conditions. Bechgaard (51) suggests that studies in these areas should be extended in order to explore the implications of these observations for both the understanding of etiology and the control of the disease.

Heredity

Differences in the prevalence and incidence of primary hypertension in the general population has been related to the genetic differences in the population by several investigators (52, 53). Platt in his study of identical twins showed only essential hypertension to be genetic and the more severe forms not to be (54). He suggests that the single gene theory might be the operative mechanism in essential hypertension, and further suggests that more needs to be known about the causes and behavior of essential hypertension. It has been postulated by several workers (31, 53, 54) that one might find that there are two populations and not a single continuous one; that there are people, in

other words, who by their genetic constitution are liable to develop hypertension in middle age and others who are not (55, 56). Platt and others think that some day investigators will be able to separate them when better methods of studying the problem are available. Sir George Pickering and other investigators believe genetic factors are only of secondary importance and that the relative contribution of inheritance and environment should be considered (57).

The pattern of familial concentration has been noted by many clinical investigators, and has been observed in a variety of family studies (58, 59). Family studies have made and can make greater contributions to our understanding of the onset of the disease (60, 61). Such studies may be able to provide clues which might indicate what factors are necessary in some cases of primary hypertension but not others.

Psychological and Social Factors

Although the psychological and social factors hypothesis has been under investigation for several decades, only a few studies have been conducted using standardized psychological tests and measurements (62, 63, 64). The evidence obtained regarding personality and social-cultural factors in the etiology of primary hypertension has been fragmentary (65, 66). Data regarding psychological variables and blood pressure can be summarized as follows:

1. It has been established beyond a reasonable doubt that acute psychological stress may initiate sudden and transient elevations of blood pressure in some persons.
2. It has not been established that repetitive or continuous psychological stress leads to sustained elevation of blood pressure in

anyone.

Essentially, three kinds of investigations have attempted to relate psychological variables to blood pressure. Some of them were described or summarized at the Prague Conference (67), and will be briefly discussed here.

Lang (68) suggests that essential hypertension is initiated by alterations in the normal function of the cerebral cortex. Experimental attempts to support this theory have consisted of the establishment of conditional vascular reflexes in animals, and in man by observing workers and patients living under allegedly difficult circumstances. In the Soviet Union and neighboring countries, the view of Lang is widely held.

In the United States the emphasis has been on case studies of the personalities of hypertensive patients with or without comparison groups. The methods employed were usually interviews, with sometimes concomitant recording of cardiovascular functions. There is a consistent theme in this work; the belief that two types of psychological conflict are more characteristic of hypertensive persons than others. These are: (a) a conflict between passive, dependent longings and aggressive, competitive impulses, and (b) a conflict between chronic anger, close to conscious awareness, and a need to repress these feelings for fear of loss of love and esteem (69).

The third kind of pertinent study has been geographically widespread in origin. Populations exposed to disastrous events have had their blood pressures measured after the event and unusually high elevated blood pressures have been observed for a time. More recently, better epidemiological surveys of groups of people living in different social

environments have provided a suggestion of an association between cultural change, degree of urbanization, and prevalence of higher blood pressures (69).

American workers have usually employed human subjects in studying psychological factors in hypertension. Among the more widely known works are the psychoanalytic studies of Alexander (70), and Saul (71), the physiological observations of Stewart Wolf and associates (72) and the papers of Harris, ostfeld, and Sokolow (73, 74, 75). In these studies and others closely related, there is also telling evidence that blood pressure is higher during threatening events than during periods of tranquility.

The studies of groups exhibiting unusually high or low prevalences of high blood pressure are also well-known. Some, such as the Texas City disaster (76), or the experience with high blood pressure in British troops in North Africa (77) are of importance, however, the results of these studies appear to be inconclusive. Recently, more complete observations have shown differences in the point prevalence of high blood pressures, and in the probable rate of rise of pressures with age in different groups with different degrees of urbanization and industrialization. The studies of Lowell (78), Lowenstien (79), and Scotch (80), are particularly germane. These authors have pointed out that differences in race, genetic endowment, body bulk, nutrition and prevalence of infections and anemia may be important determinants of the differences in blood pressure, and that the contribution to these differences of cultural stress cannot be specified except, quite possibly, in Scotch's work (80). In this work, he showed Negro-White differences

and urban-rural differences to be related to various environmental or socio-cultural factors. Scotch found statistically significant correlations between hypertension and the following:

1. Age
2. Sex
3. Menopause
4. Marital status
5. Length of residence
6. Obesity
7. Family type
8. Urban and rural communities
9. Church membership (including attendance)
10. Income

The effects of education, smoking, and alcohol intake are discussed briefly in terms of their relationship in the development of hypertension.

Education

Level of education materially affects the occupation, the physical activity and many other aspects of the way of life of the individual.

Although educational status does not markedly affect the blood pressure distribution, it is interesting that the mean blood pressure of the most highly educated group regardless of sex is less than that of the less educated in all studies reported to date. These differences were reported to be statistically significant at the five percent level (81).

Smoking

That the smoking of tobacco has an immediate effect on the cardiovascular system has been recognized for many years. The effects of both nicotine and the actual smoking of tobacco have been well studied. From such studies it is apparent that the cardiovascular changes produced by smoking tobacco are those brought about by the nicotine so administered. The greatest effect is noted when cigarette smoke is inhaled. Lesser effects are found with non-inhalation of cigarette smoke or the smoking of pipe tobacco or cigars due to the relatively slight inhalation of these smokers.

The immediate effects of smoking are an increase in pulse rate, slight increase in blood pressure, increase in cardiac output and peripheral resistance, and changes in electrocardiographic patterns. There have been many studies of the immediate effects of cigarette smoking or nicotine administration on blood pressure. The results are variable, but in general, both in animals and man, there is a rise in systolic blood pressure of 5 - 35mm. of Hg (82). On the other hand, there may be a drop in blood pressure or no effect. Diastolic blood pressure was only slightly affected (82).

Although prospective studies of the long-term effects of smoking have been only recently undertaken, it has been demonstrated that cigarette smoking is accompanied by a definite increase in the risk of dying from coronary heart disease and in developing myocardial infarction. Determination of this fact has prompted further investigation regarding the mechanism of this effect.

From the investigations in Framingham and Tecumseh, it appears

quite clear that the effect of cigarette smoking on the production of myocardial infarction and death from coronary heart disease was not through any effect on the blood pressure level. This does not rule out the possibility that the immediate temporary effect of cigarette smoking in raising blood pressure might be a factor in the precipitation of myocardial infarction and death from coronary heart disease.

Alcohol Intake

The effect of alcohol on the cardiovascular system has been the subject of controversy and conflicting reports. Although many investigations have been carried out, no consistent effect has been found.

Jellinek and Haggard state that in alcoholics, a reflexed elevation of blood pressure occurs initially which is presumably related to the agitated state of alcoholics (83). However, later with sedation, lower blood pressure has been noted. Grollman observed that in non-drinkers, small amounts of alcohol caused transient elevations in cardiac output, pulse rate, and blood pressure, and larger doses further elevated blood pressure and increased cardiac output (84). Abelman, Kowalski, and McNeely found that alcoholics had a normal cardiac output at rest, normal arterial pressure and peripheral resistance (85). In an analysis of several alcoholics, Schmall and Wiener conclude that alcoholics have both less hypotension and less hypertension than the general population (86).

In the Framingham study information was obtained relative to usual alcohol consumption. The amount of beer, hard liquor and wine consumed was recorded, using a period of a month as the time of reference.

No significant effect of alcohol on blood pressure was noted in any age or sex group, but appeared to be a slight increase in the blood pressure of persons consuming higher amounts. However, it is likely that weight may have had some effect in producing these slight increases in blood pressure rather than the high alcohol intake.

The objective of this study will be to determine those factors which contribute to the differences in rates between the white and non-white population in Oklahoma by use of death certificate data. More specifically, the study will attempt to determine the following:

1. Whether or not the geographic distribution of hypertension and hypertensive heart disease varies within the state.
2. Whether or not death rates and prevalence rates can be related to various demographic data such as reace, age, sex and county of residence.
3. Whether or not death rates for associated cardiovascular diseases (vascular lesions affecting the central nervous system) (ISC 330-334), arteriosclerotic and degenerative heart disease (ISC 420-422), and nephritis and nephrosis (ISC 590-594), also vary directly with hypertensive death rates.

CHAPTER II

METHOD AND PROCEDURES

The data for this study include all deaths from hypertension (ISC 440-443), hypertensive heart disease (ISC 444-447), vascular lesions affecting central nervous system (ISC 330-334), arteriosclerotic and degenerative heart disease (ISC 420-422), and nephritis and nephrosis (ISC 590-594), of Oklahoma residents occurring in the state during the five year period, 1958-1962. In this five year period, there were 51,557 deaths from these diseases among Oklahoma residents.

Death certificate data were obtained from coded tapes of the Vital Statistics Office of the Oklahoma State Health Department. Data regarding the prevalence of hypertension for the period under study were obtained from the Oklahoma Chronic Disease Screening Survey Program conducted by the state health department in fourteen counties of the state. However, only eight of these counties were used in this study as the remaining six did not have Negroes participating in the screening program.

The history and organization of the chronic disease screening program at the Oklahoma State Department of Health has been described by Adler, Bloss and Mosley in two reports (87, 88). The first report described the general characteristics of the screened population and the second gave a detailed analysis of the screening procedure. The racial participation of the screenees occurred in the following fashion; with

the white female taking part more than any other group and Indians slightly more than Negroes.

Blood pressure screening has been part of the multiphasic program in Oklahoma since 1959. A nurse took the blood pressure with a standard mercury manometer using a 14" cuff, on a mobile unit (87, 88). Referral levels were blood pressures of 160/100 or higher for people under 65 years old and 180/100 for people over 65 years of age. While these levels are somewhat higher than those recommended by the World Health Organization and other official agencies, it was the contention of the investigators that a large number of false-positive referrals would result from using lower levels.

Age-race-sex-specific and age-adjusted prevalence rates were calculated for each county. The age-adjusted rates were used to make comparisons between the eight counties regarding the prevalence of the disease in the three racial groups in the eight counties. The following eight counties were used in this study:

Roger	McClain
Cherokee	Hughes
Delaware	Seminole
Bryan	Jackson

Mortality data were taken from the coded tapes of the State Health Department with respect to the following items.

1. Age
2. Sex
3. Race
4. Residence

   Deaths were attributed to the county of residence, not the county in which the patient died. Mortality data from

out-of-state residents was not included in the study.

5. Underlying cause of death

The deaths from hypertensive disease and related cardiovascular-renal diseases were sorted in specific catagories using the 1955 International Statistical Classification of Diseases (89). Data were analyzed on the following classifications:

1. Cerebrovascular Disease (ISC 330-334) including the following:

   330 Subarachnoid hemorrhage

   331 Cerebral hemorrhage

   332 Occlusion of precerebral arteries

   333 Cerebral embolism and thrombosis

   334 Other and ill-defined vascular lesions affecting the central nervous system

2. Arteriosclerotic Heart Disease (ISC 420-422) including the following:

   420 Arteriosclerotic heart disease, including coronary disease

   Arterioscleratic heart (Disease)

   Heart disease specified as involving coronary arteries

   Angina pectoris without mention of coronary disease

   421 Chronic endocarditis not specified as rheumatic

   422 Myocardial degeneration

   Fatty degeneration

   Others

3. Hypertensive Disease (ISC 440-447) including the following:

   440 Hypertensive heart disease

   441 Other unspecified hypertensive heart disease

   442 Heart involvement with malignant hypertension

   443 Heart involvement with benigh hypertension

   444 Essential benign hypertension

   445 Essential malignant hypertension

   446 Hypertension with arteriular nephrosclerosis

   447 Other hypertensive disease without mention of heart

4. Nephritis and Nephrosis (ISC 590-594) including the following:

   590 Acute nephritis

   591 Nephrotic syndrome

   592 Chronic Nephritis

   593 Nephritis, unqualified

   594 Renal sclerosis, unqualified

Age-race-sex-specific and age-adjusted death rates were computed for the state during the period under study. Average annual age-adjusted death rates were calculated for each county in the state, and for each race-sex group for all underlying causes considered and plotted on state maps. These maps were used to show the geographical distribution of the average annual age-adjusted death rates by disorder. Cluster areas within the state from all diseases considered were also identified from these maps. A cluster area for purposes of this study will be considered as any area where there are four or more adjacent counties appearing in the upper quartile. All counties of the state were divided into quartiles,

and the average annual age-adjusted death rates for these counties were shown on state maps.

The 1960 Oklahoma population was used as the standard population in computing all rates for both prevalence and mortality data.

The median age-adjusted death rates were used to determine the differences in mortality from hypertensive disease (ISC 447) for each race-sex group in the four socio-economic districts of the state. The socio-economic status in the different geographic districts were determined by the United States Department of Agriculture and revised by Assal (90). The following determinants were used: educational level, median per capita income, condition of housing, and number of persons per 100,000, receiving aid to dependent children (ADC). The following methods were used to determine the socio-economic status of the nine districts and two metropolitan areas in Oklahoma as described by Assal. Each district was assigned a rank for each of the four determinants such as one for the districts belonging to the higher per capita income, high level of education and housing and low number of children receiving ADC. The number two was assigned to districts belonging to the class above state average, three to the group below state average and four to districts belonging to low classes. The value for the four determinants for each district were totaled according to the following:

1. High socio-economic state, those districts totaling 4 - 7
2. Above state average, those districts totaling 8 - 9
3. Below state average, those districts totaling 10 - 14
4. Low socio-economic state, those districts totaling 15 or more.

Urban-rural differences were observed by dividing the state into

rural or urban based on the 1960 population of these counties. All counties with populations exceeding 25,000 persons were considered urban and those populations less than 10,000 persons were considered rural. The counties between these figures were considered neither rural nor urban and were not considered in certain specific analysis. Based on this standard the ten most urban and the ten most rural counties were selected, and the age-adjusted rates by race were analyzed.

Machine sorters and computers were available for analysis of the data. Non-parametric statistical techniques were used in the analysis of the data. The chi square test for two independent samples was used to compare the racial distributions of the previously mentioned prevalence data. Spearman's Rank Order Correlation (91) was used to determine the degree of association between the age-adjusted death rates for hypertensive disease (ISC 440-447) and cerebrovascular diseases (ISC 330-334); hypertensive disease (ISC 440-447) and nephritis and nephrosis (ISC 590-594); and hypertensive disease (ISC 440-447) and arteriosclerotic and degenerative heart disease (ISC 420-422). The median test was used to determine if the non-white/white ratios obtained from the age-adjusted death rates in the ten most populated counties were from the same population of ratios as that of the ten smallest counties. All tests of significance were at the five percent level (91).

CHAPTER III

RESULTS

Prevalence Data

Data concerning the prevalence of high blood pressure (hypertension) as obtained by the Chronic Disease Screening Program of the Oklahoma State Health Department is presented in Tables 1 through 10.

Table 1 shows the number of screenees by county and level of blood pressure. There were 15,593 persons visiting the units, of which 2,744 or 17.59 percent were known to have high blood pressure. These persons were not screened because they were already under the care of a physician. The known high blood pressure groups were included because the authors are interested in total prevalence for the period under study. Hughes, Seminole, and Jackson counties show the highest percent of known blood pressure cases with 24.17, and 23.37, and 22.35 percent respectively, while Cherokee and Roger counties show percentages of 2.36 and 7.84 respectively, the lowest percent of known high blood pressure. The remaining three counties Delaware, Bryan, and McClain remain within these ranges. Of the total number screened, 12,859 or roughly 80 percent of the total screenee population, only 7.75 percent were shown to have high blood pressure. The percent of newly detected high blood pressure ranges from a low of 1.11 percent for Jackson county to a high of 22.79 for Rogers county. The mean total prevalence of high blood

TABLE 1

NUMBER OF SCREENEES BY COUNTY AND LEVEL OF BLOOD PRESSURE

COUNTY	No. of Persons Visiting Units	Known HBP*	% HBP	Screenees Studied				Total % HBP
				No. HBP	Normal BP	TOTAL	% HBP	
ROGER	2,395	188	7.84	503	1,704	2,207	22.79	30.63
CHEROKEE	1,312	31	2.36	96	1,185	1,281	7.49	9.85
DELAWARE	1,511	299	19.78	27	1,185	1,212	2.22	22.00
BRYAN	2,355	450	19.10	33	1,872	1,905	1.73	20.83
McCLAIN	1,617	277	17.13	176	1,164	1,340	13.13	30.26
HUGHES	1,752	433	24.17	73	1,246	1,310	5.57	29.74
SEMINOLE	2,575	602	23.37	61	1,912	1,973	3.09	26.46
JACKSON	2,076	464	22.35	18	1,594	1,612	1.11	23.46
TOTAL	15,593	2,744	17.59	997	11,862	12,859	7.75	23.92

*Those persons with known hypertension, already under the care of a physician and were not screened.

pressure in all eight counties is 23.92 percent, with Roger, McClain, and Hughes counties showing slightly higher total prevalence rates than the remaining five counties.

Table 2 indicates the number of screenees by race and level of blood pressure. As previously mentioned, more whites participated than Negroes as is clearly shown in this table.

Table 3 contains the age-race specific prevalence rates per 100 screenees for all eight counties combined. The rates are higher for Negroes in all age intervals considered, when compared with the other two groups. The lowest rates are experienced by whites, with Indians showing slightly higher rates than those for whites but considerably lower than those for Negroes. All rates tend to increase with age in all three racial groups, with the highest rates appearing in the 65+ age interval in all three groups.

Age-race specific prevalence ratios for all counties studied are presented in Table 4, these ratios are based on the rates presented in Table 3. The highest ratios are shown for Negro-White differences, while the lowest are seen for the Indian-White differences. The Negro-White differences, Negro-Indian differences, and the Indian-White differences all seem to decrease with age, with the greatest decrease shown for the Indian-White difference.

Table 5 represents age-race-sex specific prevalence rates per 100 screenees for all counties studied. These rates seem to be higher among Negroes, with the Negro female showing the highest rates and the White male experiencing the lowest.

These rates also seem to increase with age in the three racial

TABLE 2

NUMBER SCREENED BY RACE AND LEVEL OF BLOOD PRESSURES IN COUNTIES STUDIED

RACE	No. Screened for High Blood Pressure	No. Normal	No. High	No. Known High Blood Pressure
WHITE	14,503	11,050	919	2,522
NEGRO	420	286	42	125
INDIAN	670	512	26	137
TOTAL	15,593	11,862	987	2,744

TABLE 3

AGE-RACE SPECIFIC PREVALENCE RATES PER 100 SCREENEES
FOR ALL COUNTIES STUDIED

AGE	WHITE	NEGRO	INDIAN
20-34	5.58	29.26	13.79
35-44	10.77	25.00	17.39
45-54	20.79	40.00	18.34
55-64	33.38	48.18	38.12
65+	43.63	68.88	45.55

TABLE 4

AGE-RACE SPECIFIC PREVALENCE RATIOS
FOR ALL COUNTIES STUDIED

AGE	NEGRO/WHITE	NEGRO/INDIAN	INDIAN/WHITE
20-34	5.24	2.12	2.47
35-44	2.27	1.43	1.58
45-54	1.92	2.18	.88
55-64	1.44	1.26	1.14
65+	1.57	1.51	1.04

TABLE 5

AGE-RACE-SEX SPECIFIC PREVALENCE RATES PER 100 SCREENEES
FOR ALL COUNTIES STUDIED

	WHITE		NEGRO		INDIAN	
AGE	Male	Female	Male	Female	Male	Female
20-34	7.09	5.02	-	27.02	-	-
35-44	11.39	10.43	-	18.86	-	16.45
45-54	19.25	28.40	32.25	45.58	20.00	21.95
55-64	30.42	35.00	39.53	53.84	39.58	38.46
65+	43.41	53.91	44.73	65.90	47.05	56.06

groups, however, Negroes tend to show consistently higher rates than the other two groups even in the older age groups. In the less than 35 age groups, the white female shows the lowest rate, 5.02 per 100 screenees, while the Negro female shows the highest rate 27.02 per 100 screenees, a five-fold difference. There were no Indian rates in these age groups.

The total 1960 Oklahoma population was used as the standard for the age-adjusted rates presented in Table 6. Also shown in this table is the percent of each racial group in the counties considered. The rates are shown for each county with Rogers County experiencing the highest rate, 25.66 and Delaware County the lowest rate, 17.57 per 100 screenees. The racial composition of all counties considered is more than 80 percent white in all cases, with Negroes and Indians constituting only a small percent of these populations. The two counties showing the highest age-adjusted rates, 25.66 and 24.11 respectively, also show the highest percent of white persons in their populations, while Negroes and Indians constitute less than six percent of both populations. Delaware County, which has the lowest rate of all the counties presented, shows 15.85 percent Indian, 0.03 percent Negro, and 84.12 percent white.

The age-race adjusted prevalence rates per 100 screenees for all counties studied are shown in Table 7. The white rate (20.14) per 100 screenees is the lowest while the rate for Indians is just slightly higher (24.14), with the Negro showing the highest rate at 40.15 per 100 screenees.

The prevalence ratios for all eight counties are presented in Table 8 by race; Negro-White, Negro-Indian, and Indian-White. These data show that even with the combined data, a two-fold difference between

TABLE 6

*AGE-SEX-RACE ADJUSTED PREVALENCE RATE PER 100 SCREENEES
BY COUNTY AND PERCENT OF RACIAL GROUPS
IN COUNTY POPULATION

COUNTY	% White	% Negro	% Indian	Rate
ROGERS	94.17	1.97	3.86	25.66
CHEROKEE	90.63	1.58	7.79	20.93
DELAWARE	84.12	.03	15.85	17.57
BRYAN	95.41	2.26	2.33	19.46
McCLAIN	96.81	2.25	.94	24.11
HUGHES	86.13	4.63	9.24	22.28
SEMINOLE	81.86	9.79	8.35	21.05
JACKSON	93.21	6.36	.43	20.97

*Standard Population - Oklahoma, 1960.

TABLE 7

*AGE-RACE ADJUSTED PREVALENCE RATES PER 100 SCREENEES FOR ALL COUNTIES STUDIED

RACE	RATE
WHITE	20.14
NEGRO	40.15
INDIAN	24.45

*Standard population used in adjustment, total 1960 population. The direct method of adjustment was used.

TABLE 8

PREVALENCE RATIOS FOR ALL COUNTIES STUDIED

RACE	NEGRO/WHITE	NEGRO/INDIAN	INDIAN/WHITE
Ratio	2.00	1.61	1.21

Negroes and whites still prevails, however, the Negro-Indian difference is not as great. The combined ratios in all cases tend to follow the patterns seen in Table 4.

The frequency distributions in Table 9 and 10 are based on the age-adjusted rate of each county studied. These rates have been described in Table 6. The entries in Table 9 were obtained by applying the age-adjusted rates for each county to that county's total screenees in order to estimate the expected number of people with high blood pressure. If the age-adjusted rates were essentially equal among the counties then the relative frequency distribution of the adjusted number of screenees with high blood pressure should also be uniform among the counties. The data yield a chi square value of 34.37 with seven degrees of freedom. The probability associated with this value is less than .05, which suggests that the prevalence of high blood pressure differs in these counties.

Table 10 was obtained in the same manner as Table 9. If the age-adjusted rates were essentially equal among the counties then the relative frequency distribution of the adjusted number of screenees with high blood pressure should also be uniform among the counties. The data produced a chi square value of 32.91 with two degrees of freedom. The probability associated with this value is less than .05 which suggests that the prevalence of high blood pressure differs by race in these counties.

Mortality Data

Shown in Table 11 are the number of deaths by the International Statistical Classification for each race and sex group in all counties

TABLE 9

COMPARISON OF THE DISTRIBUTION OF ADJUSTED HIGH BLOOD PRESSURE PREVALENCE IN THE COUNTIES STUDIED BY USE OF THE CHI SQUARE TEST

County	Adjusted No. With High Blood Pressure (1)	Adjusted No. Normal (2)	No. Screened for High Blood Pressure
ROGER	614	1781	2395
CHEROKEE	274	1039	1312
DELAWARE	265	1246	1511
BRYAN	458	1897	2355
McCLAIN	389	1228	1617
HUGHES	390	1362	1752
SEMINOLE	542	2033	2575
JACKSON	435	1640	2076
TOTAL	3,367	12,226	15,593

(1) Adjusted No. with High Blood Pressure = Total Screened for High Blood Pressure X Age Adjusted Rate.

(2) Adjusted Normal = Total Screened for High Blood Pressure - Adjusted No. with High Blood Pressure - χ^2 = 34-37 reject H_o at .05.

TABLE 10

COMPARISON OF THE DISTRIBUTION OF ADJUSTED HIGH BLOOD PRESSURE PREVALENCE BY RACE IN EIGHT COUNTIES STUDIED BY USE OF CHI SQUARE TEST

RACE	Adjusted No. With High Blood Pressure (1)	Adjusted No. Normal (2)	No. Screened for High Blood Pressure
WHITE	2,920	11,583	14,503
NEGRO	168	252	420
INDIAN	163	507	670
TOTAL	3,251	12,342	15,593

(1) Adjusted no. with high blood pressure = Total screened X age-race adjusted rate.

(2) Adjusted normal = Total screened for high blood pressure - adjusted no. $\chi^2 = 32.91$ reject H_o at .05.

TABLE 11

NUMBER OF CASES BY INTERNATIONAL STATISTICAL CLASSIFICATION
FOR EACH RACE AND SEX GROUP FOR ALL 77 COUNTIES 1958-62

CAUSE ISC No.	WHITE Male	WHITE Female	NEGRO Male	NEGRO Female	INDIAN Male	INDIAN Female	TOTAL
Cerebrovascular Disease (330-334)	6,658	6,479	564	620	149	118	14,588
Arteriosclerotic Heart Disease (420-422)	19,114	10,532	859	652	294	161	31,612
Hypertensive Disease (440-447)	1,573	1,874	275	336	47	35	4,140
Nephritis and Nephrosis (590-594)	498	421	117	108	17	26	1,217
TOTAL	27,843	19,306	1,815	1,716	507	340	51,557

of the state during the period under study. All data presented in Tables 12 through 23 are based on these deaths. Because of the small number of deaths in the Negro and Indian populations, these deaths are combined in the subsequent tables and form the basis of the non-white rates presented in these tables. The total number of deaths, (51,557) represent all deaths from hypertensive disease (ISC 440-447) and related cardiovascular-renal diseases (ISC 330-334, 420-422, and 590-594) occurring during the period studied. The highest number of deaths (31,612) occurred from arteriosclerotic heart disease, while the lowest number of deaths (4,140 and 1,217) occurred from hypertensive disease, and nephritis and nephrosis respectively.

Table 12 shows the average annual age-race-sex-specific death rates for hypertensive disease (ISC 440-447) and related cardiovascular-renal disease (ISC 330-334, 420-422, and 590-594) for the white population in the state for the period under study. The age-race-sex-specific rates for hypertensive disease are similar for both sex groups, however, the 75+ age group shows a slightly higher rate for males. The age-sex-race specific death rates for cerebrovascular disease (ISC 330-334) are similar for both sex groups up to age 65. The rates in the 65-74 and 75+ age groups are higher for females showing rates of 166.20 and 200.00 as compared to 86.50 and 97.50 for males. The rates for arteriosclerotic and degenerative heart disease (ISC 420-422) remain consistently higher in all age groups for males than for females, with dramatic increase in the older age groups. Nephritis and nephrosis age-race-sex-death rates are similar for all age groups for both males and females. A marked increase with age is observed with this disease for whites as compared

TABLE 12

*AVERAGE ANNUAL AGE-RACE-SEX SPECIFIC DEATH RATES FOR HYPERTENSIVE DISEASE AND RELATED CARDIOVASCULAR-RENAL DISEASE FOR WHITE MALES AND FEMALES IN OKLAHOMA, 1958-1962

Cause ISC No.	White Males						
	<20	20-34	35-44	45-54	55-64	65-74	75+
Hypertensive Disease (440-447)	0.0	0.28	0.82	4.30	15.08	32.46	90.2
Cerebrovascular Disease (330-334)	0.46	1.36	4.72	8.70	45.0	86.50	97.50
Arteriosclerotic and Degenerative Heart Disease (420-422)	0.5	2.72	12.8	36.7	110.6	395.6	597.60
Nephritis and Nephrosis (590-594)	0.38	1.54	1.58	3.26	5.26	12.06	50.74
Cause ISC No.	White Females						
	<20	20-34	35-44	45-54	55-64	65-74	75+
Hypertensive Disease (440-447)	0.02	0.18	1.72	4.52	11.10	27.6	74.6
Cerebrovascular Disease (330-334)	0.04	1.60	5.00	11.28	31.84	166.20	200.00
Arteriosclerotic and Degenerative Heart Disease (420-422)	0.80	1.02	5.20	10.20	40.17	157.0	225.15
Nephritis and Nephrosis (590-594)	0.34	0.80	1.10	2.30	4.16	9.52	49.6

*Deaths per 100,000 population.

with the other diseases considered in this table.

Shown in Table 13 are the age-race-sex specific death rates for hypertensive disease (ISC 440-447) and related cardiovascular-renal diseases (ISC 330-334, 420-422, and 590-594) for non-whites. The rates for hypertensive disease (ISC 440-447) are higher for the non-white females than for the non-white males with the greatest difference shown in the 65-74 and 75+ age groups. The rate for cerebrovascular disease (ISC 330-334) are higher for the non-white females in the older age groups when compared to rates of males in the same age groups. In the younger age groups, the rates are about the same, with marked differences occurring in the 45-54 age group, showing rates of 15.9 and 23.0 respectively. The arteriosclerotic and degenerative heart disease (ISC 420-422) rates are higher for non-white males in all age groups, with the most pronounced increases in the older age groups. The non-white males show a ten-fold increase in rate from 45 to 75 age groups. The same is true for females, only more pronounced. The non-white female experienced slightly higher nephritis and nephrosis (ISC 590-594) rates than the non-white males. With this disease as with the others there is a definite increase with age, the higher rates shown in the older age groups, with the same shocking increase shown by the other disorders presented in this table. The rates for nephritis and nephrosis are the lowest in both sex groups and for all age intervals considered of the diseases described in this table.

Table 14 shows the age-adjusted death rates for hypertensive disease (ISC 440-447) in the ten most populated counties in Oklahoma by race. The non-white/white ratios are also presented. The white rate in

TABLE 13

*AVERAGE ANNUAL AGE-RACE-SEX SPECIFIC DEATH RATES FOR HYPERTENSIVE DISEASE AND RELATED CARDIOVASCULAR-RENAL DISEASES FOR NON-WHITE MALES AND FEMALES IN OKLAHOMA, 1958-1962

Cause ISC No.	Non-white Males						
	<20	20-34	35-44	45-54	55-64	65-74	75+
Hypertensive Disease (440-447)	0.0	0.36	3.06	14.1	40.4	75.4	162.4
Cerebrovascular Disease (330-334)	0.7	3.6	8.2	15.9	70.6	144.0	347.7
Arteriosclerotic and Degenerative Heart Disease (420-422)	0.2	4.97	35.2	141.08	424.08	807.6	1,485.5
Nephritis and Nephrosis (590-594)	0.8	1.4	2.7	6.3	10.1	32.6	146.3
Cause ISC No.	Non-white Females						
	<20	20-34	35-44	45-54	55-64	65-74	75+
Hypertensive Disease (440-447)	0.0	1.1	4.5	18.6	46.8	127.6	257.70
Cerebrovascular Disease (330-334)	0.6	2.1	5.5	23.0	48.6	230.00	356.4
Arteriosclerotic and Degenerative Heart Disease (420-422)	0.3	2.15	7.2	23.0	100.00	438.5	1,365.2
Nephritis and Nephrosis (590-594)	1.1	2.8	3.1	8.1	11.5	18.4	86.70

*Deaths per 100,000 population.

TABLE 14

AVERAGE ANNUAL AGE-ADJUSTED DEATH RATES PER 100,000 POPULATION FOR HYPERTENSIVE DISEASE (ISC 440-447) IN THE TEN MOST POPULATED COUNTIES BY RACE

Counties	Whites	Non-Whites	Non-White/White Ratio
Washington	52.40	81.70	1.55
Tulsa	23.45	83.75	3.57
Okmulgee	24.74	93.07	3.76
Muskogee	34.86	73.81	2.11
Garvin	39.48	141.76	3.56
Oklahoma	28.52	96.26	3.37
Stephens	40.10	118.33	2.95
Grady	32.32	96.07	2.97
Payne	26.73	69.75	2.60
Kay	36.50	37.96	1.04

these counties vary from county to county with Washington county showing the highest rate (52.40 per 100,000) and Tulsa showing the lowest rate (23.45 per 100,000). The rates for non-whites also vary within these counties remaining higher than the white rate in all counties, with Garvin experiencing the highest rate, (141.76) and Kay the lowest (37.96 per 100,000). The greatest white/non-white difference is seen in Okmulgee county with a ratio 3.76, while Kay county shows the lowest ratio of 1.04 for the ten counties considered. Eight of the counties show at least a two-fold difference with Washington and Kay counties showing differences less than two-fold, 1.55 and 1.04 respectively.

Average annual age-adjusted death rates for the major related cardiovascular-renal diseases (ISC 330-334, 420-422, 590-594) for the ten most populated counties are presented in Table 15 by race. Arteriosclerotic heart disease (ISC 420-422) shows the highest rates in all ten counties, and in both racial groups as compared to the other two disorders. The non-white rates are higher than the white in eight of the counties, with Oklahoma and Stephens counties showing slightly higher white rates for arteriosclerotic heart disease. The non-white rates, although higher than the white rates in all but two counties, shows no striking difference in those counties where it is higher. The highest non-white rate (352.72 per 100,000) is seen in Kay county and lowest (222.24 per 100,000) in Grady county, while the highest white rate (266.54 per 100,000) and the lowest (185.41 per 100,000) are shown for Washington and Grady counties respectively.

The rates for cerebrovascular disease (ISC 330-334) for both racial groups, and in all ten counties are lower than those presented

TABLE 15

*AVERAGE ANNUAL AGE-SEX-ADJUSTED DEATH RATES FOR THE MAJOR CARDIOVASCULAR-RENAL DISEASE (ISC 330-334, 420-422, 590-594) IN THE TEN MOST POPULATED COUNTIES BY RACE

	Cerebrovascular Disease (ISC 330-334)		Arteriosclerotic Heart Disease (ISC 420-422)		Nephritis and Nephrosis (ISC 590-594)	
COUNTIES	White	Non-White	White	Non-White	White	Non-White
Washington	59.30	95.95	266.54	302.16	9.59	10.00
Tulsa	130.81	120.52	252.13	306.74	9.14	40.42
Okmulgee	111.68	162.18	238.80	308.47	6.22	34.40
Muskogee	111.53	164.71	216.46	321.59	11.22	25.86
Garvin	123.61	137.65	231.33	304.04	6.74	29.45
Oklahoma	132.63	127.75	255.13	251.64	7.18	42.06
Stephens	69.89	102.90	277.28	261.32	2.70	15.83
Grady	125.64	214.05	185.41	222.24	6.56	10.22
Payne	117.82	127.65	189.41	253.41	11.17	8.80
Kay	127.21	124.85	345.86	352.72	7.83	59.78

*Rates per 100,000 population.

for arteriosclerotic heart disease (ISC 420-422). The rate for whites are lower than those for non-whites in seven of the counties shown, with Oklahoma, Tulsa, and Kay counties showing slightly higher rates, 132.63, 130.81, and 127.21 per 100,000 respectively. The highest non-white rate (214.05 per 100,000) is experienced by Grady county, and lowest rate, (95.95 per 100,000) is shown for Washington county.

Nephritis and nephrosis (ISC 590-594) shows the lowest rates of the three diseases presented in this table. The rates for both whites and non-whites are lower, and the county to county rates are also lower. The non-white rate is higher in nine of the counties considered, with Kay county showing the highest rate, (59.78 per 100,000) and Payne showing the lowest rate, (8.80 per 100,000). The non-white/white difference in six of the counties is more than four-fold, which is greater than the differences observed for the other two diseases considered in this table. The white rate is highest in Muskogee county and lowest in Stephens county. Payne county shows a higher white rate than that for non-whites, while Washington county shows only a small difference between the two racial groups.

Table 16 shows the average annual age-adjusted death rates and mortality ratios for hypertensive disease (ISC 440-447) in the ten least populated counties by race. The non-white rate is higher than the white rate for all ten counties, with Marshall county showing the highest rate (133.13 per 100,000), and Latimer county showing the lowest (4.02 per 100,000). The non-white/white ratios range from 5.62 to 1.02, with the greater ratios seen for Dewey, Love, Pushmataha, and Marshall counties, while the remainder of the counties show differences less than two-fold.

TABLE 16

*AVERAGE ANNUAL AGE-ADJUSTED DEATH RATES AND MORTALITY RATIOS FOR HYPERTENSIVE DISEASE (ISC 440-447) IN THE TEN LEAST POPULATED COUNTIES BY RACE

Counties	White	Non-White	Non-White/White Ratio
Coal	66.70	72.15	1.08
Cotton	20.19	38.66	1.91
Dewey	48.00	112.21	2.33
Greer	11.69	11.96	1.02
Latimer	3.75	4.02	1.07
Love	15.79	38.62	2.44
Roger Mills	48.20	51.40	1.06
Pushmataha	21.82	46.67	2.13
Marshall	23.66	133.13	5.62
Haskell	34.15	46.71	1.36

*Rates per 100,000 population.

The range is smaller between the white rates than it is for non-whites, showing approximately a two-fold difference between the rates of the two groups.

The average annual age-adjusted death rates in the ten least populated counties, for the related major cardiovascular-renal diseases (ISC 330-334, 420-422, 590-594) are presented in Table 17. Arteriosclerotic heart disease (ISC 420-422) shows higher rates than the other two disorders presented, with the whites showing higher rates than non-whites in Latimer, Roger Mills, Marshall, and Pushmataha counties. Non-whites show higher rates in all other counties, with the range in rates being higher in this group. The next highest rates are seen for cerebrovascular diseases (ISC 330-334) with the non-white experiencing higher rates in all counties except Haskell, and showing greater variability in rates than the white. Dewey county shows the highest rates of all counties for whites and non-whites with rates of (571.63 and 927.88 per 100,000) respectively. The lowest rate for whites is seen in Greer county and for non-whites, Haskell county. Nephritis and nephrosis (ISC 590-594) again shows the lowest rates for both racial groups in all counties considered, with the greatest range in rates seen in the non-white rates. The non-white/white difference observed between these rates appear to be greater for this particular disease than for the other two diseases presented. However, in Haskell county the non-white rate, (104. per 100,000) is roughly nine times higher than the (11.11 per 100,000) rate for whites. The non-white rate is also the highest rate shown for either race-sex-group presented for nephritis and nephrosis. The non-white/white difference appears to be five-fold or

TABLE 17

*AVERAGE ANNUAL AGE-ADJUSTED DEATH RATES FOR THE MAJOR CARDIOVASCULAR-RENAL DISEASES (ISC 330-334, 420-422, 590-594) IN THE TEN LEAST POPULATED COUNTIES BY RACE

COUNTIES	Cerebrovascular Disease (ISC 330-334)		Arteriosclerotic Heart Disease (ISC 420-422)		Nephritis and Nephrosis (ISC 590-594)	
	White	Non-White	White	Non-White	White	Non-White
Coal	109.89	147.14	271.21	270.31	5.10	48.83
Cotton	102.41	342.27	258.93	333.21	15.57	44.46
Dewey	571.63	927.88	224.67	224.43	17.22	20.00
Greer	95.40	291.10	244.53	362.81	11.80	15.00
Latimer	121.66	152.06	230.18	72.16	10.26	24.58
Love	139.55	323.20	208.32	232.41	9.39	17.24
Roger Mills	312.35	336.65	330.84	224.34	2.90	4.42
Pushmataha	105.78	133.89	209.63	149.64	9.42	10.40
Marshall	166.72	168.99	286.58	269.76	1.84	18.21
Haskell	124.71	110.55	188.95	290.79	11.11	104.34

*Rates per 100,000 population.

greater in the following counties for nephrosis and nephritis: Coal, Haskell, and Marshall counties. The greatest non-white/white difference (9.8) is seen for Marshall county, with the remainder of the counties showing differences considerably less than this.

In order to demonstrate the geographical distribution of mortality from hypertensive disease (ISC 440-447) in Oklahoma, the average annual age-adjusted death rates for the period under study were depicted by quartiles in Figures 1 - 4 for white males, white females, non-white males, and non-white females. Geographical clustering of hypertensive disease for the white males and females appears not to be appreciable for the period studied as shown in Figures 1 and 2 respectively, however, the western border of the state seems to show clustering in both instances. With the exception of the cluster area just described in Figures 1 and 2 there appears to be no definite geographical pattern established in either case. The lack of a pattern can be further observed in Figure 1, when one observes the western part of the state where the clustering of counties appear. It can be observed that all counties of this area do not appear in the cluster, with the three counties of the Panhandle area appearing in the third and lower quartiles respectively. Along the southern border of the state as seen in Figure 1, only two counties appear in the upper quartile while the other counties are divided among the other three quartile with the majority of counties in this area appearing in the lower quartile. The majority of counties on the eastern border of the state are in the upper quartile counties, but no definite geographic patterns are established (Figure 1). The geographical distribution of hypertensive disease death (ISC 440-447) for

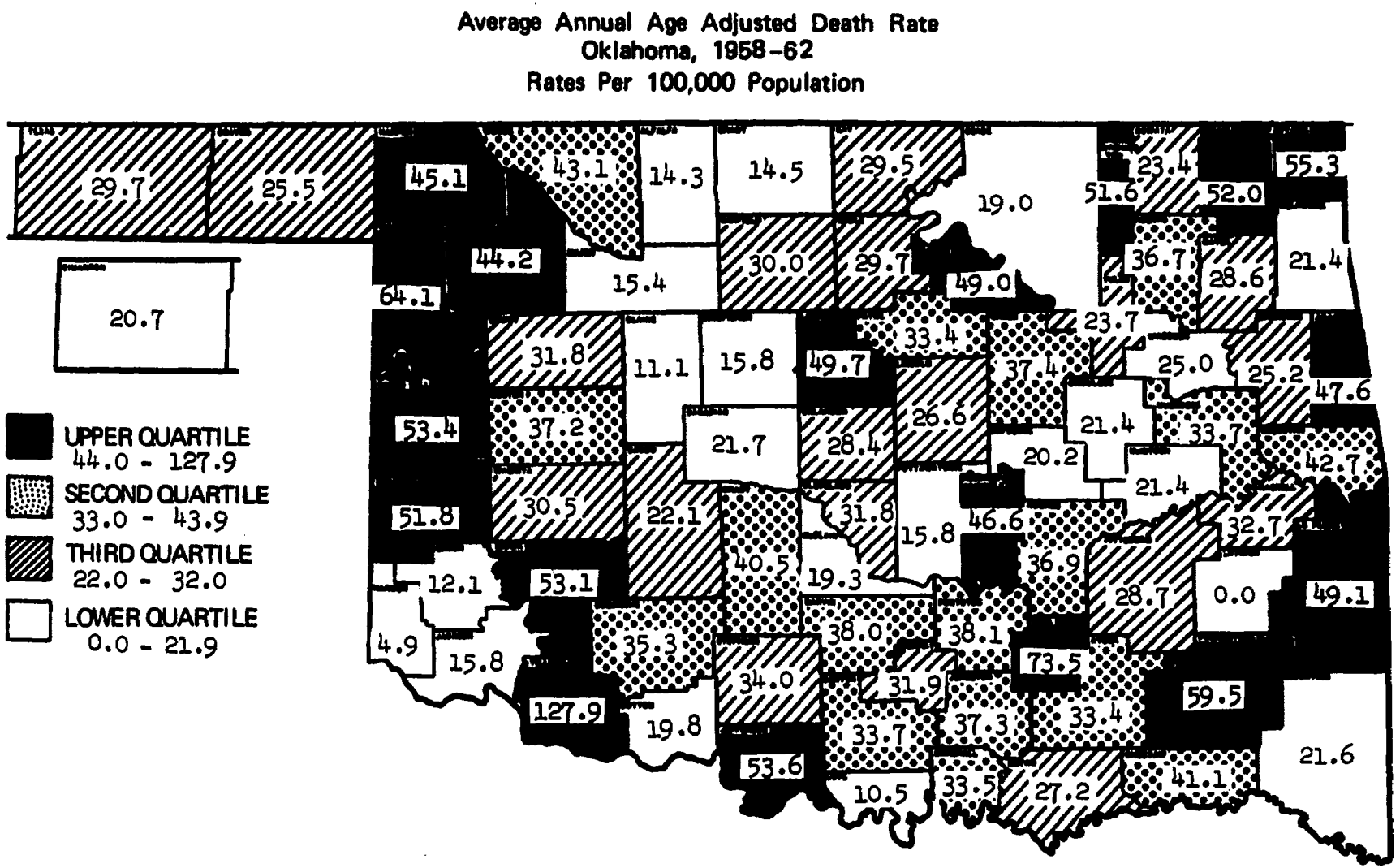

Figure 1. Hypertension and Hypertensive Heart Disease (ISC 440-447), White Males

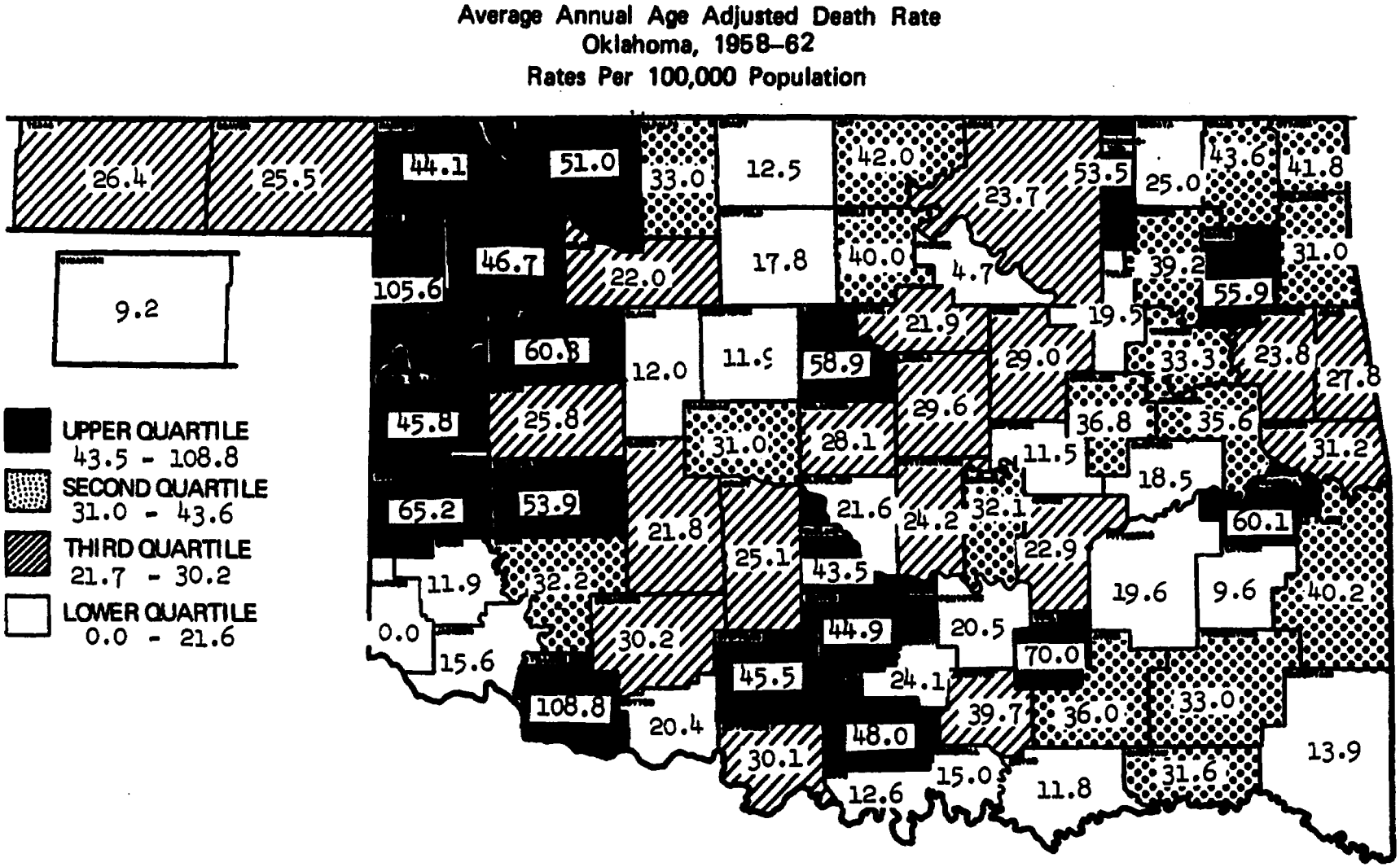

Figure 2. Hypertension and Hypertensive Heart Disease (ISC 440-447), White Females

white males (Figure 1) in the central part of the state where one of the largest urban areas is located, show average annual age-adjusted death rates which appears to be randomly distributed between the four quartiles. The two most populated counties, Oklahoma county and Tulsa county, though located in different areas of the state, both appear in the third quartile (Figure 1). Figure 2 which shows the geographic distribution of hypertensive disease (ISC 440-447) for white females shows an identical geographical pattern in the Panhandle area as was described in Figure 1. The same pattern is true for the southern border of the state, however, in the case of white females (Figure 2) more counties in this area appear in the lower quartile with only one county in the upper quartile in this region. The central area counties are distributed in the same manner as described in Figure 1, with the counties in the Oklahoma county area appearing in all four quartiles. Oklahoma county appears in the third quartile, the same quartile it appeared in Figure 1. Most of the counties on the eastern border (Figure 2) appear in the second quartile with only one county appearing in the lower quartile and none in the upper quartile. Tulsa county, the second most populated area in the state is found in the lower quartile for white females (Figure 2) as compared to the third quartile for white males in Figure 1.

Figures 3 and 4 show the geographical distribution of the average annual age-adjusted death rates in the state for non-white males and non-white females respectively for the period under study. The geographical distribution of mortality for non-whites does not follow the same pattern as that of the white. The most populated counties in the state rank in the upper quartile for both sex groups (Figures 3 and 4). Tulsa

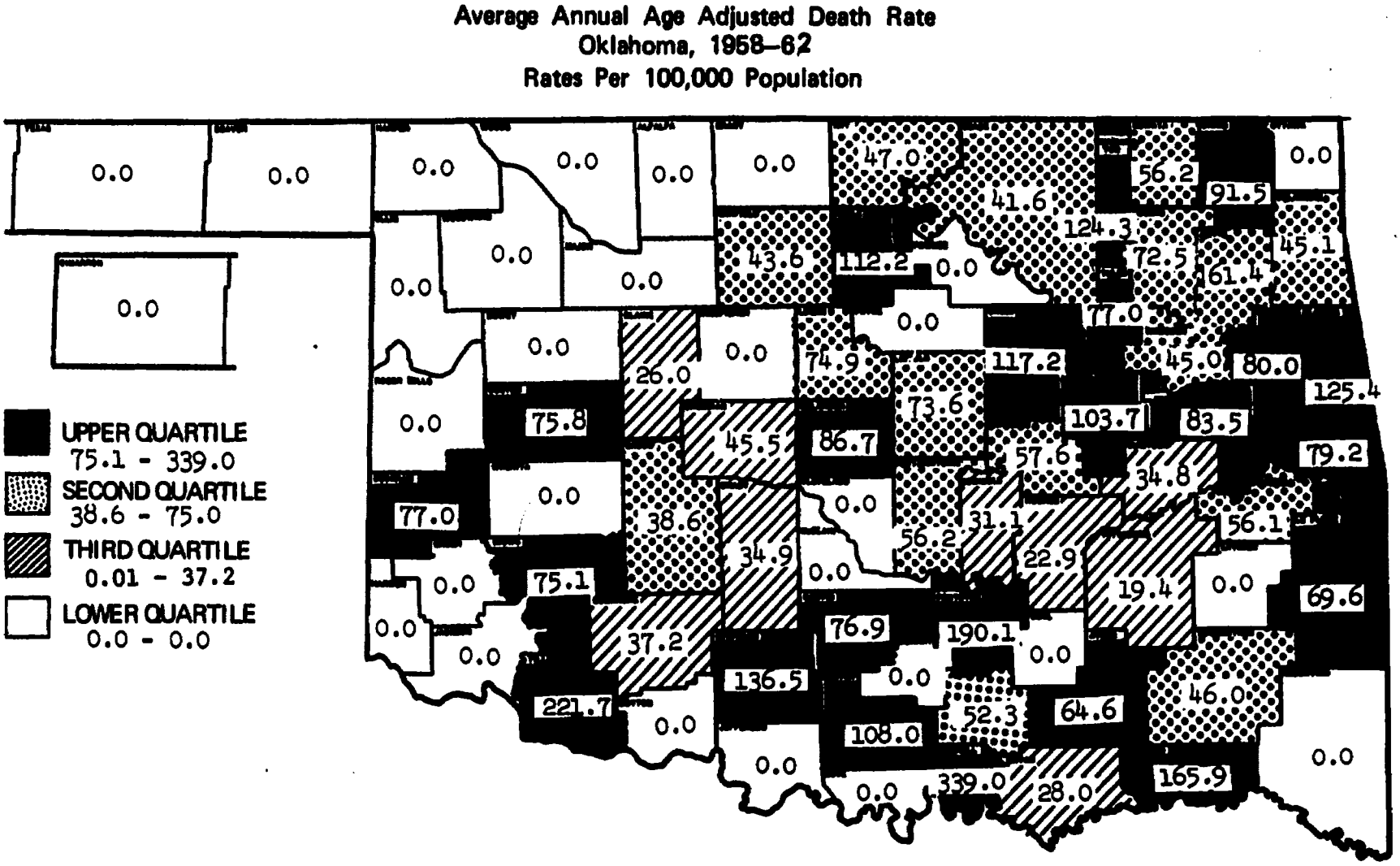

Figure 3. Hypertension and Hypertensive Heart Disease (ISC 440-447) Non-White Males

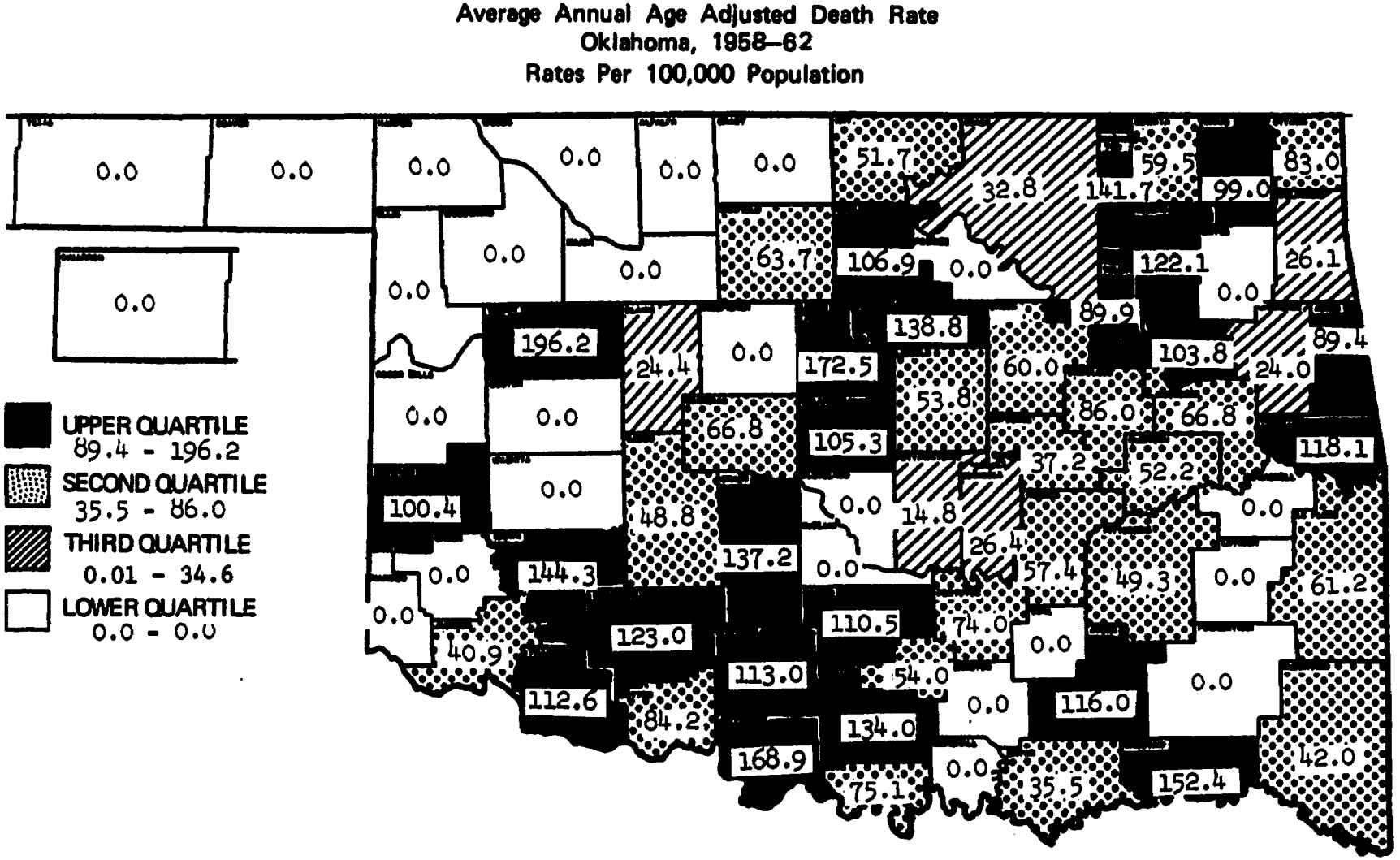

Figure 4. Hypertension and Hypertensive Heart Disease (ISC 440-447) Non-White Females

area counties appear in the second quartile (Figure 3) and the upper quartile (Figure 4) as compared to the third quartile in Figures 1 and 2. The only cluster area (Figure 3) appears on the eastern border of the state, while most of the counties on the southern border tend to fall in the lower quartile. The southern border counties not falling in the lower quartile were; Choctaw, Marshall and Tillman counties, these counties appeared in the upper quartile in Figure 3. Most of the counties in the central portion of the state are in the upper and second quartiles. One fourth of the counties in the state appear in the lower quartile in both Figrues 3 and 4. The non-white female (Figure 4) distribution is very similar to that for non-white males (Figure 3) with the following exceptions: The one cluster area appears in the south central region instead of the eastern border (Figure 3); all counties on the southern border are located in the upper and second quartiles. The majority of counties in the central portion of the state (Figure 4) are located in the second quartile, with four exceptions, two are located in the lower quartile and two in the third quartile.

The median age-adjusted death rates for hypertensive disease (ISC 440-447) by socio-economic area, race, and sex for the state during the period under study is presented in Table 18. The rates tend to increase with a decrease in socio-economic area in both race-sex groups. The lowest rates in all socio-economic areas were experienced by the white female, with rates ranging from 26.4 in the high socio-economic area to 33.0 per 100,000 for the low socio-economic area. The rates experienced by the white male are higher than those for the white females in three of the areas, with the female rate being slightly higher in the

TABLE 18

*MEDIAN AGE-ADJUSTED DEATH RATE FOR HYPERTENSIVE DISEASE (ISC 440-447) BY SOCIO-ECONOMIC AREA, RACE, AND SEX FOR THE STATE, 1958-1962

Socio-Economic Areas	White Males	White Females
High Socio-economic	29.5	26.4
Above State Average	33.4	30.2
Below State Average	36.9	29.1
Low Socio-Economic	32.8	33.0

Socio-Economic Areas	Non-White Males	Non-White Females
High Socio-economic	0.0	0.0
Above State Average	38.6	59.5
Below State Average	52.3	57.4
Low Socio-Economic	56.1	61.2

*Rates per 100,000 population.

low socio-economic area. The lowest rates for white males 29.5 and females 26.4 appeared in the high socio-economic area, and the highest rate, 36.9 for white males is seen in the below average area, while the highest 33.0 for females appeared in the low socio-economic area. The highest area rates experienced by either race-sex groups are shown for the non-white female in three socio-economic areas, with the highest rate, 61.2 per 100,000 appearing in the low socio-economic area, and the lowest 57.4 per 100,000 in the below state average area. The high socio-economic area shows no rates for non-white males or females. The above state average area show slightly higher rates for females of both racial groups, with the highest rate 59.5 per 100,000 for the non-white female. The non-white male rates are lower than those for non-white females in the three areas, above state average, below state average, and low socio-economic areas. The socio-economic distribution of Oklahoma counties in 1950, is shown in Figure 5 and divides the state into four socio-economic areas. These areas served as the basis of the data presented in Table 18. It can be observed from Figure 5 that more of the counties are located in the above state average area, with the other counties distributed equally between the other socio-economic areas.

The data presented in Table 19 is concerned with the median test to determine if the non-white/white ratios for hypertensive disease (ISC 440-447) for the ten most populated counties and the ten least populated counties are from the same population of ratios or that the median is the same for both sets of ratios. The ratios from the ten most populated counties range from 1.04 to 3.56 as compared to those for the least populated counties which range from 1.04 to 5.62. Observation of these ratios

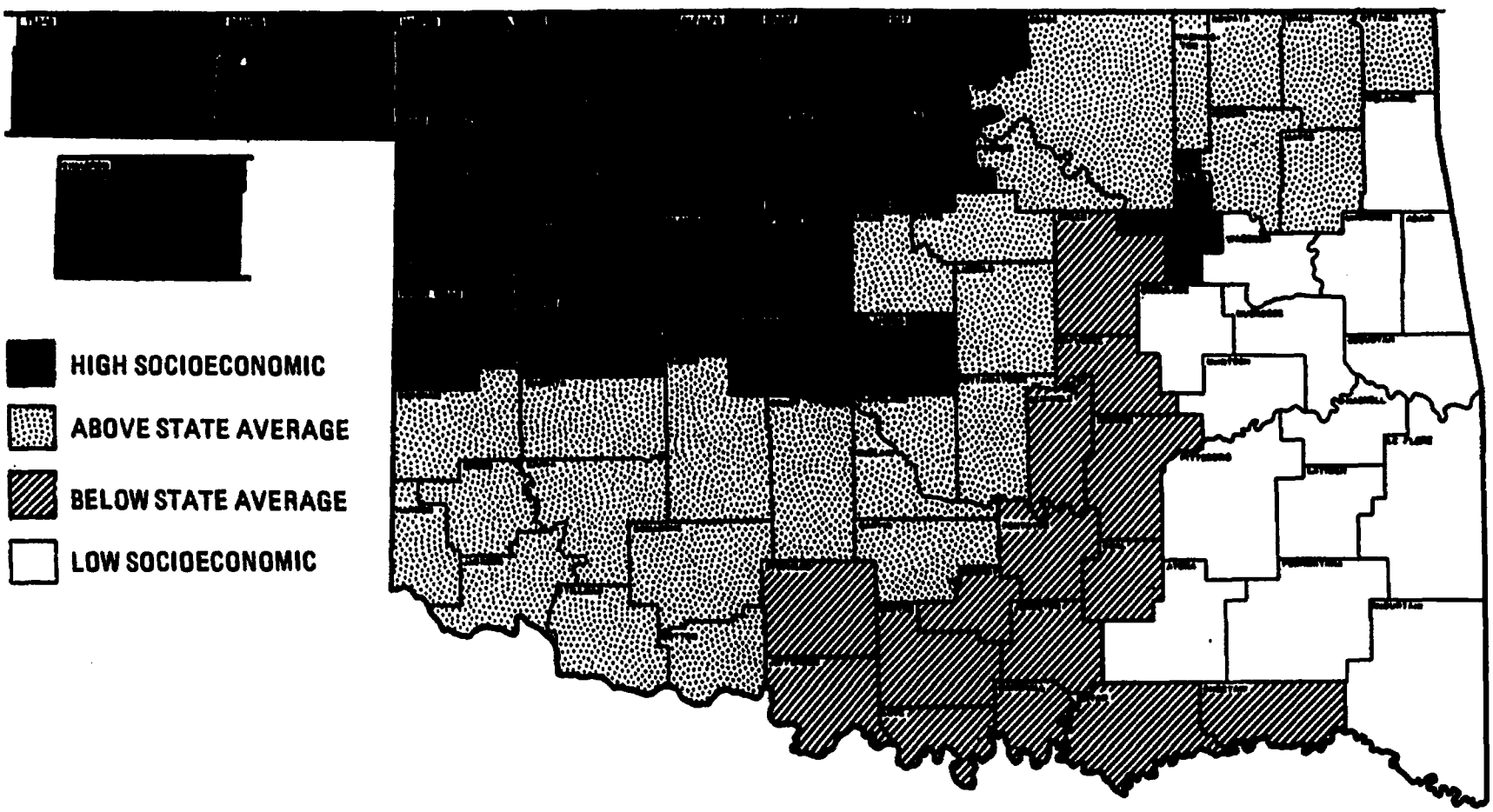

Figure 5. Socioeconomic Distribution of Oklahoma Counties (1950)

TABLE 19

MEDIAN TEST TO DETERMINE IF THE MEDIAN NON-WHITE/WHITE RATIO FOR THE TEN MOST POPULATED AND THE TEN LEAST POPULATED COUNTIES FOR HYPERTENSIVE DISEASE (ISC 440-447) ARE FROM THE SAME POPULATION OF RATIOS

°Most Populated Counties	°Least Populated Counties
1.55	1.08
3.57	1.91
3.76	2.33
2.11	1.02
3.56	1.07
3.37	2.44
2.95	1.06
2.97	2.13
2.60	5.62
1.04	1.36

MEDIAN TEST

	Counties with High NW/W	Counties with Low NW/W	Total
No. of counties above median	7	3	10
No. of counties below median	3	7	10
	10	10	20

°Median = 2.33, χ^2 value 1.80; df = 1, α .05

indicate great similarity between ratios, although there are differences in the range of ratios with the higher range appearing in the least populated counties. The ratios were arranged in descending order from 5.62 to 1.02; the median obtained from this array was 2.33. The counties were divided into the number of counties above the median and the number of counties below the median and placed into high non-white/white ratios of counties and low non-white/white ratio counties. The expected frequency was obtained and a chi square test was performed yielding a chi square value of 1.80 at one degree of freedom with an alpha level of .15, thereby allowing acceptance of the null hypothesis that the two groups of ratios could have come from a population with the same median.

Table 20 presents correlation coefficients for hypertensive disease (ISC 440-447) with associated cardiovascular-renal disease (ISC 330-334, 420-422, and 590-594) for all counties in the state. For each county the age-adjusted death rate for hypertensive disease and that for cerebrovascular disease gives an associated pair of measurements, since they refer to the same county. Thus, for the entire state there were seventy-seven such pairs. A correlation analysis of these pairs was performed to measure the degree of association of these pairs. A correlation coefficient of 0.797 resulted and was found to be significant at .05, thereby suggesting a degree of association between age-adjusted rates. Similarily, hypertensive disease and arteriosclerotic heart disease age-adjusted rates were considered. A correlation analysis of these pairs was performed to measure the degree of association of these pairs. The resulting correlation analysis produced a correlation coefficient 0.690 which was significant at .05, suggesting a degree of association

TABLE 20

CORRELATION COEFFICIENTS FOR HYPERTENSIVE DISEASE (ISC 440-447) WITH ASSOCIATED CARDIOVASCULAR-RENAL DISEASES (ISC 330-334, 420-422, 590-594) FOR ALL COUNTIES

*ISC 3 and ISC 1	*ISC 3 and ISC 2	*ISC 3 and ISC 4
0.797	0.769	0.690

t values 14.48, 12.01 and 11.35 were all greater than the critical t value 2.00 at α .05, reject H_0.

*ISC 3 (ISC 440-447), ISC 1 (ISC 330-334), ISC 2 (ISC 420-422), ISC 4 (ISC 590-594).

between these rates in the state. The age-adjusted death rates from hypertensive disease and nephritis and nephrosis were treated in the same fashion. A correlation coefficient of 0.769 was obtained and found to be significant at .05, thereby suggesting a degree of association between these rates in the state during the period studied.

Table 21 shows the age-adjusted death rates per 100,000 population for the major cardiovascular-renal diseases in several southwestern states and the United States. The rates for Oklahoma remain lower than those of the other states for hypertensive disease (ISC 440-447), cerebrovascular disease (ISC 330-334) and arteriosclerotic heart disease (ISC 420-422), but the age-adjusted rate for nephritis and nephrosis (ISC 590-594) is higher in Oklahoma than the other states listed. Also, shown in Table 21 are the age-adjusted death rates for hypertensive disease (ISC 440-447) and related cardiovascular-renal diseases in the United States, 1960. The Oklahoma rates are higher than the rates for the United States for cerebrovascular disease 125.5, as compared to 108.0, and for nephritis and nephrosis with rates of 2.08 and 0.9 respectively.

Figures 6 through 9 represents the geographical distribution of the average annual age-adjusted rates for cerebrovascular disease (ISC 330-334) for white males, white females, non-white males and non-white females. The geographical distribution of cerebrovascular disease for white males (Figure 6) tends not to follow any definite geographical pattern, with the majority of the upper quartile counties located in the central and northeastern region of the state, including both major population centers, Tulsa and Oklahoma counties where definite cluster areas exists.

TABLE 21

AGE-ADJUSTED DEATH RATES PER 100,000 POPULATION
FOR MAJOR CARDIOVASCULAR-RENAL DISEASES
IN THE SOUTHWEST UNITED STATES, 1960

Cause ISC No.	*Oklahoma	*Arkansas	*Louisiana	*Texas	*United States
Hypertensive Disease (440-447)	35.4	48.1	58.3	47.0	42.1
Cerebrovascular Disease (330-334)	125.5	134.6	199.0	146.0	108.0
Arteriosclerotic Disease (420-422)	279.4	299.7	289.8	290.3	307.4
Nephritis and Nephrosis (590-594)	2.08	1.6	0.8	1.1	0.9

*Vital Statistics of the United States (1960), Vol. II, pp. 1-24. Standard Population - United States, 1960.

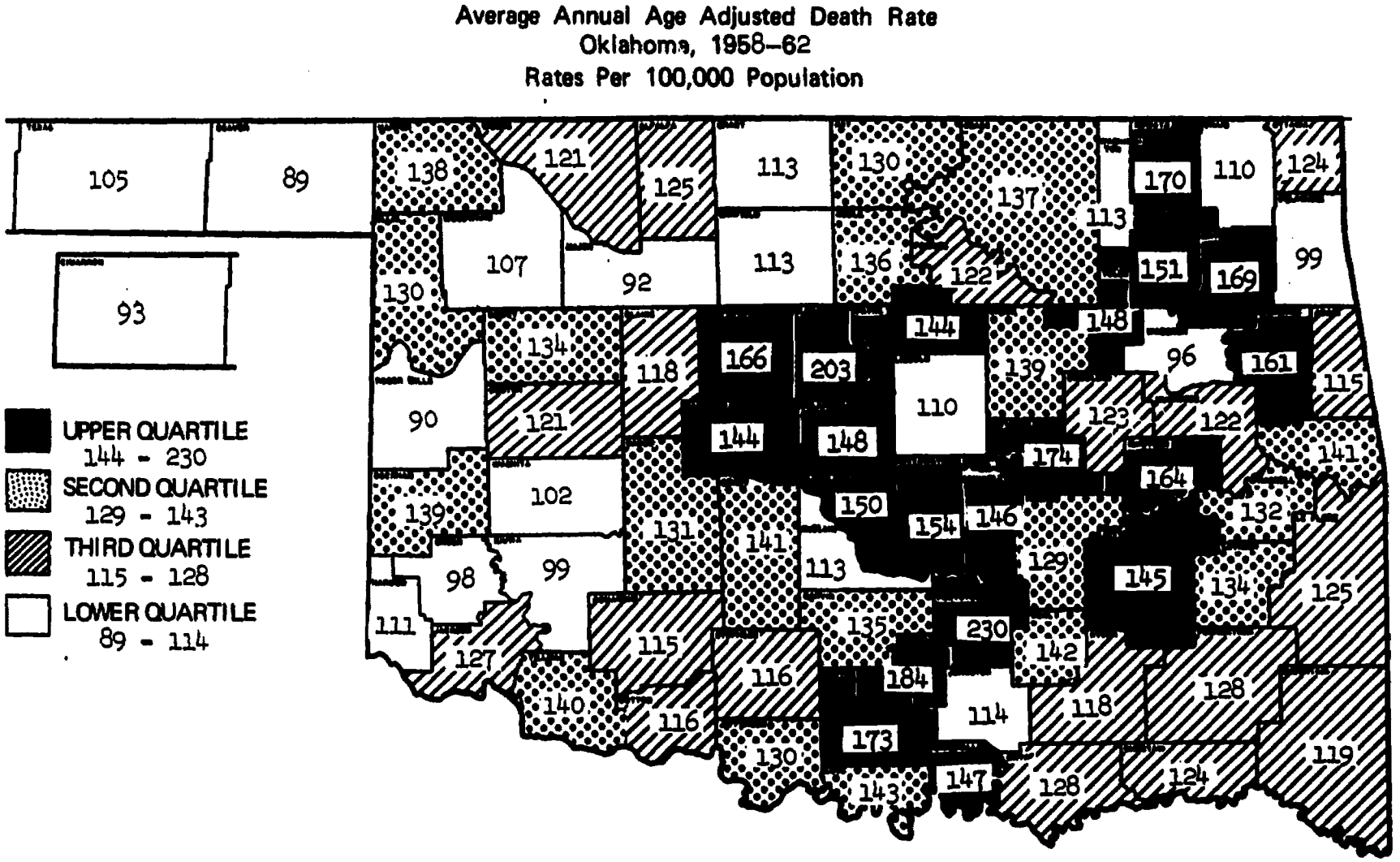

Figure 6. Cerebrovascular Disease (ISC 330-334), White Males

The white female (Figure 7) distribution like that of the white male (Figure 6) follows no definite pattern, however, the same numbers of counties, 19 appear in the upper quartile but their location in the state is quite unlike that for counties in the same quartile in Figure 7. Only one of the populated areas, Oklahoma county appears in a cluster area. Tulsa county falls in the second quartile, with its adjacent counties appearing in all four quartiles. A second cluster area exists on the southern border of the state where five of its eleven counties appear in the upper quartile.

The geographical distribution of the age-adjusted death rates for the non-white males (Figure 8) and non-white females (Figure 9) does not in either case follow that of the white male (Figure 6) or the white female (Figure 7). The distribution in both instances follows no definite pattern, however, three cluster areas occur. These areas appear in the central portion and northeastern border in the case of the non-white male (Figure 8) and for the non-white female (Figure 9) the cluster areas appear on the southeastern border of the state. Pushmataha county which appears in the cluster shows the highest rate, 1,176 per 100,000 population of either race-sex group for either cause of death considered in the entire study.

The geographical distribution of arteriosclerotic and degenerative heart disease (ISC 420-422) is shown in Figures 10 through 13 for white males, white females, non-white males and non-white females, during the period of study. The distribution of arteriosclerotic heart disease for white males (Figure 10) and white females (Figure 11) tend not to follow any definite geographical pattern. In Figures 10 and 11, the

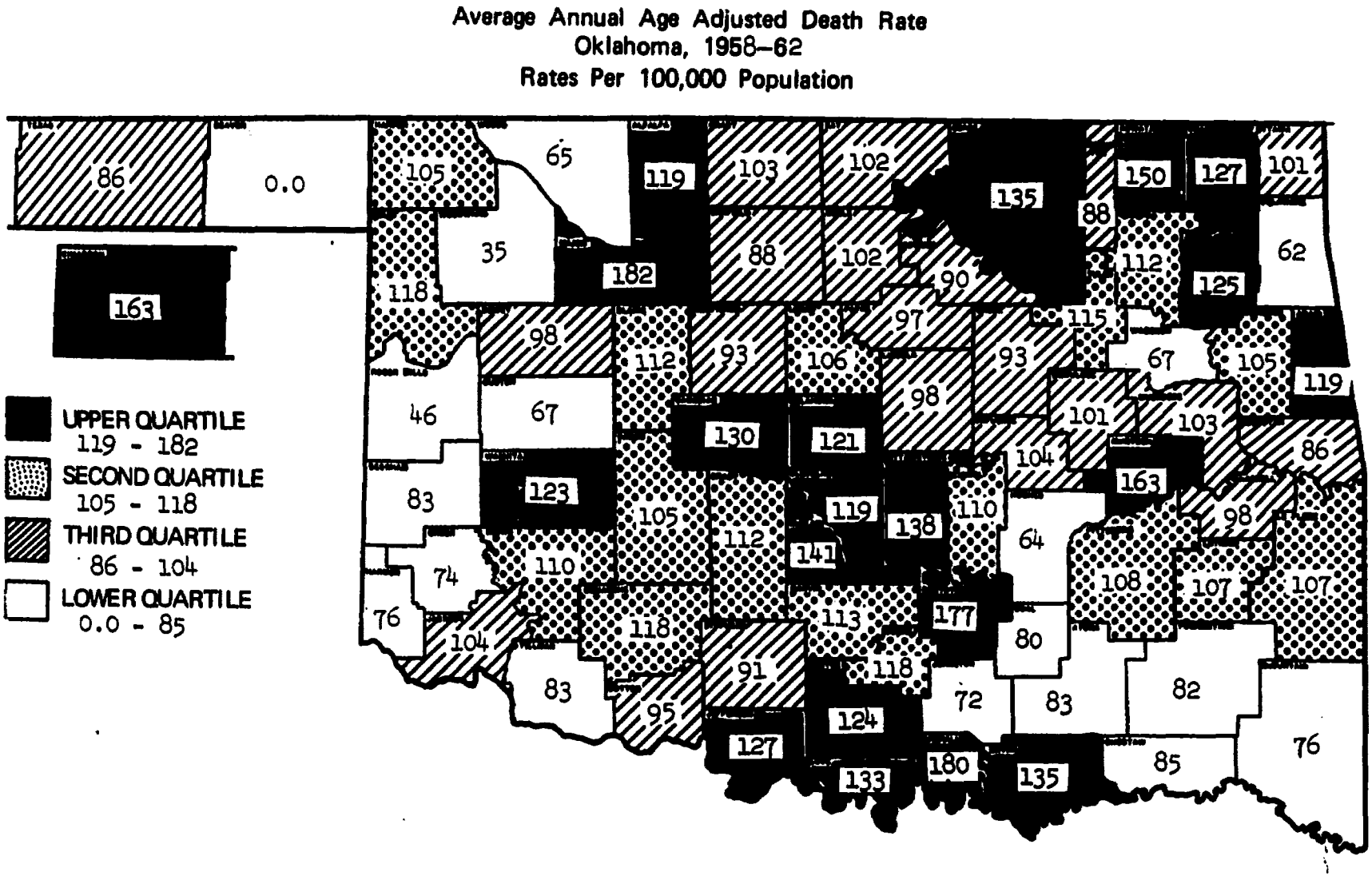

Figure 7. Cerebrovascular Disease (ISC 330-334), White Females

Average Annual Age Adjusted Death Rate
Oklahoma, 1958-62
Rates Per 100,000 Population

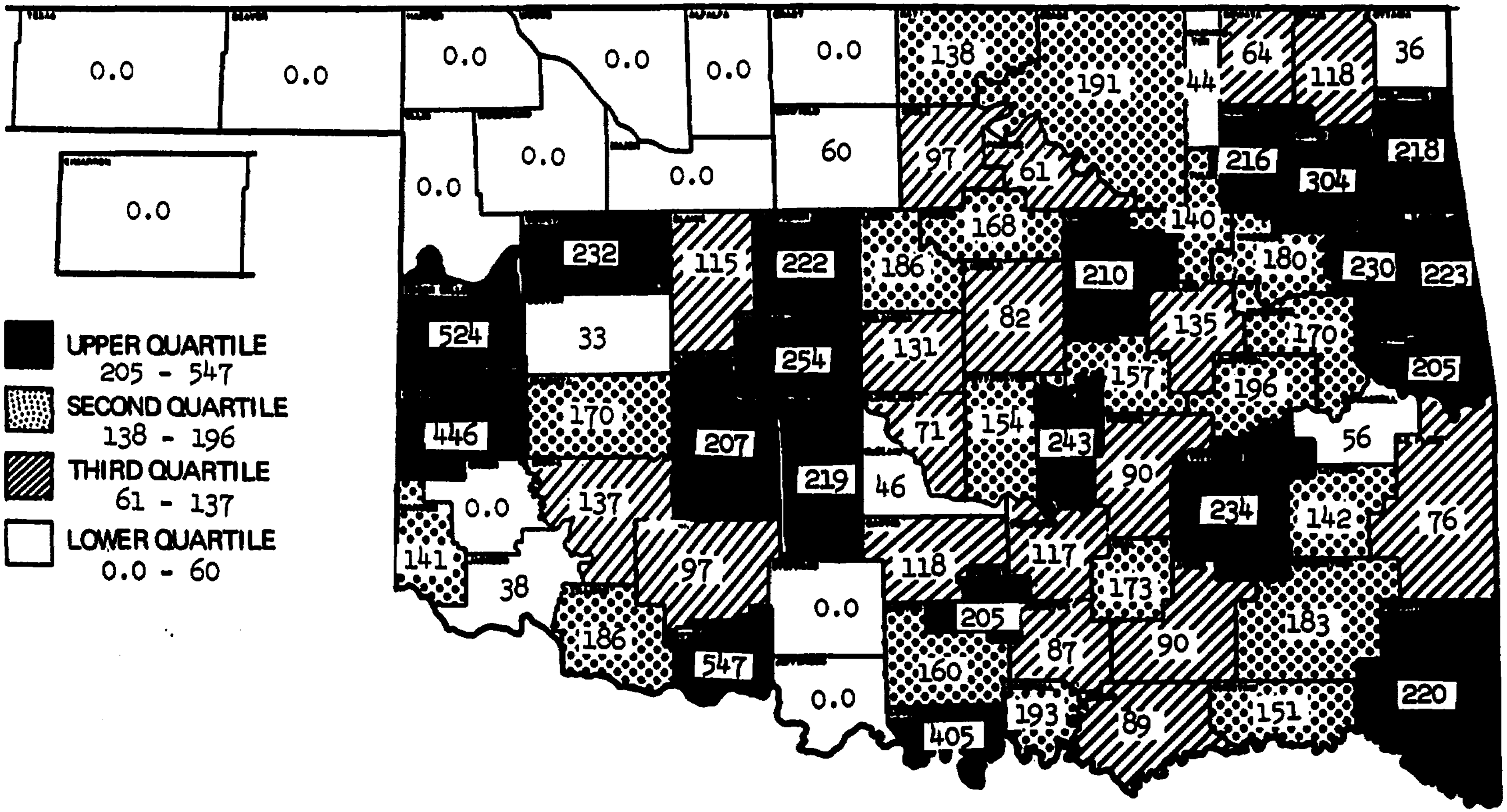

Figure 8. Cerebrovascular Disease (ISC 330-334), Non-White Males

Average Annual Age Adjusted Death Rate
Oklahoma, 1958–62
Rates Per 100,000 Population

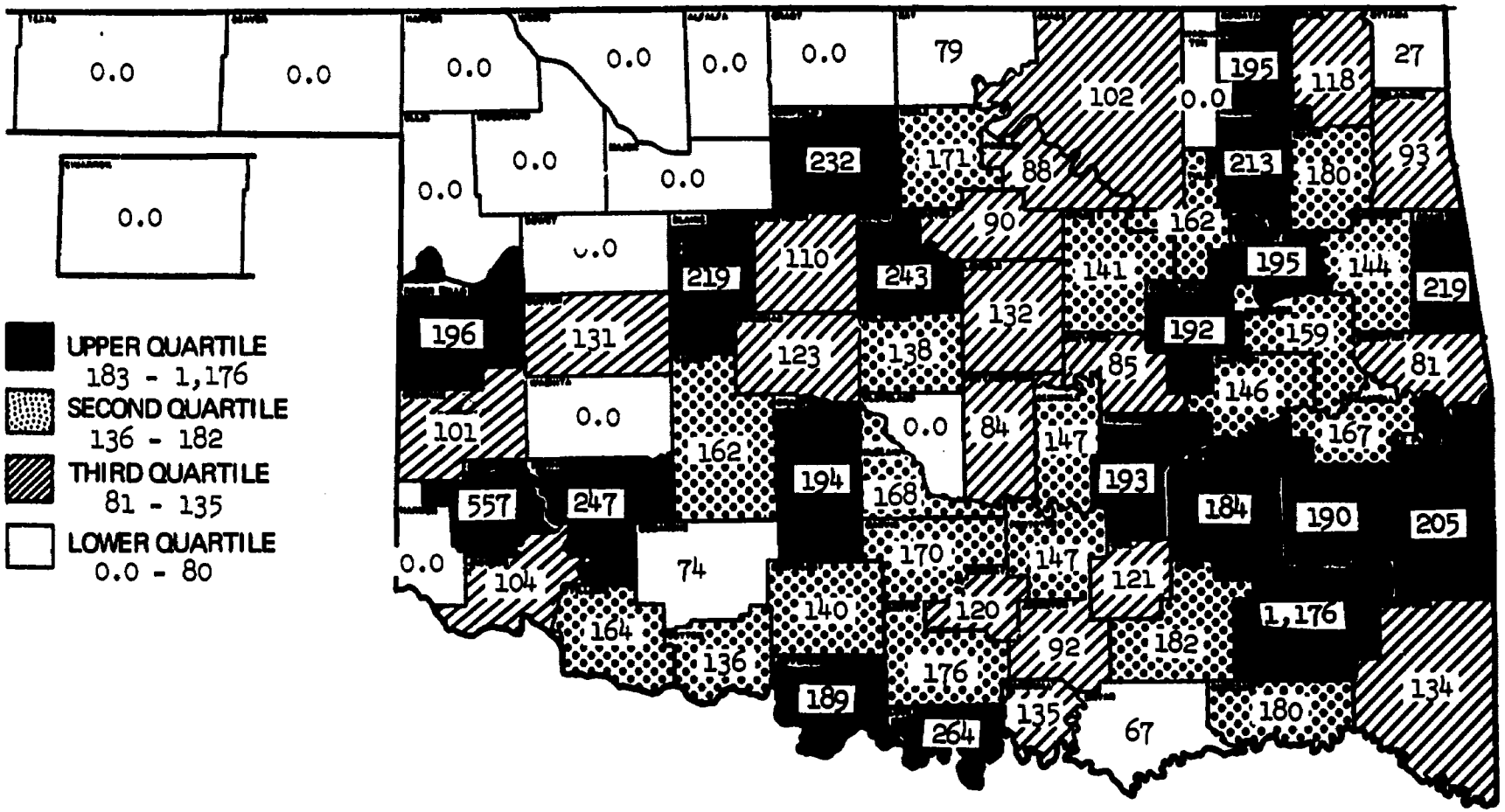

Figure 9. Cerebrovascular Disease (ISC 330-334), Non-White Females

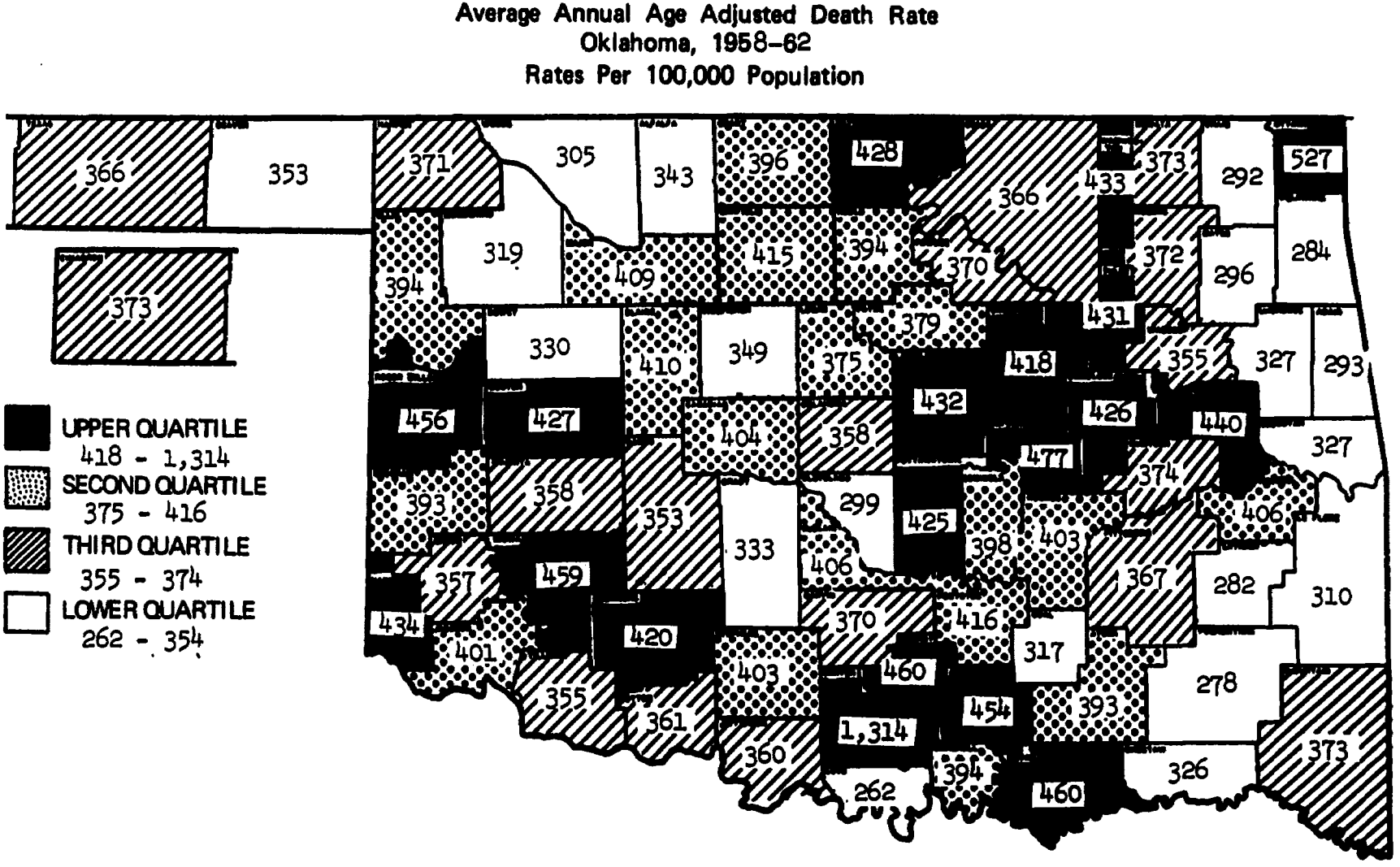

Figure 10. Arteriosclerotic and Degenerative Heart Disease (ISC 420-422), White Males.

Average Annual Age Adjusted Death Rate
Oklahoma, 1958–62
Rates Per 100,000 Population

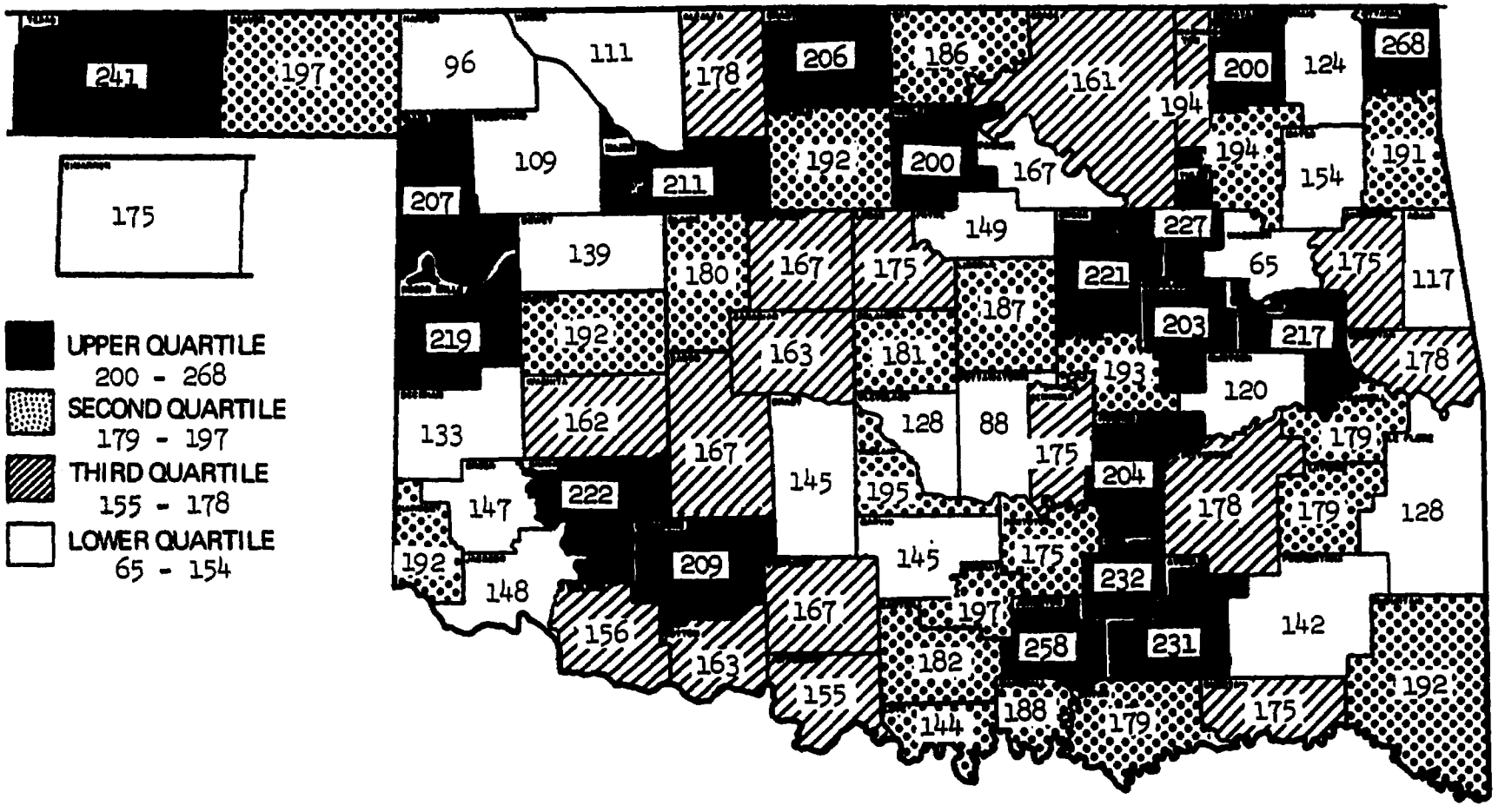

Figure 11. Arteriosclerotic and Degenerative Heart Disease (ISC 420-422), White Females.

cluster areas, in the northeast central part of the state are composed of the same counties; Lincoln, Creek, Tulsa, Okmulgee, Okfuskee, Muskogee, and Washington counties. Although, Oklahoma county is adjacent to the cluster areas it appears in the third quartile in Figure 10 and in the second quartile in Figure 11. The white male distribution (Figure 10) in the Panhandle area shows two counties in the third quartile and one in the low quartile. In the white female (Figure 11) distribution one county appears in the upper quartile, while of the remaining two counties appear in the second quartile and low quartile. All four quartiles are found in the central part of the state in both Figures 10 and 11, however, most of the counties in Figure 10 are in the second and third quartile. The lower quartile counties for white males (Figure 10) appear on the eastern border of the state and include ten of the twelve counties of the area. The lower quartile counties, white females (Figure 11) appear in all of the state and tend not to be located in any one area, with only one appearing on the southern border where the other nine are split between the second and third quartile.

The distribution of arteriosclerotic heart disease in non-white males (Figure 12) and non-white females (Figure 13) like that for white males (Figure 10) and white females (Figure 11) follows no established pattern. The cluster areas found in Figures 10 and 11 do not appear in the same geographical area, for the non-white males (Figure 12) it appears in the northeast central part of the state, and for the non-white females (Figure 13) in the south central portion of the state in Garvin, Pontotoc, Coal, Atoka, Johnston, and Marshall counties. The two most populated counties, Oklahoma and Tulsa (Figure 12) appear in the third and second

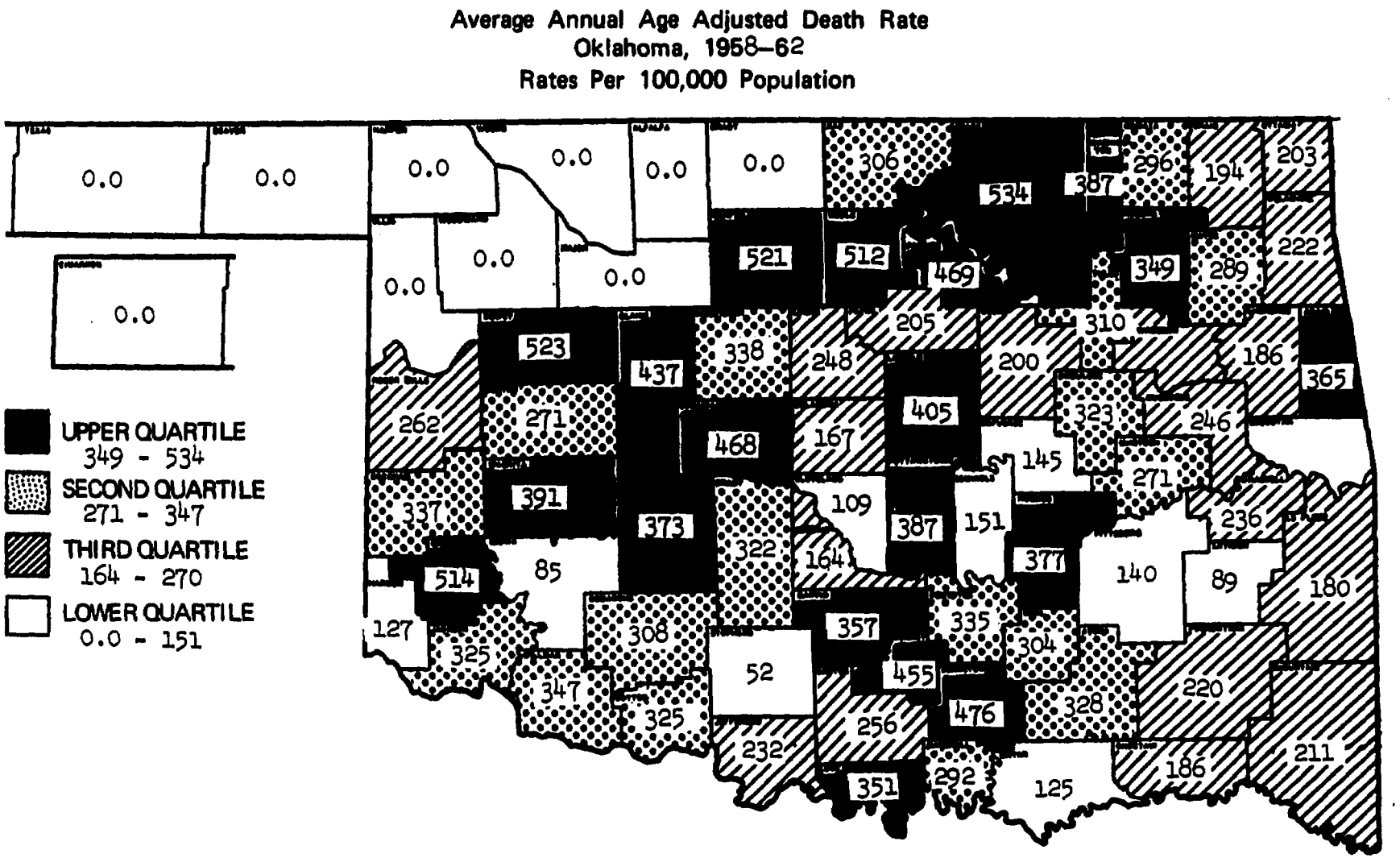

Figure 12. Arteriosclerotic and Degenerative Heart Disease (ISC 420-422), Non-White Males.

Average Annual Age Adjusted Death Rate
Oklahoma, 1958-62
Rates Per 100,000 Population

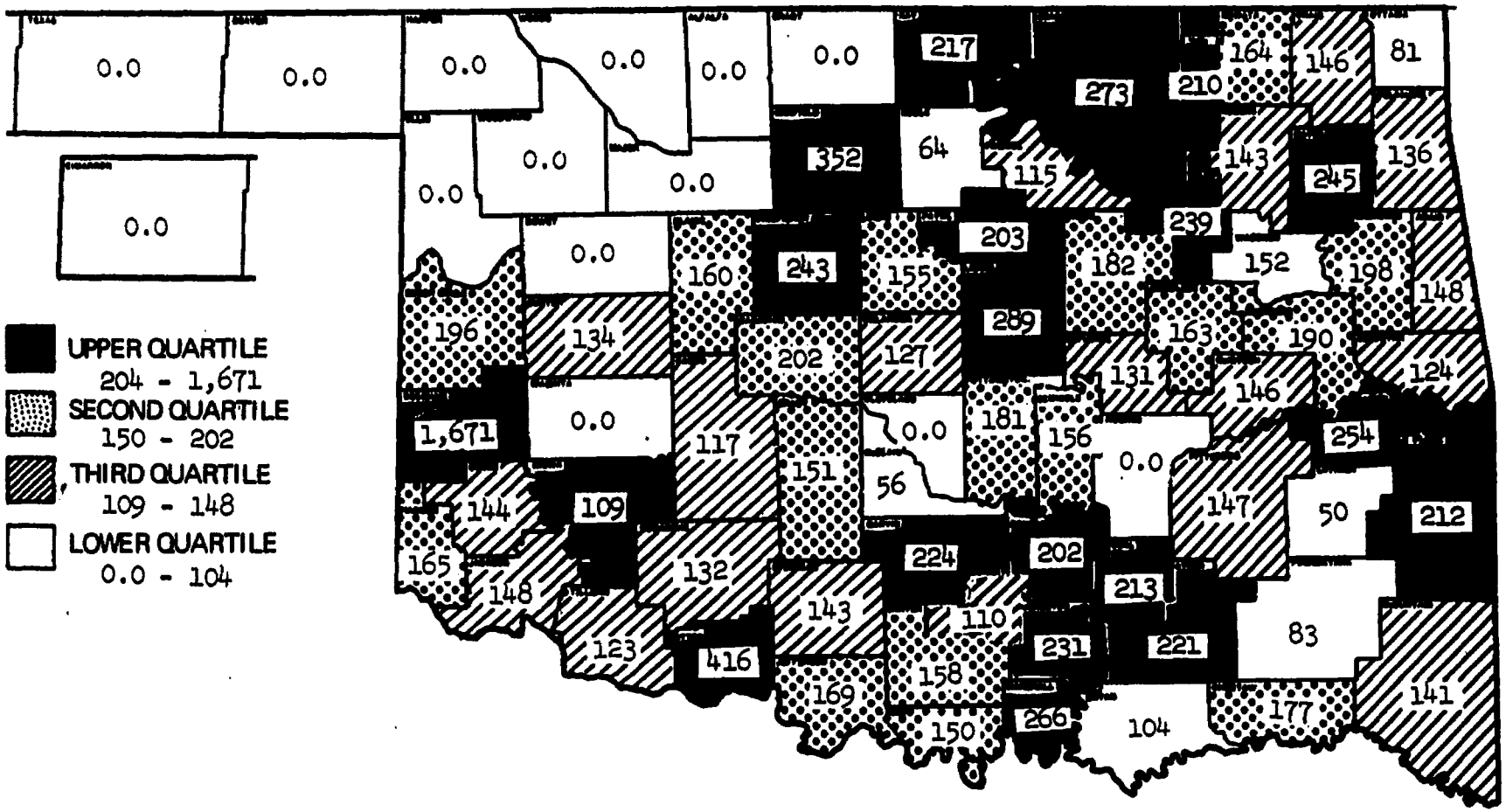

Figure 13. Arteriosclerotic and Degenerativ : Heart Disease (ISC 420-422) Non-White Females

quartiles respectively. All counties in the Panhandle and northwest portion show no rate at all in either Figure 12, non-white males or Figure 13 non-white females.

The average annual age-adjusted death rate for arteriosclerotic and degenerative heart disease (ISC 420-422) are higher than all the rates presented for any of the other diseases.

The distribution of the average annual age-adjusted death rate for nephritis and nephrosis (ISC 590-594) for white males, white females, non-white males, and females is presented in Figures 14 through 17. The distribution for white males (Figure 14), and white females (Figure 15) shows no cluster areas, with most of the counties (Figure 14) appearing in the second and third quartiles. In Figure 15, the southern border shows the highest age-adjusted of any other area in the state, with seven of its eleven counties appearing in the upper quartile (Figure 15).

The non-white male (Figure 16) distribution of nephritis and nephrosis shows no rate at all for over two-thirds of the counties in the state. There is one cluster area which extends from Oklahoma county in the central part of the state to Latimer county in the southeast portion of the state. The geographical distribution of the age-adjusted death rates from nephritis and nephrosis for non-white females (Figure 17) appears to be similar to the non-white male distribution (Figure 16) for nephritis and nephrosis. Like the non-white male distribution, a large number of counties show no rates at all.

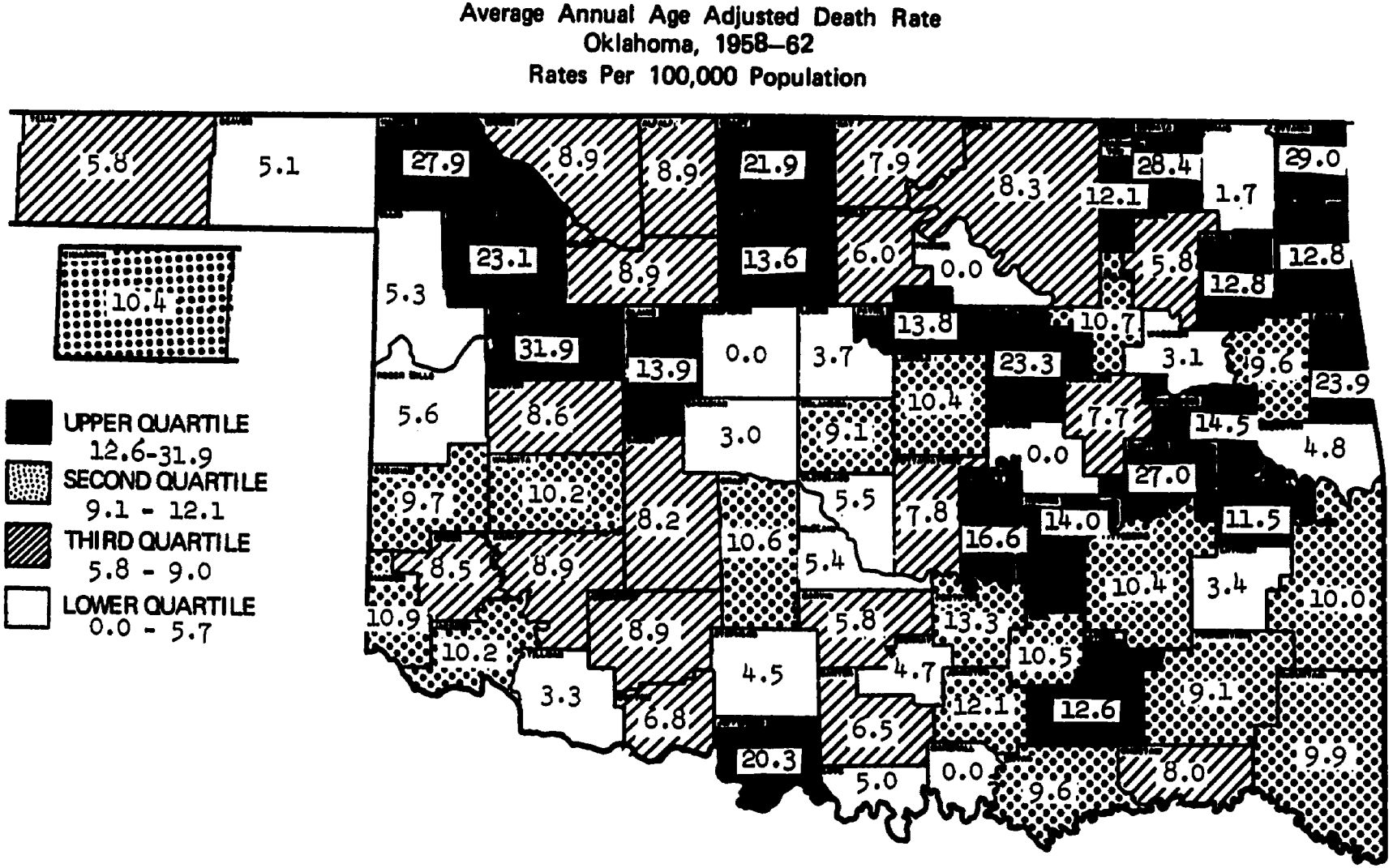

Figure 14. Nephritis and Nephrosis (ISC 590-594), White Males

Average Annual Age Adjusted Death Rate
Oklahoma, 1958-62
Rates Per 100,000 Population

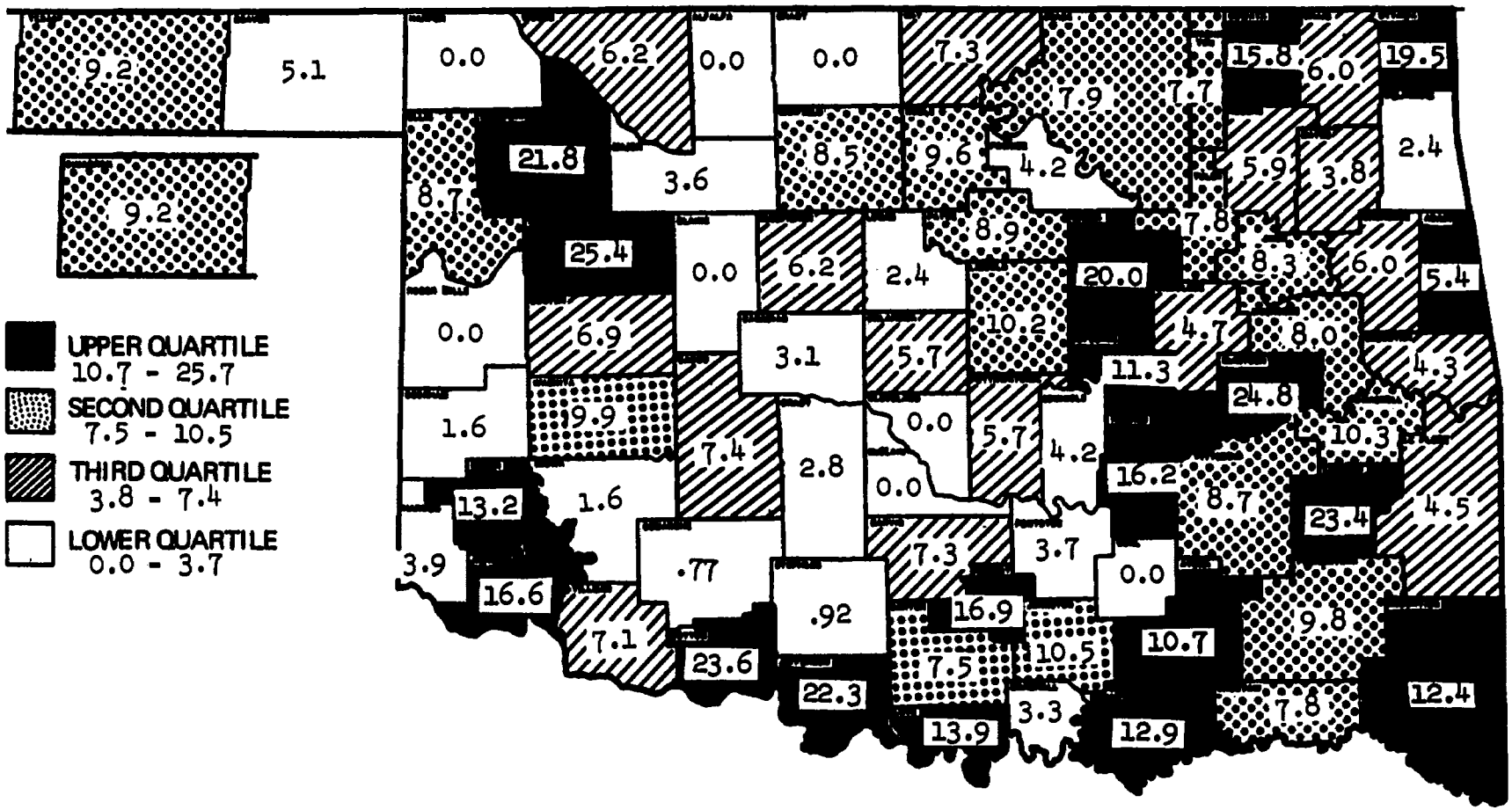

Figure 15. Nephritis and Nephrosis (ISC 590-594), White Females

Average Annual Age Adjusted Death Rate
Oklahoma, 1958-62
Rates Per 100,000 Population

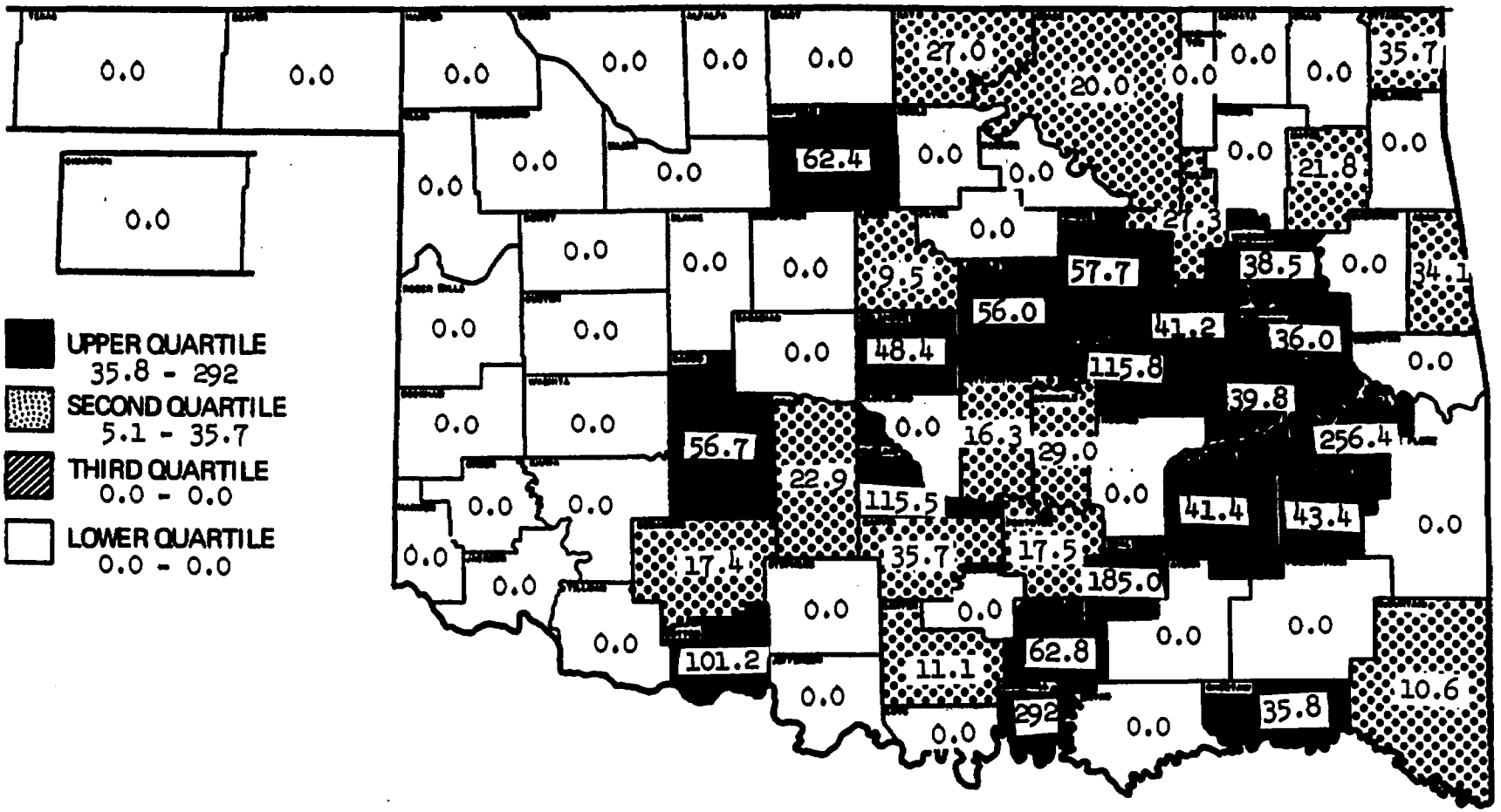

Figure 16. Nephritis and Nephrosis (ISC 590-594), Non-White Males.

Average Annual Age Adjusted Death Rate
Oklahoma, 1958-62
Rates Per 100,000 Population

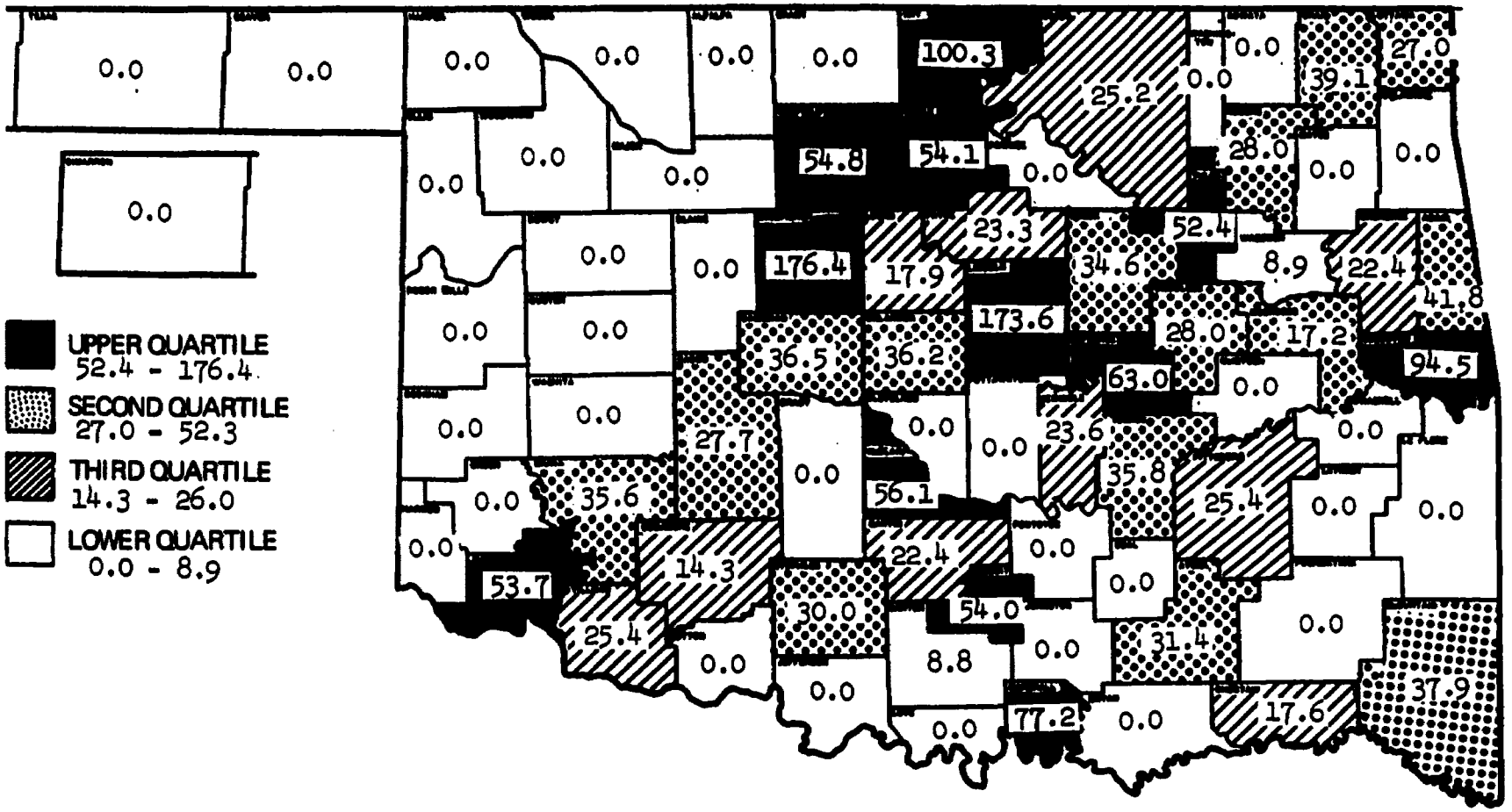

Figure 17. Nephritis and Nephrosis (ISC 590-594), Non-White Females.

CHAPTER IV

DISCUSSION

Age Distribution

The distribution of age-specific prevalence rates for the period studied are consistent with the trends reported for the rest of the United States (85).

One of the most obvious findings in almost all such surveys of the general population is that blood pressure on the average increases markedly with age, as is shown in Tables 4, 5, and 6. It is worth bearing in mind that age may be considered as one way of measuring the duration of environmental influences. In light of this, it would appear from this somewhat limited study that in the general population of these eight counties of the state that the rate of changes of blood pressure in the middle-aged is largely determined by both genetic and environmental factors or a combination of the two.

The trend in middle aged mortality from major cardiovascular causes including hypertensive disease (ISC 440-447) shows an increase with age as can be observed in Tables 13, 14, and 15. However, the mortality rates from hypertensive disease in all age groups seem to be lower than those of the other major cardiovascular diseases considered, except for the nephritis and nephrosis death rates. A downward trend in mortality rates from hypertensive disease with time has been

reported by other researchers whose findings have been similar. There are possibly several reasons for this decline: (1) improvement in treatment, (2) changes in classification of the cause of death, or (3) a true change in the prevalence of the disease (94).

A number of factors seem to be of importance when considering the age distribution of prevalence and mortality rates from hypertensive disease. However, there is disagreement about their magnitude and about the nature of their effects (85). Based on present evidence one would include such environmental influences as occupational exposure, composition of the diet, the nature of the water supplies and salt intake. In addition to these there are personal factors, not easily categorized as either genetic or environmental, for example, body weight.

Sex Differences

In the United States, hypertension tends to be more prevalent in women than in men, but more severe in men (95). The sex differences in Oklahoma seem to follow the pattern described above, with females showing the higher prevalence rates in the three racial groups considered as is shown in Table 6. The differences are quite marked after age 45, but prior to this age group the rates appear to be about the same.

In attempting to explain these sex differences it was suggested by Miall that personal factors, such as psychological factors, body weight, pregnancy, and parity might be responsible for the higher rates in females after the reproduction years (94). He further notes that the severity of the disease in males is possibly related to prolonged occupational exposure, depending on the occupation. This coupled with other environmental factors might be an important determinant in the severity of the

disease.

Sex difference in terms of the mortality experience from hypertensive disease (ISC 440-447) as presented (Figures 1, 2, 3, and 4: Tables 13 and 14), all show the white female experiencing slightly lower rates than her male counterpart, while the non-white female shows the highest age-adjusted rates of either race-sex-group considered. Similar findings along these same lines have been reported by several investigators (94). An explanation for the differences described above have been suggested by Lew. He suggests that systolic blood pressures in the range from 140 to 160 mm. Hg and diastolic blood pressures in the range from 90 to 100 mm. Hg, coupled with overweight adds to the prospective mortality, producing a significant curtailment in the expectation of life for individuals who are both overweight and hypertensive compared to corresponding hypertensives free of overweight. He further suggests evidence shows that women tolerate hypertension much better than men, and that women's longevity is much less affected than men's by a similar degree of overweight and hypertension (96). This along with other factors such as the psychological factors, differences in occupation, and personal habits might be a possible explanation for the differences seen between sexes. The high age-adjusted rates seen for the non-white female might possibly be related to her work role in the family, however, the contribution of the genetic factors coupled with the various environmental factors mentioned previously must also be considered.

Racial Difference

The non-white population of Oklahoma showed dramatic increases over the white population in mortality and prevalence from hypertensive

disease (ISC 440-447) (Tables 6, 9, 11, 13 and 14). Similar findings have been reported by several investigators (6, 76, 55). However, interpretation of the age-adjusted differences are difficult because the non-white population of Oklahoma constitutes two different racial groups, with the Negro population constituting over sixty percent and located mostly in the metropolitan counties. The Indian population which makes up the remainder of the non-white population is located primarily in the rural counties. It is rea[redacted]hat the availability and pattern of medical care [redacted]tudy for the two racial groups were not simil[redacted]ts that practices in treating hypertension [redacted] differences in hypertension, because much [redacted]erning Negro-white differences was present [redacted] of effective anti-hypertensive agents (97). [redacted] racial differences in hypertension could not be attributed to social class and occupation, but rather these differences are related to the genetic constitution of the populations (28). In dealing with racial differences, several factors other than the genetic constitution of the populations have to be considered. Diet, personal habit, sodium intake, body weight, and psychological factors have to be considered as possible contributory factors. Eighty percent of the non-white deaths from hypertensive disease in Oklahoma occurred in the Negro population. Also, Negroes comprise over sixty percent of the total non-white population, therefore, it is reasonable to assume that a combination of the above factors, both genetic and environmental, might be responsible for the differences in the age-adjusted rates for whites and non-whites in Oklahoma.

disease (ISC 440-447) (Tables 6, 9, 11, 13 and 14). Similar findings have been reported by several investigators (6, 76, 55). However, interpretation of the age-adjusted differences are difficult because the non-white population of Oklahoma constitutes two different racial groups, with the Negro population constituting over sixty percent and located mostly in the metropolitan counties. The Indian population which makes up the remainder of the non-white population is located primarily in the rural counties. It is reasonable to assume that the availability and pattern of medical care during the period under study for the two racial groups were not similar. However, McDonough suggests that practices in treating hypertension could not account for racial differences in hypertension, because much of the existing evidence concerning Negro-white differences was present prior to the introduction of effective anti-hypertensive agents (97). He also noted that racial differences in hypertension could not be attributed to social class and occupation, but rather these differences are related to the genetic constitution of the populations (28). In dealing with racial differences, several factors other than the genetic constitution of the populations have to be considered. Diet, personal habit, sodium intake, body weight, and psychological factors have to be considered as possible contributory factors. Eighty percent of the non-white deaths from hypertensive disease in Oklahoma occurred in the Negro population. Also, Negroes comprise over sixty percent of the total non-white population, therefore, it is reasonable to assume that a combination of the above factors, both genetic and environmental, might be responsible for the differences in the age-adjusted rates for whites and non-whites in Oklahoma.

Geographical Differences

Hypertension may cause death by coronary artery disease, cerebral vascular accident or renal failure. For this reason, deaths attributed to one of these categories could possibly be due to hypertensive disease and be signed out by the attending physician to one of the previously mentioned categories (diagnostic transfer). Because of the possibility of diagnostic transfer occurring between the categories of primary hypertension (ISC 440-447) and related cardiovascular-renal diseases, the geographical patterns of hypertensive disease mortality might have been seriously affected by such transfers. These findings have been reported by several studies (13, 52).

The data in this study combine hypertensive disease deaths with and without mention of associated heart disease in trying to reduce the affects of diagnostic transfer. In the case of the Oklahoma non-white populations the rates in the middle age groups for both cerebral vascular and nephritis and nephrosis are small compared with the rates for hypertension. However, correlation does appear when individual diseases are considered. A correlation analysis of the age-adjusted death rates from hypertensive disease and nephritis and nephrosis for all seventy-seven counties in the state yield a correlation coefficient of 0.690 (Table 20). A similar analysis of the age-adjusted rates from hypertensive disease and cerebrovascular disease resulted in a higher correlation coefficient of 0.797 (Table 20). Both coefficients were found to be significant at the five percent level, thereby suggesting an association between these rates in the state during the study period, and further suggesting that the occurrence of diagnostic transfers were not likely

in either case. Therefore, the differences in mortality patterns are not affected in either case by diagnostic transfer. These findings are similar to those reported by Rose (13).

The situation may be different with regard to arteriosclerotic heart disease (ISC 420-422). Here in the case of middle aged non-whites we are dealing with rates of the same order as those for hypertension; and in the case of the white population the arteriosclerotic heart disease rates are in some instances greater. Theoretically, then there is an opportunity for varying frequencies of transfers occurring on a large scale to account for the regional differences in hypertension mortality rates. In Oklahoma there is clearly a need for validity checks on the mortality statistics themselves, and also for field studies to determine the true nature of regional variation in blood pressure distribution and in the prevalence of hypertensive disease. Certain considerations, however, make it difficult to believe that the patterns of hypertensive mortality which are presented in Figures 1 through 4 are wholly due to diagnostic artifact:

1. The patterns are not consistent and yet in many instances it cuts across considerable variation in economic development and presumably in medical standards. This seems to be the case with whites and non-whites (Figures 1 - 4), however, the patterns for whites appear to be a more consistent one than the non-white pattern. Pushmataha county, located in the southeastern part of the state, shows the highest age-adjusted rate of all counties and appears in the upper quartile in all four race-sex groups.

2. Diagnostic transfers would have to be mainly into or out of the category of arteriosclerotic heart disease as suggested by Rose (13). If such transfers occurred on a significant scale, then one might also expect evidence of some transfer of the sex difference which is so characteristic of arteriosclerotic heart disease. However, this is not apparent as shown in Figures 10 through 13. The sex differences for hypertension mortality in Oklahoma is near unity and similar to sex differences described by other investigators (1, 5, 6). Furthermore, the geographical patterns for hypertension (Figures 1 through 4) are different from that for arteriosclerotic heart disease (Figures 10 through 13).

The only relationship which appears is a broad one with population density. But if factors linked with urbanization possess any casual importance as has been suggested by several investigators (10, 11, 12), this is certainly only part of the picture for high rates are seen in rural counties (Tables 16 and 17). An attempt to show urban-rural differences by presenting the age-adjusted rates for hypertensive disease (ISC 440-447), and related cardiovascular-renal diseases (ISC 420-422, 330-334, and 590-594) as shown in Tables 14, 15, 16, and 17. These rates have been described in CHAPTER III. Because of the similarity in the age-adjusted death rates from the ten most populated counties and the ten least populated counties, and since the rates were shown in both instances by race, the non-white/white differences in both areas also appeared to be similar. The non-white/white ratios when both urban and rural counties were considered together range from a low of

1.04 to a high of 5.62, which appeared in the least populated counties. A statistical analysis of the non-white/white ratios from these counties was performed (Table 19). The differences between the ratios of the ten most populated counties and the ten least populated was found not to be statistically significant at .05. Therefore, this suggests that the urban-rural differences described by other workers seem not to be as pronounced in Oklahoma. These findings are similar to those of other investigators (13, 14, 18). A possible explanation for this lack of difference in Oklahoma might in some way be connected with factors other than population density.

Such patterns as shown by hypertensive disease (Figures 1 through 4) and related cardiovascular-renal diseases (Figures 6 through 17) should prompt a search for an association with some factor linked with geographical characteristics of the land. Among these one must include the nature of the water supply, since there is now evidence of an association between the "hardness of water" and the level of mortality from cardiovascular diseases (22).

The geographical patterns of the related cardiovascular-renal diseases for both race-sex groups were not similar to those for hypertensive disease in any instance. However, the geographical patterns for the related cardiovascular-renal diseases were just as inconsistent as the distributions of hypertensive disease. This further suggest that transfers did not play a part in affecting these patterns.

Socio-Economic Differences

The differences in mortality by socio-economic districts is not easy to interpret in this type of study because the average annual

age-adjusted rates were calculated by county and not by districts. The overlapping of high rate counties from one socio-economic district to another appeared to be quite frequent. In spite of this the median average annual age-adjusted rates were taken for each district by race and presented in Table 21. Higher rates in both racial groups seem to be associated with the low socio-economic districts, and lower rates with the high socio-economic districts for whites. There were no rates for non-whites in the high district The adjusted mortality differences by social class may be influenced by differences in psychological factors, personal habits, occupation and body weight. The observed increases in the prevalence and mortality from hypertensive disease among the low socio-economic groups have been attributed to occupation, personal habits and body weight by several investigators (15, 20, 61, 66, 77 and 95).

CHAPTER V

SUMMARY

Mortality data from hypertensive disease (ISC 440-447) and related cardiovascular-renal diseases (ISC 330-334, 420-422, and 590-594) occurring to 51,557 Oklahoma residents during 1958-1962, and prevalence data obtained from the Chronic Disease Screening Survey Program of the Oklahoma State Health Department during the same period were analyzed. Age-race-sex specific and adjusted death rates and prevalence rates for all data were calculated for the period under study. Average annual age-adjusted death rates for whites and non-whites by sex were plotted on county maps. Data on the socio-economic distribution, geographic differences, age, sex, and race were analyzed.

The age-sex, and racial differences for hypertensive disease in Oklahoma are consistent with those reported in the rest of the United States. The nature of the association of these three variables with blood pressure and hypertensive disease mortality are well-known though the mechanisms involved are not. A definite increase with age was observed for both prevalence and mortality data. Negroes showed higher age-adjusted prevalence rates than whites or Indians, and Indians showed higher rates than whites. Non-whites showed higher adjusted mortality rates, with the non-white female experiencing the highest rates. Sex differences were observed in all three racial groups with

males showing higher rates generally, except in the case of the Negro.

The geographical distribution of adjusted mortality from hypertensive disease seem not to follow any definite patterns in either race-sex group considered. However, the geographical patterns of white males and females tend to be more related than the geographical patterns for non-white males and females. These patterns seem not to have been influenced by diagnostic transfers of related cardiovascular-renal diseases as evidenced by the fact that high positive correlation coefficients were obtained in each instance, when these disorders were correlated with hypertensive disease.

The distribution of the median age-adjusted death rates tend to be related to the socio-economic districts of the state. Higher rates in both racial groups seem to be associated with the low socio-economic districts, and lower rates with the high socio-economic districts for whites.

LIST OF REFERENCES

1. Stamler, J., Cardiovascular Diseases in the United States. The American Journal of Cardiology. 65: 320-340, 1960.

2. Metropolitan Life Insurance Company. Changing Mortality in the Types of Heart Diseases. Statistical Bulletin Metropolitan Life Insurance Company. 1962.

3. Chase, H. C., Variations in Heart Disease Mortality Among Counties of New York State. Public Health Reports. 78: 534, 1963.

4. Ferris, E. D., The Role of Hypertension. American Journal of Public Health. 50: 11-13, 1960.

5. Puffer, Ruth R., and Griffith, Wynne, G., Patterns of Urban Mortality. Pan American Health Organization. Washington, D. C., 1967.

6. Stamler, J., Kjelsberg, M., Hall, Y., and A. Scotch, N., Epidemiologic Studies on Cardiovascular-Renal Diseases. An Analysis of Mortality by Age, Race, Sex-Occupation and by Place of Residence, Including Urban-Rural Comparison, Journal of Chronic Diseases. 12: 440-456, 1960.

7. Breslow, L., and Buell, P., Mortality from Coronary Heart Disease and Physical Activity of Work in California. Journal of Chronic Diseases. 11: 420-425, 1960.

8. Mann, G. V., Roels, A. O., and Price, D. L., Cardiovascular Disease of African Pygmies - A Survey of the Health Status, Serum Lipids and Diet of Pygmies in the Congo. Journal of Chronic Disease. 15: 1017-1024, 1962.

9. Kannel, W. B., Epidemiology of Coronary Heart Disease. Geriatric. 17: 675-691, 1966.

10. John, B. C., and Remington, R. D., A Sampling Study of Blood Pressure Levels in White and Negro Residents of Nassau, Bahamas, Journal of Chronic Diseases. 13: 39-51, 1961.

11. Lennard, H. L., and Glock, C. Y., Studies in Hypertension. VI. Differences in the Distribution of Hypertension in Negroes and Whites: An Appraisal. Journal of Chronic Diseases. 5: 186-193, 1957.

12. Foe, S. M. and Skinner, J. S., Physical Activity and Cardiovascular Deaths. American Journal of Cardiology. 14: 731-737, 1964.

13. Rose, Geoffrey, The Distribution of Mortality from Hypertension Within the United States. Journal of Chronic Diseases. 15: 1017-1024, 1962.

14. Rose, Geoffrey, Cardiovascular Disease Mortality Among Negro Children. Archives Environmental Health. 5: 412, 1962.

15. Rose, Geoffrey. A. Study of Blood Pressure Among Negro Children. Journal of Chronic Diseases. 15: 373-380, 1962.

16. Sauer, H. I., and Enterline, P. E., Are Geographic Variations in Death Rates for the Cardiovascular Real? Journal of Chronic Diseases. 10: 513-519, 1959.

17. Takashashy, E. J., On the Geographic Distribution of Death Rates from Vascular Lesions Affecting the Central Nervous System in the United States. Tohoku Journal of Experimental Medicine. 76: 225-232, 1962.

18. Stamler, J., Arteriosclerotic Coronary Heart Disease, the Major Challenge to Contemporary Public Health and Preventive Medicine. Connecticut Medicine. 28: 675, 1964.

19. Stamler, R., Racial Differences in Heart Disease Risk Factors. Socio-psychological Aspects. The University of Chicago Press, 1962.

20. Wylie, E. C., Recent Trends in Mortality from Cerebrovascular Accidents in the United States. Journal of Chronic Diseases. 14: 213-220, 1961.

21. Weiner, L., Bellows, M. T., and McAvory, G. A., Use of Multiple Causes in the Classification of Deaths from Cardiovascular-Renal Diseases. Journal - American Public Health Association. 45: 492-501, 1955.

22. Yates, P. O., A Change of the Patterns of Cerebral Vascular Diseases. Lancet. 1: 65-59, 1964.

23. Spain, D., and Brades, V., Relationship of Sex, Age, and Physical Activity of Sudden Death from Coronary Occlusion. Journal of American Medical Association. 174: 384-388, 1960.

24. Pennel, M. Y., and Glover, M., Negro Mortality and Urban and Rural Mortality from Selected Causes in the North and South. Public Health Reports. 66: 295-305, 1951.

25. Schroeder, H. A., Degenerative Cardiovascular Diseases in the Orient. II: Hypertension. Journal of Chronic Diseases. 8: 312-316, 1958.

26. Schroeder, H. A., Relation Between Mortality from Cardiovascular Disease and Treated Water Supplies. Variation in States and 163 Largest Municipalities of the United States. Journal of American Medical Association. 172: 1902-1910, 1960.

27. Schroeder, H. A., and Nason, Alexis. Essential Trace Metals in Man: Cobalt. Journal of Chronic Diseases. 20: 869, 1967.

28. Schroeder, H. A., and Nason, Alexis. Essential Trace Metals in Man: Zinc. Relations to Environmental Cadmium. Journal of Chronic Diseases. 20: 179, 1967.

29. Lindeman, Robert, D., and Assenzo, Joseph R., Correlation Between Water Hardness and Cardiovascular Deaths in Oklahoma Counties. American Journal of Public Health. 54: 1071, 1964.

30. Dorn, N. P., Tobacco Consumption and Mortality from Cancer and Other. California Medicine. 89: 267, 1958.

31. Pickering, George. Hyperpiesio, High Blood Pressure Without Evident Cause. British Medical Journal. ii: 959-968, 1965.

32. Schweitzer, M. D., Gearing, E. R., and Perera, G. A., The Epidemiology of Primary Hypertension. Journal of Chronic Diseases. 18: 13-17, 1323, 1965.

33. Sacks, M. I., Aortic and Coronary Artherosclerosis in the Three Racial Groups in Cape Town. Circulation. 22: 87-96, 1960.

34. Bennet, C., Fokuyama, G., and McBride, T., Cardiovascular-Renal Mortality in Hawaii. American Journal of Public Health. 52: 1408-1412, 1962.

35. Kaaus, A. S., and Lilienfeld, A. M., Some Epidemiologic Aspects of the High Mortality Rate in the Young Widowed Group. Journal of Chronic Diseases. 10: 307, 1959.

36. Geiger, H. J., and Scotch, N. A., The Epidemiology of Essential Hypertension. Journal of Chronic Diseases. 16: 1151, 1963.

37. Gordon, Travis and Waterhouse, Alice, Hypertension and Hypertensive Heart Disease. Journal of Chronic Disease. 19: 1089-1100, 1966.

38. Groen, J. J., and Kallner, G., Mortality from Coronary (Arteriosclerotic) Heart Disease and Cerebrovascular-Accidents Among Eastern Immigrants in Israel. 21: 25-34, 1968.

39. Stamler, J., Kjelsberg, M., and Enterline, P. E., Epidemiologic Studies on Cardiovascular-Renal Disease. III. Analysis of Mortality by Age-Sex-Nationality. Journal of Chronic Diseases. 12: 464-470, 1960.

40. Gulranick, Lillian, Some Problems, in the Use of Multiple Causes of Death. Journal of Chronic Diseases. 19: 979-990, 1966.

41. James, George. Accuracy of Cause of Death Statements of Death Certificates. Public Health Reports. 70: 39-51, 1955.

42. Lowe, C. R., and McKeown, T., Arterial Pressure in an Industrial Population and its Bearing on the Problem of Essential Hypertension. Lancet. i: 1086, 1962.

43. Lowenstein, F. W., Blood Pressure in Relation to Age and Sex in the Tropics and Sub-tropics. Lancet. i: 389, 1961.

44. Epstein, F. H. Epidemiologic Studies on the Nature of High Blood Pressure. National Kidney Foundation Report. 1: 263-276, 1966.

45. Fulmer, H. A., Roberts, R. W., Coronary Heart Disease Among Navajo Indians. Annals of Internal Medicine. 59: 740-764, 1963.

46. Garcia-Palmeri, M. R., Coronary Heart Disease Mortality - A Death Certificate Study. Journal of Chronic Diseases. 18: 1317-1323, 1965.

47. Doyle, J. T., Dawber, T. R., Kannel, W. B., and Heslin, S. A., Cigarette Smoking and Coronary Heart Disease. New England Journal of Medicine. 266: 796, 1962.

48. Dawber, Thomas, R., Kannel, William B., and Kagan, Abraham. Environmental Factors in Hypertension. Journal of Chronic Diseases. 20: 2614-2620, 1967.

49. Chapman, J. M., and Massey, F. J., The Interrelationship of Serum Cholesterol, Hypertension, Body Weight, and Risk of Coronary Disease. Journal of Chronic Disease. 17: 933-937, 1964.

50. Bounton, R. E., and Todd, R. L., Relation of Body Weight and Family History of Hypertension Disease to Blood Pressure Levels in University Students, American Journal of Medical Science. 216: 392-397, 1948.

51. Bechgaard, P., Arterial Hypertension: Follow-Up Study of One Thousand Hypertonics. Acta Medical Scand. 172: 3, 1946.

52. Ambard, L., and Beaujard, E., Causes of Arterial Hypertension. Archives of General Medicine. 46: 520, 1940.

53. Doyle, A. E., and Fraser, P. E., Essential Hypertension and Inheritance of Vascular Reactivity. Lancet. 2: 590, 1961.

54. Acheson, Roy and Fowlem, Gilliam. On the Inheritance of Stature and Blood Pressure. Journal of Chronic Diseases. 20: 731-745, 1967.

55. Johnson, B. C., Epstein, F. H., Distribution and Familial Studies of Blood Pressures and Serum Cholesterol Levels in a Total Community. Tecumesh, Michigan. Journal of Chronic Diseases. 18: 147, 1965.

56. Phillip, J. H., and Burch, G. E., A Review of Cardiovascular Diseases in the White and Negro Races. Medicine. 39: 241, 1960.

57. Phillips, J. H. and Burch, G. E., Cardiovascular Diseases in White and Negro Races. American Journal of Medical Science. 238: 97, 1959.

58. Shah, V. V., Environmental Factors and Hypertension, with Particular Reference to Prevalence of Hypertension in Alcohol Addicts and Teetotalers, The Epidemiology of Hypertension. Grune and Stratton. pp. 204-213, 1967.

59. Howard, Jan and Tiedman, The Relative Effectiveness of Antihypertensive Drugs in Caucasians and Negroes. Clinical Pharmacy and Therapeutics. 8: 502-520, 1967.

60. Moser, M., Epidemiology of Hypertension with Particular Reference to the Bahamas. Part I., American Journal of Cardiology. 4: 727-733, 1966.

61. Moriyama, I. M., Stamber, J., Observation of Possible Factors Responsible for the Sex and the Race Trends in Cardiovascular-Renal Diseases. Public Health Reports. 66: 355, 1951.

62. Miall, W. E., and Oldham, P. D., The Hereditary Factors in Arterial Blood Pressure. British Medical Journal. 1: 75, 1963.

63. Epstein, Frederick, Epidemiologic Studies on the Nature of High Blood Pressures. Journal of Chronic Diseases. 17: 263-267, 1967.

64. Epstein, Frederick, The Epidemiology of Coronary Heart Disease. Journal of Chronic Diseases. 19: 735-774, 1965.

65. Comstock, G. W., An Epidemiological Study of Blood Pressure Levels in a Bi-racial Community. American Journal of Hygiene. 65: 268-274, 1957.

66. Campbell, M., The Main Cause of Increased Death Rate from Disease of the Heart, 1920 to 1959. British Medical Journal. 2: 712, 1963.

67. Keys, A., and White, P. D., Cardiovascular Epidemiology. Hoeber, 1956.

68. Pickering, George, The Nature of Essential Hypertension. Grune and Stratton, 1961.

69. Campbell, M., Death Rates from Diseases of the Heart, 1876 to 1959. British Medical Journal. 2: 528, 1963.

70. Alexander, D., McKee, and Adkins, E. E., Developmental Patterns of Coronary and Aortic Atherosclerosis in Young Negroes of Haiti and the United States. Annals of Internal Medicine. 61: 300, 1966.

71. Saul, K. L. and Ivrahim, M. A., The Distribution of Cardiovascular Disease in the Community. Annals of the Internal Medicine. 58: 627, 1963.

72. Wolf, S., Pfeiffer, J. B., Ropley, H. S. Winters, O. S., and Wolf, H. G., Hypertension as a Reaction Pattern to Stress: Summary of Experimental Data on Variations in Blood Pressure and Renal Blood Flow. Annals of Internal Medicine. 29: 1056, 1948.

73. Harris, R. E., The Distribution of Disease in the Population. Journal of Chronic Diseases. 12: 471-483, 1959.

74. Harris, R. E., Soklow, M., and Hunt, S. P., Responses to Psychologic Stress in Persons who are Potentially Hypertensive. Circulation. 7: 874-886, 1954.

75. Ostfeld, A. M., and Lebovits, B. Z., Blood Pressure Liability: A Correlative Study. Journal of Chronic Disease. 12: 428-437, 1960.

76. Sokolow, M., High Blood Pressure After Battle. Lancet. 1: 238, 1948.

77. Ruskin, A., Beard, O. W., and Schaffer, R. L., Elevated Arterial Pressure in Victims of the Texas City Disaster. American Journal of Medicine. 4: 228-239, 1960.

78. Poindexter, J. A., Epidemiological Survey Among Gola Tribes in Liberia. American Journal of Tropical Medicine. 11: 30-37, 1953.

79. Lowell, R. R., and Rogerson, G. W., The Casual Arterial Pressure of Fijians and Indians in Fiji. Annals of Medicine. 9: 4-12, 1960.

80. Scotch, N. A., Socio-cultural Factors in the Epidemiology of Zulu Hypertension. American Journal of Public Health. 53: 1205, 1963.

81. Loewenstine, F. W., Blood Pressure in Relation to Age and Sex in the Tropics and Subtropics. Lancet. 1: 389, 1961.

82. Pollack, H., and Krueger, D. E., Epidemiology of Cardiovascular Diseases Methodology. Hypertension and Atherosclerosis. American Journal of Public Health. 48: 20, 1960.

83. Perera, G. A., Hypertensive Vascular Disease: Description and Natural History. Journal of Chronic Disease. 15: 1017, 1962.

84. Conference on Culture, Society and Health. Special Sociocultural Factors in Chronic Organic Diseases. Annals of New York Academy of Science. 84: 789-797, 1960.

85. Thomas, C. B., Familial and Epidemiologic Aspects of Coronary Diseases and Hypertension. Journal of Chronic Diseases. 7: 198, 1958.

86. Francis, T., Aspects of the Tecumseh Study. Public Health Report. 76: 963 -977, 1961.

87. Adler, Jack, Bloss, Claude, and Mosley, Kirk. The Oklahoma State Department of Health Mobile Multiphasic Screening Program for Chronic Disease: Part I. American Journal of Public Health. 56: 918-925, 1966.

88. Adler, Jack, Bloss, Claude and Mosley, Kirk. The Oklahoma State Department of Health Mobile Multiphasic Screening Program for Chronic Disease: Part II. American Journal of Public Health. 56: 2066-2181, 1966.

89. International Classification of Diseases. World Health Organization Publication, 8th Edition: 16-20, 1958.

90. Assal, Nabih R., Geographic and Secular Clustering of Malignant Disease in Oklahoma, 1956-1965. Unpublished Dissertation.

91. Siegel, Sidney, Non-Parametric Statistics. McGraw-Hill Book Company, New York, pp. 42-111, 1956.

92. Epstein, F. M., Epidemiology Research in the Total Communities. Method of Information in Medicine. 3: 18-27, 1964.

93. Stamler, J., and Lindberg, H., Epidemiological Analysis of Hypertension Diseases in the Labor Force of a Chicago Utility Company. Proceedings of the Council for High Blood Pressure Research. p. 23, 1959.

94. Moriyama, I. M., Inquiry into Diagnostic Evidence Supporting Medical Certification of Deaths. American Journal of Public Health. 48: 1376, 1958.

95. Smith, T. H., Causes of Death in Treated Hypertensive Patients. British Medical Journal. 2: 1221, 1963.

96. Miall, E. W., and Oldham, P. D., Factors Influencing Arterial Pressure in the General Population in Jamaica. British Medical Journal. 2: 497, 1962.

97. Paffenbarger, Ralph, Milling, Robert, and Poe, Norman. Trends in Death Rates from Hypertensive Disease in Memphis, Tennessee, 1920-1960. Journal of Chronic Diseases. 19: 65-69, 1966.

98. Lew, A. E., Some Implications of Mortality Statistics Relating to Coronary Artery Disease. Journal of Chronic Diseases. 6: 192-209, 1957.

99. Huchard, J., Hypertension and Nephritis. Lea and Febiger, Philadelphia, 5th Edition, 1954.

100. Kuller, Lewis, Lilenfeld, A., Epidemiological Study of Sudden and Unexpected Deaths Due to Arteriosclerotic Heart Disease. Circulation. 34: 1056-1068, 1967.

101. Kuller, Lewis, Cerebrovascular Disease Mortality Among Baltimore Residents Ages 20-64. American Journal of Public Health. 57: 2089-2103, 1967.

102. Kobayashy, J., Geographic Relationship Between Chemical Nature of River Water and Death Rate from Apoplexy. Berichte Institute of Landwirtsch. 11: 12, 1957.

103. Kean, B. H., The Blood Pressure of the Cuna Indians. Archives of Internal Medicine. 83: 355, 1949.

104. Lindeman, Robert D., Bloss, Claude, M., Brown, Forest R., and Moseley, Kirk T., The Oklahoma State Department of Health Mobile Unit Chronic Disease Screening Program. Journal of Oklahoma State Medical Association. 20: 365-375, 1962.

105. Lilienfeld, Abraham, and F., Gifford, Alice, J., Chronic Diseases and Public Health. The John Hopkins Press, 1966. p. 561.

106. Meeneley, G. R., Tucher, R. G., Darby, W. J., and Auerbach, S. H., Chronic Sodium Chloride Toxicity, Hypertension, Renal and Vascular Lesions. Annals of Internal Medicine. 39: 999, 1953.

107. McDonough, J. R., Garrison, C. E., and Hames, C. G., Blood Pressure and Hypertensive Disease Among Negroes and Whites. A Study of Evans County, Georgia. Annals of Internal Medicine. 61: 208-228, 1964.

108. McLeahy, R., Influence of Water Hardness and Rainfall on the Incidence of Cerebrovascular Accident Mortality in Ireland. Journal of Irish Medicine. 55: 17-18, 1964.

109. Manison, S. L., and Morris, J. W., The Nature of Essential Hypertension . Lancet. ii: 829, 1960.

110. Moriyama, I. M. and Worelsey, T. D., Statistical Studies of Heart Disease, Race and Sex Differences in Trends of Mortality from the Major Cardiovascular-Renal Diseases. Public Health Reports. 66: 355, 1951.

111. Tukel, W. J., and Lewis, R. J., A Short Time Community Study of Coronary Heart Disease: A Preliminary Report of the North Dakota Study. American Journal of Cardiology. 49: 1630, 1962.

112. Miller, D. C., Spencer, S. S., and White, P. D., Survey of Cardiovascular Disease Among Africans in the Albert Schweitzer Hospital in 1960. American Journal of Cardiology. 10: 432, 1962.

113. Platt, R., Heredity in Hypertension. Lancet. i: 25, 1964.

114. Winkelstein, W., Familial Aggregation of Blood Pressure. Journal of American Medical Association. 195: 848-850, 1966.

115. Vital Statistics of the United States. National Vital Statistics Division, Public Health Service. Washington, D. C., II: pp. 1-23, 1960.

116. World Health Organization Technical Report Series No. 168. Hypertension and Coronary Heart Diseases, Classification and Criteria for Epidemiological Studies. Geneva, 1959.

117. Metropolitan Life Insurance Company. A Decade of Population Growth, White and Non-White. Statistical Bulletin Metropolitan Life Insurance Co., 1962.

118. Ferris, E. D., The Role of Hypertension. American Journal of Public Health. 50: 11-13, 1960.

119. Dixon, W. J., and Massey, F. J., Introduction to Statistical Analysis. Edition 2. McGraw-Hill Book Company, 1957.