SUMMARY Differences in blood pressure between blacks and whites in the United States are now well documented. The causes of these differences remain speculative. Genetic factors, personal characteristics, renal physiology, endocrine factors, autonomic nervous system function, cardiac function and various environmental factors are examined in the present review as potential determinants of racial blood pressure differences. Racial differences in renal physiology and environmental influences such as socioeconomic status seem to be likely candidates for important contributions to blood pressure differences. Further research aimed specifically at black-white blood pressure differences is of great importance to a fundamental understanding of the etiology and prevention of essential hypertension. (Hypertension 1: 468-475, 1979)

KEY WORDS • hypertension • blood pressure • blacks • Negro • race • pathophysiology • epidemiology

Several decades after the advent of a reliable method for indirect measurement of blood pressure, reports of population surveys of blood pressure began to appear. The first systematic surveys were in whites only. However, in the 1930's, data began to emerge suggesting higher blood pressure levels in blacks in selected populations. Yet as late as 1951, black-white differences in blood pressure were felt by some to be not well substantiated by published evidence due to methodological problems of the studies. The Muscogee County Georgia Study firmly established black-white differences in a biracial community using standardized methods. Since that time, these findings have been replicated in numerous surveys in the United States and in the West Indies. Population-based data on blood pressure from the many countries in Africa show variable results with regard to interracial comparisons.

Consistent with the higher average blood pressure readings in blacks compared to whites in the United States is the well-documented finding of a greatly increased prevalence of hypertension in blacks as compared to whites. This excess seems almost entirely due to essential hypertension. There is little hard data concerning the true population prevalence or incidence of secondary hypertension in blacks or in whites. The relative frequency of secondary hypertension may be lower in blacks, perhaps less than 5% of all adult hypertension. Examination of the racial composition of series of cases or autopsies with various
forms of secondary hypertension, especially in the absence of adequate control series, may be misleading because of possible bias in selection of cases for secondary hypertension evaluation. Nevertheless the following observations are of some interest.

Renovascular hypertension seems much less common among black hypertensives. Less than 10% of cases of renovascular hypertension have been found among blacks in a Dallas clinic population, even though blacks make up 75% of the hypertensive clinic population. Little information exists on the true prevalence of primary aldosteronism; however, autopsy series indicate that black women may have a greater frequency of adrenal adenomas than white women. Pheochromocytoma is said to occur in about 0.08% of autopsies in blacks and about 0.06% of hospital series must be regarded cautiously since they and whites.

Coarctation of the aorta is said to occur in approximately equal frequency in blacks and whites. All such estimates from autopsy or hospital series must be regarded cautiously since they may reflect neither the true population prevalence nor the black-white prevalence ratio due to selection factors, which may differ between races. Little information exists on the relative frequencies of other forms of secondary hypertension in blacks and whites.

The remainder of this paper will deal with essential hypertension. The known determinants of blood pressure will be considered and each evaluated as a possible explanation for black-white blood pressure differences. In addition, other hypothesized explanations for black-white blood pressure differences will be considered.

Genetic Factors

Many studies have shown in various populations that blood pressure is under genetic control. It is not yet clear, however, to what degree genes exert their effect on blood pressure via the various well-known determinants of blood pressure such as body weight, rather than by more direct or hitherto unknown mechanisms. Such “direct” genetic control of blood pressure may account for some of the black-white differences observed but there is little evidence on this from studies which have controlled for the various known determinants of blood pressure. Several studies have attempted to use skin color and blood groups as genetic markers to study black-white blood pressure differences. However one must examine the findings of these studies very carefully with regard to the various confounding factors which may relate to both skin color and blood pressure. Initial reports from the Charleston, South Carolina heart study indicated that darker skin color is related to higher blood pressure among blacks. However, in later reports, when social class was controlled for, this apparent effect of skin color disappeared. The Detroit Project studies of skin color and blood pressure in 1000 blacks and whites found that among blacks darker skin color was significantly related to blood pressure independent of environmental stressors, area of residence, education, income, and other factors, especially in younger black men. Among whites, there was no significant correlation of skin color and blood pressure, although there was a significant relationship of ethnic origin and blood pressure. Methodologic differences in sample selection and classification of skin color among blacks may account for the discrepancies between the Detroit Study and other studies. This is suggested by the fact that no consistent relation between blood pressure and socioeconomic status was found in Detroit in contrast to other studies.

In addition to studies of skin color, the Detroit Project utilized a family-set method to study family aggregation of hypertension and to generate estimates of heritability of blood pressure among blacks and whites. These analyses of data from 2305 individuals in 461 five-member family sets reveals no significant racial differences in family aggregation of hypertension or in heritability of systolic or diastolic blood pressure.

No correlation between blood pressure and the degree of black-white admixture as measured by blood group analysis was found in northeastern Brazil. A statistically significant regression of diastolic blood pressure on a serologically determined estimate of percent African admixture was found in a United States black clinic population undergoing multiphasic screening. However multiple possible confounding factors were not controlled.

The African gene pool in black populations of the United States originates chiefly from the various peoples of the West African coast. Blood pressures of these peoples measured in Africa are generally found to be lower than those of United States blacks and may be lower, the same, or higher than United States whites depending on the circumstances and techniques of measurement used. Little information exists on the blood pressures of African blacks and whites living in Africa under similar circumstances or of recent African immigrants living in the United States.

Thus there is little hard evidence from studies of skin color or blood groups to support or refute the hypothesis that black-white differences in blood pressure arise from single or multiple genes, although it is clear from family studies of blood pressure that genetic factors are important within each group.

Personal Characteristics and Blood Pressure

Blood pressure increases with age in black and white populations in the United States. Black and white blood pressures are similar until adolescence. Thereafter blood pressure and the prevalence of hypertension, increase much more rapidly with age in blacks than whites. Particularly steep rises have been noted in rural populations after the age of 25 in males and 35 in females. Because of these differences it is important to consider age-specific data when comparing the blood pressures in black and white populations.

The sex differences in blood pressure observed in black adults have not been observed in white adults. In
white populations the blood pressure is equal in both sexes until adulthood. Thereafter men's blood pressures tend to be greater than women's until about age 45, when women's blood pressures become greater than men's. In the Charleston Heart Study, black women had greater blood pressure than black men at all ages over 35 and the rise with age was steeper in the black women. Black women and black men had similar blood pressures at all ages between 8 and 65 years in the Muscogee County Georgia Study except for slightly higher systolic blood pressure in women after age 35. In Evans County, systolic blood pressure was higher in black women than in black men after age 25; diastolic blood pressure was also higher in black women than black men. However, in the Evans County Study the blood pressures in white women were higher than in white men after 30 years and diastolic blood pressures were similar in white men and women at all ages. These differences in the age-sex blood pressure distribution of black and white populations have not been explained.

Weight and various indices of obesity show similar relationships to blood pressure in blacks and whites, differing in the strength of association in some studies. In the Charleston Heart Study, blood pressure was weakly correlated with weight in all race-sex groups; however, the correlation coefficients were slightly larger for whites than for blacks. The correlations among blacks were not significantly different from zero or from the correlations among whites. No clear correlation between relative weight and prevalence of hypertension was found in blacks, although such a relationship was apparent in whites. As in many other studies, the prevalence of obesity was noted to be highest among black females. Quetelet Index was strongly related to blood pressure in all groups except black men, among whom only weak and inconsistent associations were found in young adults in the Evans County Georgia Study. Later multivariate analyses of the Evans County data revealed that the slope of regression of blood pressure on Quetelet Index was greater in whites than blacks when age-sex standardized Z scores for blood pressure were used. Consequently, black-white blood pressure differences were greatest in the lean and least in the fat. The slope of regression of blood pressure change on weight change over a 7-year follow-up period was the same in blacks and whites. Computations of attributable risks based on these data suggest that by controlling both baseline weight and weight gain over a follow-up period, 47.7% of new cases of hypertension could be "prevented" in whites but only 27.5% in blacks. Weight distributions were grossly similar in blacks and whites, although white men tended to be heavier than black men and black women tended to be heavier than white women. It was also estimated that a 27.9% decrease in incidence of hypertension in whites and a 16.9% decrease in blacks might be achieved by weight control in the 12% of whites and 14% of blacks who were overweight. In contrast, similar correlations of blood pressure with weight in all age-race-sex groups were reported in Chicago community surveys, suggesting urban-rural and north-south differences in the relationship of weight to blood pressure in blacks.

Renal Physiology

There is growing evidence that differences in renal physiology may play a role in black-white blood pressure differences. The renin-angiotensin system is more frequently suppressed relative to sodium intake and excretion in blacks than in whites. Many investigators have reported lower plasma renin activity levels relative to sodium excretion in clinical series of black as compared to white hypertensives. In various studies, 36–62% of black hypertensives have relatively suppressed plasma renin activities as compared to 19–55% of white hypertensives. In addition, plasma renin activities are lower in black than white normotensives. Most studies report a decrease in plasma renin activity with age in whites and blacks. One study, however, reported an increasing frequency of low plasma renin levels with age in black hypertensives but not in white hypertensives, leading to the postulation that this may explain the greater excess mortality and morbidity from hypertensive disease in blacks compared to whites under the age of 50 than thereafter. However, this finding and the apparent protective effect of low renin levels demonstrated in this study have not been substantiated in numerous other studies. All of these studies are limited by being clinic-based rather than population-based and frequently without adequate control series. Therefore, the possible differential operation of various selection factors may have led to inaccurate estimates of absolute and relative frequencies of low renin levels.

Renal control of plasma volume through the excretion of sodium and water may be somewhat different in black as compared to white hypertensives and low as compared to normal-renin hypertensives. Decreased urinary sodium excretion and creatinine clearance in black low-renin hypertensives as compared to those with normal renin has been reported. In addition, decreased frequency of blood pressure rise with upright posture was associated with low renin. Decreased blood urea nitrogen concentrations in low-renin hypertensives, perhaps indicating plasma volume expansion, have been reported. Others, however, report no difference in serum creatinine in low-renin hypertensives as compared to those with normal renin. Black hypertensive men were found to have greater plasma volume than white hypertensive men after correction for body surface area in one small series of patients. Decreased excretion of sodium and potassium during the 24 hours after a saline load has been reported in blacks compared to whites. In this group of subjects, baseline plasma renin activities and plasma aldosterone levels and sodium excretions were comparable in blacks than in whites after a saline load and after furosemide. The investigators reporting these data hypothesize that blacks may have evolved more efficient mechanisms for renal sodium conservation to cope...
with the low sodium intake in tropical or subtropical environments and that this protective mechanism may place blacks at a disadvantage when exposed to high sodium intakes of Western diets resulting in a greater propensity to hypertension. Oral and intravenous sodium loading has recently been reported to lead to steeper blood pressure increases in black than white normotensives. Studies of renal function and electrolyte excretion in free-living black and white children in Bogalusa, Louisiana reveal no racial differences in sodium excretion but a consistently lower potassium excretion in blacks than whites in all blood pressure strata. Twenty-four-hour dietary recalls revealed no difference in sodium or potassium intake between blacks and whites. Although such dietary data are generally considered less accurate than urinary excretions, these findings may indicate a differential handling of potassium by the kidney in black as compared to white children at an age when blood pressures are similar.

Renal vasodepressor substances may play a role in blood pressure control. Kallikrein, an enzyme which produces the precursor of bradykinin from its substrate, kininogen, occurs in lower concentrations in the urine of hypertensives as compared with normotensives. Dietary sodium intake of less than 20 mEq/day is associated with increased urinary excretion of kallikrein as compared to intakes greater than 150 mEq/day. This response tends to be blunted in black men with essential hypertension. Several investigators have reported racial differences in urinary excretion of kallikrein. In a group of 700 children, urinary kallikrein concentration has been reported to be only about one-third as high in blacks as in whites. However there were no black-white blood pressure differences in these children, even though kallikrein concentration was inversely related to blood pressure in both groups. In a study of adults, black normotensives had lower urinary kallikrein excretions than whites on a high sodium diet; excretions were not significantly different in black as compared to white hypertensives. Urinary kallikrein excretions were similar in black and white normotensives on a low sodium diet but black hypertensives had lower levels than white hypertensives. Urinary kallikrein excretion was correlated with renal blood flow in all groups except black normotensives on a low sodium diet. Information on possible racial differences in other renal vasodepressors, such as prostaglandins, is not available. Although the findings in these studies are not easily interpreted, they do indicate that renal vasodepressors may be a fruitful area for future research.

Renal vascular responses to blood pressure elevations of any cause might be important in the self perpetuation of hypertension and in racial differences in absolute blood pressure levels, hypertension prevalence and renal function. In the study cited above, black and white normotensives had similar renal blood flows on high and low sodium diets. However, on the low sodium diet, black hypertensives had lower renal blood flows than black or white normotensives or white hypertensives. A higher prevalence of proteinuria at each level of blood pressure in blacks as compared to whites has been reported. However, this might indicate a longer duration of elevated blood pressure in blacks rather than a racial difference in renal response to elevated blood pressure. Blacks with malignant hypertension have been reported to show myxoid intimal thickening of small intrarenal arteries as the predominant lesion rather than the fibrinoid necrosis or atypical necrosis that is described in whites. Again it is uncertain whether these findings represent racial differences in response of renal vasculature to a given level of elevated blood pressure or merely different duration, severity and extent of therapy in blacks versus whites with malignant hypertension.

Endocrine Factors

The higher prevalence of low-renin hypertension in blacks than in whites would suggest that different mineralocorticoid secretion by the adrenal cortex could play a role in racial differences of blood pressure. A study of 15,000 patients over the age of 20 years autopsied at Johns Hopkins Hospital found a highly significant excess of all forms of hypertension in patients with adrenal cortical abnormalities at autopsy compared with matched controls. Analysis by age and race revealed greater excesses of hypertension in younger blacks than in younger whites with adrenal cortical abnormalities. A higher prevalence of cortical adenomas was found in black women compared to white women under the age of 60 years. The prevalence was also higher in black men as compared to white men but this did not reach statistical significance. Blacks with adrenal cortical abnormalities had 1.9 times the prevalence of hypertension as controls, while in whites the excess was 1.5 times. Considering only essential hypertension, the excess prevalence was 1.76 times in blacks and 1.17 times in whites with adrenal cortical abnormalities. This differential was most marked in younger blacks. A strong association of adrenal abnormalities with accelerated hypertension was shown in younger blacks but not among whites. Of possible relevance is a study reporting that 91% of a series of low-renin hypertensives who were surgically explored had adrenal cortical abnormalities. Since all but a few of these cases were white, no information on racial differences in the pathology of low-renin hypertension is available.

Autonomic Nervous System Function

Possible differences in autonomic nervous system functioning in blacks and whites provides another set of puzzling findings regarding racial blood pressure differences. Two possible indicators of autonomic nervous activities suggest racial differences but not in the expected direction. Plasma renin activity could be an
indicator of the level of sympathetic tone. Lower plasma renin activity levels in blacks would therefore tend to indicate a decreased sympathetic tone. In addition, among adults heart rates are lower in blacks than in whites, again consistent with a lesser amount of sympathetic tone. Black infants have been reported to have more rapid heart rates than white infants, however, among children aged 5 to 14, blacks had slower heart rates. In infants and children there are no consistent black-white blood pressure differences despite the heart rate differences. The findings among adults are puzzling since heart rate is in general positively correlated with blood pressure and the incidence of hypertension. Although interpretation of dopamine-beta-hydroxylase (DBH) levels among individuals is controversial, it is of interest to note reports that DBH levels were not different in hypertensives versus normotensives but were lower in blacks than whites in both normal and hypertensive adults. Dopamine-beta-hydroxylase levels have also been reported to be lower in black than white children. Again this would tend to indicate a lower level of sympathetic tone in blacks. How this might relate to higher blood pressures and greater frequency of hypertension in black adults is unclear. However, a unifying hypothesis might be that decreased sympathetic tone and consequent lower plasma renin activity and heart rate could be a result of relative volume expansion of primary renal origin.

Cardiac Function

Alterations of cardiac function have been identified in subgroups of hypertensive patients early and late in the course of essential hypertension. Little information exists, however, about racial differences in cardiac function. Blacks have been included in series of cases of hyperkinetic circulatory states with and without hypertension. Pulse pressure was reported to be greater in blacks than whites after age 25 in Muscogee County Georgia. However, the relative frequencies of such states in black and white normotensives and hypertensives is unknown.

Hypertensive heart disease is more prevalent in blacks than whites even after controlling for blood pressure level at the time of examination. More left ventricular hypertrophy noted on electrocardiogram and more cardiac enlargement found by x-ray and physical examination at each blood pressure level in blacks as compared to whites was reported from the Evans County Georgia Study. The Hypertension Detection and Follow-up Program found more left ventricular hypertrophy by electrocardiogram in blacks than whites even after adjustment for age and blood pressure level among 10,940 hypertensives aged 30 to 69 years. Whether these findings indicate an increased susceptibility to hypertensive heart disease at a given blood pressure level or simply a longer duration of elevated blood pressure and less effective treatment in blacks as compared to whites must remain speculative.

Hypertensive Vascular Disease

Gross racial differences exist in the occurrence of hypertensive vascular disease. As reported above, population studies indicate higher prevalence of hypertensive renal and heart disease in blacks than whites even after stratifying on age and blood pressure level at examination. An increased prevalence of hypertensive retinal changes at diastolic blood pressure levels above 100 mm Hg in blacks as compared to whites also has been reported. Clinical studies tend to support these findings, although there is one report of the relative rarity of severe hypertensive retinopathy in Africans with severe hypertension.

Vital statistics indicate that, overall, blacks die three times as frequently as whites from hypertensive diseases in the United States. Of special interest is the fact that between the ages of 35 to 54 years, blacks died six to 10 times as frequently from hypertensive disease according to 1973 statistics, an excess far out of proportion to the approximate two times excess of prevalence of hypertension in blacks. After the age of 55 years the excess mortality is more nearly proportionate to the prevalence, especially in the older age groups. Some of this age-related difference in excess mortality in blacks as compared to whites could be an artifact of the death certification process. However, even when all cardiovascular diseases are considered together, the excess mortality in younger blacks as compared to whites is disproportionate to hypertension prevalence, especially in women.

The degree to which the foregoing observations indicate 1) greater susceptibility of blacks to vascular damage from hypertension; 2) earlier onset and greater severity of hypertension in blacks; or 3) later detection and less adequate treatment and control of hypertension in blacks is not clear. There is at least evidence for the latter two possibilities, whereas there is no direct evidence for the former.

Population, clinical, pathological studies and vital statistics indicate that blacks may have a greater susceptibility to atherosclerosis of the intracranial vessels and a lesser susceptibility to that of coronary vessels in response to hypertension and other risk factors. In large autopsy series in New Orleans and in Norway, more atherosclerosis in intracranial arteries and as much or more in cervical arteries was found in blacks as compared to whites; whites had more atherosclerosis in the aorta and coronary arteries. The Joint Study of Extracranial Artery Occlusion reported greater frequency of intracranial occlusion in blacks and greater frequency of extracranial occlusion in white patients. Black patients had fewer transient ischemic attacks and more severe hypertension and tended to be younger than white patients. In the Evans County Georgia Study, black men had less coronary heart disease than white men at each level of cholesterol and blood pressure. Among young women, coronary heart disease occurred with equal frequency in blacks and whites but among older women the rates were greater in whites than in blacks. Death rates for ischemic heart disease are similar in
black and white men whereas those for cerebrovascular disease are twice as high in blacks. Among women both ischemic heart disease and cerebrovascular disease rates are twice as high in blacks as in whites.8

Environmental Factors

A number of environmental factors that may be related to essential hypertension have been identified. Several of these may affect blacks more frequently or to a greater extent than whites and hence might be related to racial blood pressure differences. Dietary factors, particularly sodium intake, have long been postulated as contributing to black-white differences in blood pressure levels in the United States. However, there is no objective documentation of higher sodium intakes in blacks than in whites despite many anecdotal observations to the contrary. Both black and white Americans have high sodium intakes. Studies of sodium intakes in blacks and whites in Evans County Georgia found no difference in black and white sodium intakes as measured by 24-hour urine collection.76 Similar findings are reported from Mississippi.77 Lower intakes of potassium and calcium in blacks as compared to whites resulting in higher sodium-to-potassium and sodium-to-calcium ratios in blacks have been reported from Mississippi.78 Black children in Louisiana excreted less potassium than whites, although dietary recalls indicated similar intakes. The ratio of urinary sodium-to-potassium excretion has been positively correlated to blood pressure.79 Both potassium and calcium have been shown to promote sodium excretion in animal studies.80 Thus a culturally and economically determined relative deficiency of dietary potassium and calcium among blacks has been postulated to contribute to the higher blood pressures and occurrence of hypertension observed among blacks as compared to whites.81

Blacks are more frequently exposed to conditions of poverty, low occupational and educational status and high levels of socioecologic stress, all of which are related to the prevalence of hypertension in both blacks and whites.82, 83 In addition, racial discrimination has long been postulated to be a stress-creating factor for blacks in the United States which might play a role in the pathogenesis of essential hypertension.84 Differences in the level of occupational or leisure-time physical activity seem unlikely to be sufficient to explain the higher blood pressures or prevalence of hypertension among blacks, although there is little information on this point. Effects of oral contraceptives or toxemia of pregnancy in black women as compared to white women are more likely to be related to a common cause of increased hypertension rather than be the causes themselves.

Summary and Conclusions

Racial differences in renal physiology may play a role in causing racial blood pressure differences. Although these differences may be genetically determined, there is little evidence for black-white blood pressure differences from other genetic mechanisms. Obesity may be relatively less important in the etiology of hypertension in blacks. Other nutritional factors such as a relative deficiency of potassium and calcium intake may be involved in racial differences. In addition, psychological and socioeconomic factors probably contribute to the excess of hypertension and higher blood pressure levels in blacks. Excess black mortality and morbidity from hypertension-related diseases out of proportion to the prevalence of hypertension itself is probably the result of earlier onset and greater severity of high blood pressure and less adequate therapy in blacks, although differential susceptibility cannot be excluded.

Some progress has been made since the statement in 1955 that "data . . . do not suffice to indicate the importance of genetic, psychologic, and social factors in explaining racial differences."85 However, more research aimed at the mechanisms of black-white blood pressure differences is of great importance to a fundamental understanding of essential hypertension, as well as the effective control of the leading health problem for the black population of the United States. Clinical and community trials are needed in the primary prevention of essential hypertension through the reduction of obesity, sodium intake, and physical inactivity. These should be undertaken in black as well as white populations as a necessary supplement to current efforts in hypertension detection and control.86

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