Risk Factors for Coronary Heart Disease in African Americans

The Atherosclerosis Risk in Communities Study, 1987-1997

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Background: As part of the Atherosclerosis Risk in Communities Study, the race-specific incidence rates and risk factor prediction for coronary heart disease (CHD) were determined for black and white persons over 7 to 10 years of follow-up, from 1987 to 1997.

Methods: The sample included 14,062 men and women (2298 black women, 5686 white women, 1396 black men, and 4682 white men) aged 45 to 64 years who were free of clinical CHD at baseline.

Results: Average age-adjusted incidence rates (95% confidence intervals) for CHD per 1000 person-years were as follows: black women, 4.9 (4.6-5.2); white women, 4.0 (3.5-4.6); black men, 10.6 (8.9-12.7); and white men, 12.5 (11.5-13.7). Incidence rates (95% confidence intervals) using a definition for CHD that excluded revascularization procedures were as follows: black women, 5.1 (4.2-6.2); white women, 4.0 (3.5-4.6); black men, 10.6 (8.9-12.7); and white men, 12.5 (11.5-13.7). Hazard rate ratios (95% confidence intervals) were as follows: black women, 1.8 (1.2-2.8); white women, 3.3 (2.4-4.6); black men, 1.6 (1.1-2.5); and white men, 2.0 (1.6-2.6). Low-density lipoprotein cholesterol level was similarly predictive in all race-sex groups (hazard rate ratio, 1.2-1.4 per SD increment of low-density lipoprotein cholesterol level). High-density lipoprotein cholesterol level seemed somewhat more protective in white than in black persons.

Conclusions: Findings from this study, along with clinical trial evidence showing efficacy, support aggressive management of traditional risk factors in black persons, as in white persons. Understanding the intriguing racial differences in risk factor prediction may be an important part of further elucidating the causes of CHD and may lead to better methods of preventing and treating CHD.

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The relationship between incidence of coronary heart disease (CHD) and standard risk factors, including low-density lipoprotein (LDL) cholesterol level, high-density lipoprotein (HDL) cholesterol level, blood pressure, smoking, and diabetes mellitus, is well established and is described in several populations, including participants of the Atherosclerosis Risk in Communities (ARIC) Study.1-4 However, race-specific CHD incidence data for black persons are sparse and, to our knowledge, have not yet been reported for participants of the ARIC Study.5,14

Race as a health factor is a complex subject. Controversy exists as to whether health issues should even be considered in terms of race, because race is defined with such difficulty and intertwined with socioeconomic status.13-17 Nevertheless, the disparity in health outcomes based on assessment by race is well documented.18-27 This analysis attempts to quantify the risk factor associations for CHD in black persons, with a comparison group of white persons in the same standardized study.

One of the unique features of the ARIC Study, a large population-based study of cardiovascular disease risk factors, is the inclusion of many black persons. The black participants included in the ARIC Study reside principally in Jackson, Miss, with a few in North Carolina. The white participants in the ARIC Study reside in North Carolina, Maryland, and Minnesota. The size of the ARIC Study population and the standardization of methods across centers provide strengths.28
STUDY POPULATION

The ARIC Study is a prospective study of risk factors for CHD and carotid atherosclerosis in 15,792 men and women in 4 communities in the United States. Participants aged 45 to 64 years were examined and followed up in Forsyth County, North Carolina; Jackson; suburban areas of Minneapolis, Minn; and Washington County, Maryland. Each study site selected a representative sample from its entire community population, with the exception of Jackson, where a representative sample only of black persons was selected. Race was self-reported. Communities in Maryland and Minnesota had fewer than 1% black persons; Forsyth County had 12% black persons. From 1987 to 1989, community residents were recruited and participated in an examination that included interviews; an electrocardiogram (ECG); pulmonary function tests; blood drawn for measurements of lipid levels, lipoprotein levels, hemostatic factors, and chemistries; and a noninvasive B-mode ultrasonographic measurement of the intimal-medial thickness (IMT) of the extracranial carotid arteries. Approximately 46% of eligible participants in Jackson and 66% in the other 3 communities were examined. Subsequent examinations took place from 1990 to 1992, from 1993 to 1995, and from 1996 to 1998.

STUDY VARIABLES

Each Field Center of the ARIC Study received the approval of its Institutional Review Board before initiation. At the baseline visit, the study was explained to each prospective participant, and each participant was then required to read and sign a detailed informed consent document.

Methods for blood collection and for centralized measurement of plasma total cholesterol, triglyceride, HDL cholesterol, calculated LDL cholesterol, and glucose levels have been reported elsewhere. Estimates of intrapersonal variability in blood measurements have been reported. Prevalent diabetes mellitus was defined as a fasting glucose level of 126 mg/dL or more (≥7.0 mmol/L), a nonfasting level of 200 mg/dL or more (≥11.1 mmol/L), a self-reported physician diagnosis of diabetes mellitus, or pharmacologic treatment for diabetes mellitus. Controlled diabetes mellitus was defined as a fasting glucose level of less than 130 mg/dL (<7.2 mmol/L) among those classified as diabetic.

Methods have been reported for ascertainment of body mass index (BMI) (calculated as weight in kilograms divided by the square of height in meters) and resting systolic blood pressure (SBP) and diastolic blood pressure (DBP). Weight was measured with the participant wearing a scrubsuit and no shoes. Obesity was defined as a BMI of 30 or higher, overweight as a BMI of 25 or higher and less than 30, and lean as a BMI of less than 25. Prevalent hypertension was defined as an SBP of 140 mm Hg or higher, a DBP of 90 mm Hg or higher, or self-reported use of antihypertensive medications in the past 2 weeks. Controlled hypertension was defined as an SBP lower than 140 mm Hg and a DBP lower than 90 mm Hg among those classified as hypertensive. Participants were defined as current smokers, ex-smokers, or never smokers, and pack-years of cigarettes smoked and current ethanol consumption (measured in grams per week) were estimated from interview. Educational level was defined as low for those who did not complete high school. Mid education was defined as a high school graduate and possibly some vocational school. High education was defined as at least some college. Procedures for determining ankle-brachial index and estimating ECG left ventricular hypertrophy (LVH) have been previously published. Peripheral arterial disease was defined as an ankle-brachial index of less than 0.85 for women and less than 0.90 for men. Prevalent CHD at baseline was defined, for exclusion, as a self-reported history of a physician-diagnosed heart attack, evidence of a prior myocardial infarction by ECG, or self-report of cardiovascular surgery or coronary angioplasty.

Bilateral B-mode ultrasonographic examinations of the carotid arteries were performed at 3 sites: the distal 10 mm of the common carotid artery, the bifurcation, and the proximal 10 mm of the internal carotid artery. Ultrasonographers were trained at the ARIC Study’s Ultrasound Reading Center and certified annually. Measurements were made from videotapes at the reading center without knowledge of any characteristics of the participants. The analysis uses the combined intima plus media thickness (IMT) of the extracranial carotid arteries. Approximately 46% of eligible participants in Jackson and 66% in the other 3 communities were examined. Subsequent examinations took place from 1990 to 1992, from 1993 to 1995, and from 1996 to 1998.

ASCERTAINMENT OF INCIDENT EVENTS

Coronary heart disease incident events were ascertained by contacting participants annually, identifying hospitalizations and deaths during the previous year, and surveying discharge lists from local hospitals and death certificates from State Vital Statistics Offices for potential cardiovascular events from 1987 through 1997.

Trained abstractors obtained hospital medical records, recorded presenting symptoms and cardiac enzyme levels, and photocopied up to 3 ECGs for each person. The ECGs were coded using Minnesota Code at the University of Minnesota. Out-of-hospital deaths were investigated by death certificates and, in most cases, by an interview with one or more next of kin and a questionnaire filled out by the patient’s physician. Coroners reports or autopsy reports, when available, were obtained. Details on quality assurance for ascertainment and classification of events have been published.

A CHD incident event was defined as a validated definite or probable hospitalized myocardial infarction, a definite CHD-related death, an unrecognized myocardial infarction detected by comparison of triennial ARIC Study examination ECG with baseline ECG, or a CHD-related revascularization (surgery, angioplasty, stenting, or any combination of these). The criteria for definite or probable hospitalized myocardial infarction were based on combinations of chest pain symptoms, ECG changes, and cardiac enzyme levels. The criteria for definite fatal CHD were based on chest pain symptoms, medical history, and underlying cause of death from the death certificate. A Morbidity and Mortality Classification Committee reviewed potential clinical events and determined the final diagnosis. Unrecognized incident myocardial infarction was determined from ECGs obtained at the 4 ARIC Study visits (a new major Q wave, a minor Q wave with ischemic ST-T changes, or a myocardial infarction by computerized NOVACODE criteria, confirmed by a side-by-side visual comparison of baseline and follow-up ECGs).

DATA ANALYSIS

From the 15,792 ARIC Study participants, we excluded the nonwhite persons in Minneapolis and Washington County and participants in Forsyth County who were neither black nor white (103 persons total). An additional 763 were excluded for prevalent CHD at baseline, 339 others for unknown status regarding prevalent CHD, and 525 for missing information on the risk factors.
factors of interest in this analysis. That left 14,062 individuals (2,298 black women, 5,686 white women, 1,396 black men, and 4,682 white men) for analysis.

For descriptive purposes, age-adjusted means or proportions (adjusted to the mean age of the entire sample, 54 years) of baseline risk factors were calculated by race, sex, and incident disease status by analysis of covariates methods for linear or logistic regression. Crude race- and sex-specific cumulative incident CHD rates were calculated as number of events divided by cumulative follow-up time, and age-adjusted rates and their 95% confidence intervals (CIs) were computed from Poisson regression by analysis of covariance methods overall and by risk factor level. For each risk factor considered, age-adjusted hazard rate ratios (HRRs) and their 95% CIs were computed by Cox proportional hazards regression, comparing putative higher risk levels with lowest risk level for categorical risk factors. Continuous risk factors were categorized by standard cut points or by overall population tertiles, or HRRs were presented for a 1-SD difference in risk factor level. Multivariable-adjusted HRRs were derived from Cox proportional hazards regression models including age, continuous LDL and HDL cholesterol levels, current and former smoking, hypertension, and diabetes mellitus, with or without educational level.

RESULTS

For the most part, participants who developed CHD had more adverse age-adjusted mean baseline risk factor levels than those who did not develop CHD in all race-sex groups. However, 95% CIs overlapped for cases and noncases for SBP and DBP for black men, for DBP for white women, for cigarette pack-years for black women and black men, and for BMI for all race-sex groups except white women (data available at: http://www.bios.unc.edu/cscc/ARIC).

Most categorical CHD risk factors and atherosclerosis markers also were more favorable in noncases than in incident cases. Former smoking status, midlevel education, controlled hypertension, and controlled diabetes mellitus generally were not significantly different in any race-sex groups, nor was ECG LVH in white women or men. The absolute difference between cases and noncases was especially large for hypertension in black women (data available at: http://www.bios.unc.edu/cscc/ARIC).

Table 1 shows age-adjusted 1987 to 1997 CHD incidence rates by race, sex, and risk level. Overall incidence rates were substantially higher in men than women for black and white persons. Sex differences in black persons were less than in white persons. Racial differences in overall incidence rates tended to be small when the standard ARIC Study CHD event definition was used. When revascularization procedures were excluded from the CHD event definition, rates were substantially lower in white persons, but changed little for black persons.

Coronary heart disease incidence rates were higher in hypertensive than nonhypertensive persons for all race-sex groups, but, interestingly, the incidence for nonhypertensive black persons was lower than for white persons. Incidence rates were significantly higher for elevated LDL cholesterol levels for white men and women but not for black men and women. Rates were higher for those with a low (vs high) HDL cholesterol level in all race-sex groups. Rates were significantly higher for diabetic than nondiabetic persons. Incidence rates were higher in current smokers than in never smokers for all race-sex groups. Incidence rates were higher in those with lower vs the highest educational level. Rates were substantially higher in black women with ECG LVH compared with those without ECG LVH. Rates were higher in all groups for those with a low vs a high ankle-brachial index or with a high vs a low carotid IMT.

Table 2 shows the age-adjusted HRRs for CHD considering each major risk factor one at a time. The relationship for hypertension was especially strong in black women, in part reflecting the low rate in the nonhypertensive black women (Table 1), but was statistically significant in all race-sex groups. Systolic blood pressure was significantly and positively associated with CHD in all groups except black men. Diastolic blood pressure was significant only in black women and white men. For LDL cholesterol level, the relation with CHD was strong, positive, and statistically significant for all groups. For HDL cholesterol level, the inverse relationship was statistically significant in all groups. Diabetes mellitus was a significant risk factor in all groups, especially white women. Current smoking was an especially strong predictor of CHD in women, and was statistically significant in all groups. Body mass index was weakly predictive of CHD in all but black men. Graduating from high school, compared with less education, was associated with a reduced CHD risk in all groups except black men. An educational level that included some college was associated with a lower CHD risk in all groups. Electrocardiographic LVH was an especially strong risk factor in black women, was statistically significant in black men, and was not significant for white men or women. Peripheral arterial disease, measured by ankle-brachial ratio of SBP (<0.85 for women and <0.90 for men), was a strong risk indicator in all race-sex groups. Carotid IMT was predictive in all groups, but marginally so in black men.

Table 3 shows multivariable-adjusted HRRs for CHD. All variables from Table 2 that remained statistically significantly associated with CHD for all race-sex groups in the multivariable model included hypertension, LDL cholesterol level, HDL cholesterol level, diabetes mellitus, and current smoking. Current smoking seemed to be a stronger predictor of CHD in women than in men. Hypertension was a particularly strong predictor in black women (HRR, 4.8; \( P = .02 \) for difference by race) than in other groups. Diabetes mellitus seemed to be a stronger predictor in white women (HRR, 3.3; \( P = .03 \) for difference by race) than in other groups. The HDL cholesterol level was a stronger predictor in white men than in black men (\( P = .04 \)).

When educational level was added to the model shown in Table 3, the associations of the other risk factors with CHD changed little. The associations for education variables, however, were weaker than those shown in Table 2. There still was an independent association of CHD with a high vs a low level of education for black men (HRR, 0.6; 95% CI, 0.4-1.0) and white women (HRR, 0.7; 95% CI, 0.4-1.0) and possibly for black women (HRR, 0.7; 95% CI, 0.4-1.1), but not for white men (HRR, 0.9; 95% CI, 0.7-1.1).

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This study has several key findings. By using the standard ARIC Study definition that includes coronary revascularization, the incidence rates of CHD were similar in black and white persons, although there were somewhat higher rates in black women than white women and slightly lower rates in black men than white men. When revascularization procedures were excluded, rates for black men and women changed little, but rates were substantially lower for white men and women. These data confirm lower use of revascularization procedures in black compared with white persons and suggest possible underestimation of incident CHD in black persons or overestimation in white persons when revascularization procedures are included in the definition of CHD.46-48

Traditional CHD risk factors in white populations were also associated with CHD in this population of black persons. However, black and white differences that have clinical significance in risk factor associations were noted. In particular, hypertension is a more powerful predictor in black women than in other race-sex groups. Diabetes mellitus was a weaker predictor in black women than in white women.

Reported racial/ethnic differences in CHD incidence are not entirely consistent.5-12 Most recent reports suggest similar incidence rates for black and white men and higher rates for black than for white women. Reports49,50 of CHD-
related mortality (as opposed to CHD incidence) in the ARIC Study and elsewhere often demonstrate higher rates for black than for white men. There are several reasons why black-white ratios may be higher for CHD-related mortality vs CHD incidence. One of the most important of these is the underestimation of true rates in black persons or the overestimation of true rates in white persons of incident cases because of lower procedure rates in black persons, as previously noted. Also, case fatality rates for CHD are higher in black than in white persons, contributing to higher mortality rates.46 Misclassification of cause of death may contribute to inflation of the numerator in incidence rates for black persons in some evaluations. Undercounting black men in the census may account for underestimation of the denominator for some populations, thereby inflating mortality rates.

In our study, and in others,51 hypertension was a particularly powerful risk factor for CHD in black persons, especially in women. High HRRs for hypertension in black women seem to be explained by a relatively low CHD rate in those without hypertension. The CHD incidence rate (95% CI) for black women without hypertension was 1.6 (1.0-2.8) per 1000 person-years vs 2.9 (2.4-3.5) for white women. The CHD incidence rate (95% CI) for black women with hypertension was 7.6 (6.2-9.5); and for white women with hypertension, 7.5 (6.1-9.2). If replicated, understanding why normotensive black women are at such low risk may lead to a better understanding of how to prevent CHD in others. Another potential explanation might be that black women with hypertension have a higher blood pressure or a longer duration of hypertension than comparative groups.

In this and some other studies,2,51 diabetes mellitus was a weaker predictor of CHD in black than in white persons. Higher HRRs for white women with diabetes mellitus seem to be accounted for by excess CHD events in this group compared with nondiabetic persons. Table 1 shows an age-adjusted CHD incidence rate (95% CI) of 15.7 (12.1-20.4) per 1000 person-years for white women with diabetes mellitus compared with 9.3 (6.7-12.9) for black women. Rates for nondiabetic persons were similar (3.2 and 4.0, respectively).

### Table 2. Age-Adjusted HRRs for CHD From 1–Risk Factor Cox Proportional Hazards Models, by Race and Sex*

<table>
<thead>
<tr>
<th>Risk Factors (Increment)</th>
<th>Women</th>
<th>White</th>
<th>Men</th>
<th>White</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypertension</td>
<td>5.3 (2.8-10.0)</td>
<td>2.7 (2.0-3.6)</td>
<td>2.0 (1.3-3.0)</td>
<td>1.8 (1.5-2.1)</td>
</tr>
<tr>
<td>Systolic BP (1 SD)</td>
<td>1.7 (1.5-1.9)</td>
<td>1.4 (1.2-1.6)</td>
<td>1.1 (1.0-1.3)</td>
<td>1.3 (1.2-1.5)</td>
</tr>
<tr>
<td>Diastolic BP (1 SD)</td>
<td>1.4 (1.2-1.7)</td>
<td>1.0 (0.8-1.2)</td>
<td>1.0 (0.8-1.2)</td>
<td>1.1 (1.0-1.2)</td>
</tr>
<tr>
<td>LDL cholesterol level (1 SD)</td>
<td>1.3 (1.1-1.6)</td>
<td>1.4 (1.3-1.6)</td>
<td>1.2 (1.0-1.4)</td>
<td>1.4 (1.2-1.5)</td>
</tr>
<tr>
<td>HDL cholesterol level (1 SD)</td>
<td>0.6 (0.5-0.8)</td>
<td>0.5 (0.5-0.6)</td>
<td>0.7 (0.6-0.9)</td>
<td>0.5 (0.5-0.6)</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>2.3 (1.5-3.5)</td>
<td>5.1 (3.7-6.9)</td>
<td>1.7 (1.1-2.6)</td>
<td>2.4 (1.9-3.0)</td>
</tr>
<tr>
<td>Current smoker vs never smoker</td>
<td>2.6 (1.7-4.0)</td>
<td>2.9 (2.2-4.0)</td>
<td>1.7 (1.1-2.6)</td>
<td>1.8 (1.4-2.3)</td>
</tr>
<tr>
<td>Former smoker vs never smoker</td>
<td>1.0 (0.5-1.9)</td>
<td>1.0 (0.6-1.6)</td>
<td>0.8 (0.5-1.3)</td>
<td>1.3 (1.0-1.8)</td>
</tr>
<tr>
<td>BMI (1 SD)</td>
<td>1.2 (1.0-1.4)</td>
<td>1.2 (1.1-1.4)</td>
<td>1.0 (0.8-1.2)</td>
<td>1.1 (1.0-1.2)</td>
</tr>
</tbody>
</table>

*HRR indicates hazard rate ratio; CHD, coronary heart disease; CI, confidence interval; BP, blood pressure; LDL, low-density lipoprotein; HDL, high-density lipoprotein; BMI, body mass index; ECG, electrocardiographic; LVH, left ventricular hypertrophy; ABI, ankle-brachial index; and IMT, intimal medial thickness.

### Table 3. Adjusted HRRs for CHD From Multivariable Cox Proportional Hazards Models for a Given Difference in Risk Factor Level, by Race and Sex*

<table>
<thead>
<tr>
<th>Risk Factors (Increment)</th>
<th>Women</th>
<th>White</th>
<th>Men</th>
<th>White</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypertension</td>
<td>4.8 (2.5-9.0)</td>
<td>2.1 (1.8-2.9)</td>
<td>2.0 (1.3-3.0)</td>
<td>1.6 (1.3-1.9)</td>
</tr>
<tr>
<td>LDL cholesterol level (1 SD)</td>
<td>1.3 (1.1-1.5)</td>
<td>1.3 (1.1-1.4)</td>
<td>1.2 (1.0-1.4)</td>
<td>1.4 (1.3-1.5)</td>
</tr>
<tr>
<td>HDL cholesterol level (1 SD)</td>
<td>0.8 (0.6-1.0)</td>
<td>0.6 (0.5-0.8)</td>
<td>0.8 (0.6-1.0)</td>
<td>0.6 (0.5-0.7)</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>1.8 (1.2-2.8)</td>
<td>3.3 (2.4-4.6)</td>
<td>1.6 (1.2-2.5)</td>
<td>2.0 (1.6-2.6)</td>
</tr>
<tr>
<td>Current smoker vs never smoker</td>
<td>2.6 (1.7-4.1)</td>
<td>3.0 (2.2-4.0)</td>
<td>1.9 (1.2-3.0)</td>
<td>1.7 (1.3-2.2)</td>
</tr>
<tr>
<td>Former smoker vs never smoker</td>
<td>1.0 (0.5-1.9)</td>
<td>1.2 (0.7-1.9)</td>
<td>0.8 (0.5-1.4)</td>
<td>1.3 (0.9-1.7)</td>
</tr>
</tbody>
</table>

*Adjusted for age and the other variables in the model (hypertension, LDL cholesterol level, HDL cholesterol level, diabetes mellitus, and smoking status). HRR indicates hazard rate ratio; CHD, coronary heart disease; CI, confidence interval; LDL, low-density lipoprotein; and HDL, high-density lipoprotein.
Higher risk coupled with a much higher prevalence of hypertension than in white persons causes the population-attributable risk of CHD for hypertension to be much higher in black persons (black women, 68.0%; and black men, 34.6%) than in white persons (white women, 21.6%; and white men, 13.4%). The higher prevalence of obesity in black women accounts for part of this high prevalence of hypertension, although BMI was not itself a strong risk factor for CHD in our age-adjusted analysis. As with hypertension, though, the much higher prevalence of diabetes mellitus in black than in white persons makes the population-attributable risk for diabetes mellitus comparable for black persons (black women, 13.6%; and black men, 9.1%) and white persons (white women, 14.2%; and white men, 8.0%), despite the lower HRR associated with diabetes mellitus in black persons.

Another potential explanation for different HRRs for diabetes mellitus and hypertension between black and white persons is the rate of control of these risk factors to standard values, as noted in guidelines. Control rates for diabetes mellitus were poor in all groups, but more so in black men and women (data available at: http://www.bios.unc.edu/cscce/ARIC). Yet, the patterns of control across race-sex groups are not consistent with the CHD HRRs related to diabetes mellitus and hypertension, suggesting that poorer control rates do not account for racial differences in HRRs.

Plasma lipid levels were associated with carotid IMT more strongly in white than in black persons in the ARIC Study cohort. However, in this analysis using incident CHD as the outcome, LDL cholesterol level was almost as strong a predictor in black as in white persons. High-density lipoprotein cholesterol level seemed less predictive in black than in white persons.

Socioeconomic status is inversely associated with CHD and often remains predictive independent of other CHD risk factors. Some of the black-white difference in health problems is attenuated by adjustment for socioeconomic status. Yet, it has been argued that it is impossible to completely adjust for socioeconomic status using traditional measures. In the multivariable model of risk, educational level was an independent predictor of CHD in white women and black men, and its inclusion in regression models had almost no impact on the predictability of other risk factors.

There are several limitations of this study and data analysis. Relatively low response rates, especially in Jackson, suggest that incidence rates may be overestimated or underestimated. Those with lower educational levels were less likely to participate and more likely to have a higher CHD risk. This most likely would underestimate incidence in black compared with white persons in this cohort. Also, in a population with a high disease burden occurring relatively early in life, such as in the Jackson cohort, confounding by comorbidity is challenging. Coronary heart disease incidence certainly is impacted by early deaths from other illnesses, including other cardiovascular diseases such as stroke. Another limitation of this analysis is the challenge of generalizability of the Jackson cohort to black persons living outside Jackson.

Other limitations include those often associated with large, prospective, observational studies, although unavailability for follow-up was minimal in this study. Although many participants did not participate in clinic examinations 2 to 4, fewer than 4% have been unavailable for surveillance follow-up during the period of this study, and all risk factor variables used for analysis were based on baseline examination data. Despite great effort to minimize measurement errors, in large multicenter studies, this possibility is always a limitation. The relatively small sample size and the small number of events in black men is a recognized limitation. Expansion of this cohort and continued surveillance through the Jackson Heart Study will aid in future analysis.

To our knowledge, this study provides the largest cohort of black persons in a prospective population study with carefully verified events. The Charleston Heart Study and the Evans County Heart Study have longer follow-up periods reported, but are restricted to fatal events and based on smaller cohorts of black persons from an earlier generation. Other studies have reported CHD-related mortality or unvalidated CHD incidence.

Racial differences noted in the ARIC Study must be interpreted with caution, because almost all of the black persons were in one geographic location—Jackson, Mississippi’s rates of CHD and stroke are among the highest in the nation in all race-sex groups. Most of the major findings in this report, however, confirm findings for black persons in other locations. The smaller sample size of black men and the limited number of events present a problem of power in demonstrating associations. In some instances, the failure to show a relationship for a given risk factor may be due to limited power. Expansion of the Jackson cohort and a longer follow-up period should help confirm or expand these initial findings.

In summary, CHD incidence in the ARIC Study was similar in black and white men and marginally higher in black women than in white women using standard definitions. Excluding revascularization procedures from the definition of CHD events revealed higher rates in black than in white persons, for men and women. The traditional risk factors were associated with CHD incidence in black persons, as in white persons. Hypertension seemed to be a more potent predictor, and diabetes mellitus less predictive, in black than in white persons.

Findings from this study, along with clinical trial evidence showing efficacy, support aggressive management of traditional risk factors in black persons, as in white persons. Particularly, the study supports aggressive management of hypertension in black women. Understanding the intriguing racial differences in risk factor prediction may be an important part of further elucidating the causes of CHD and may lead to better methods of preventing and treating CHD.

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