Comparison of Risk Factors for Cardiovascular Mortality in Black and White Adults

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Background: Recent attention to racial and ethnic disparities in health outcomes highlights the excess coronary heart disease mortality in black patients compared with white patients. We investigated whether traditional cardiovascular disease (CVD) risk factors were similarly associated with CVD mortality in black and white men and women.

Methods: Participants included 3741 black and 33 246 white men and women (44%) without a history of myocardial infarction, aged 18 to 64 years at baseline (1967-1973) from the Chicago Heart Association Detection Project in Industry study. Blood pressure, total cholesterol level, body mass index, cigarette smoking, and physician-diagnosed diabetes were assessed at baseline using standard methods.

Results: Through 2002, there were 107, 1586, 177, and 2866 deaths from CVD in black women, white women, black men, and white men, respectively. In general, the magnitude and direction of associations between traditional risk factors and CVD mortality were similar by race. However, in black women the multivariable-adjusted hazard ratio (HR) per 12 mm Hg of diastolic blood pressure was 1.08 (95% confidence interval [CI], 0.90-1.29), whereas it was 1.31 in white women (95% CI, 1.25-1.38). There was no association between higher cholesterol level (per 40 mg/dL [1.04 mmol/L]) and CVD mortality in black men (HR, 0.94; 95% CI, 0.80-1.10), whereas the risk was elevated in white men (HR, 1.21; 95% CI, 1.16-1.26).

Conclusions: Most traditional risk factors demonstrated similar associations with mortality in black and white adults of the same sex. Small differences were primarily in the strength, not the direction, of association.

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compared with white Americans prompted us to reevaluate the question of whether traditional risk factors are similarly associated with CVD mortality in black and white adults. We are uniquely able to test this question in the largest sample to date of employed black and white men and women residing in the urban North who were followed for more than 30 years. Using data from the Chicago Heart Association Detection Project in Industry (CHA) study, we tested the hypothesis that traditional risk factors for CVD are similarly associated with mortality from all CVDs (which includes all circulatory diseases) and ischemic CHD in black and white men and women.

METHODS

Methods of the CHA study have been previously described. Briefly, all employees 18 years and older at 84 Chicago-area companies and organizations (approximately 75,000) were invited to undergo screening for heart disease risk factors from 1967 through 1973. A total of 39,523 black and white men and women were screened. The overall response rate was 53%; no information was available on separate response rates for black and white participants. The study received institutional review board approval at periodic reviews, and participants provided informed consent. For this analysis, we excluded participants for the following reasons: lost to follow-up (n = 82), prevalent myocardial infarction at baseline (n = 473), and race other than black or white or age younger than 18 years or older than 64 years at baseline (n = 1981). Following exclusions, 36,987 participants remained: 2299 black women, 13,822 white women, 1442 black men, and 19,424 white men.

Screening was performed by 2 trained and standardized 4-person field teams. At baseline, age, sex, race/ethnicity, years of education, smoking status and number of cigarettes per day, medical history, and current use of medications to control hypertension, cholesterol level, or diabetes was queried. Height and weight were measured; body mass index (BMI) was calculated as weight in kilograms divided by the square of height in meters. Blood pressure (BP) was obtained via a single casual supine measurement using a mercury sphygmomanometer. Pulse pressure was calculated as the difference between systolic and diastolic BP. Analyses of BP were restricted to adults not using antihypertensive medications. Standardized high-quality methods were used for determination of total serum cholesterol level. Diabetes was determined from self-reported previous physician diagnosis and reported use of diabetes medications.

Vital status was ascertained through 2002, with a mean follow-up of 32 years. Before 1979, several methods were used including direct mail, telephone, contact with employers, and matching of records with Social Security Administration files. From 1979 through 1994, vital status was determined from the National Death Index (NDI), with multiple causes of death coded from death certificates by trained research staff according to the International Classification of Diseases, Eighth Revision (ICD-8). Coding decisions were cross-checked by study team members; all coders were blinded to baseline data. For deaths occurring between 1995 and 1998 and 1999 and 2002, NDI Plus service was used to obtain International Classification of Diseases, Ninth Revision (ICD-9) (1995-1998) and International Statistical Classification of Diseases, 10th Revision (ICD-10) (1999-2002) multiple cause of death coding. For this report, the underlying cause of death was used. Mortality from CHD (ie, ischemic heart disease) was defined as ICD-8 and ICD-9 codes 410.0 to 414.9 and ICD-10 codes, 120.x to 125.x; CVD mortality (ie, all circulatory diseases) was defined as ICD-8 and ICD-9 codes 390.0 to 458.9 and ICD-10 codes, 100.0 to 199.9.

On average, black participants were nearly 8 years younger than white participants at baseline (mean age, 33.1 vs 41.0 years). Means or proportions of each risk factor except current smoking were lower among black women, who were an average of 11 years younger than white women (Table 1). Black and white men were closer in age (36 vs 40 years, respectively), and levels of the risk factors were similar with the exception of smoking, which was higher in black men. Following age-adjustment, the distribution of characteristics was similar among black and white women. However, age-adjusted mean systolic and diastolic BPs were higher among black men (141.1/83.7 mm Hg) compared with white men (138.7/81.2 mm Hg).

Follow-up time and mortality rates are presented in Table 2. While crude mortality rates were lower in blacks than in whites, age-standardized rates of CVD and CHD mortality were similar between black and white women. Age-standardized CVD mortality rates were higher in black men, but CHD mortality rates were similar.

The distribution of baseline risk factors was calculated by race. Age was standardized to the population age distribution using the direct method, and mortality rates were calculated per 10,000 person-years. We tested and confirmed the proportional hazards assumption using log-log survival plots. We categorized baseline age into 6 groups with roughly equal numbers of persons (18-29 years, 30-36 years, 37-43 years, 44-50 years, 51-57 years, and 58-64 years) and calculated age-specific hazard ratios using Cox proportional hazards regression. We selected this technique, which allows a separate baseline hazard in each stratum but constrains the effect of the main exposure and covariates to be the same for all levels of the stratification variable, rather than age adjustment because of the marked difference in the age range of the black and white adults.

First, we fit models pooled by race (adjusted for education) that included an interaction term between race and the given risk factor. Hazard ratios (HRs) and 95% confidence intervals (CIs) were calculated for each risk factor per standard deviation using the multiplicative interaction term and lower-order terms. Next, we included all noncollinear variables (separate models were fitted for systolic and diastolic BP) and interaction terms between race and each risk factor to calculate race-specific HRs. Because of the potential for spurious associations due to the smaller sample size, fewer end points, and age differences in blacks compared with whites, we applied more stringent criteria for acknowledging heterogeneity in the association. Our criteria were as follows: (1) statistical significance of the interaction term was determined using a Bonferroni correction for 56 statistical tests resulting in a critical value of P < .001 and (2) evidence of marked differences in the magnitude and direction of effect by race. In our final analysis, we built multivariable models (separately by sex) that included all noncollinear traditional risk factors (ie, age, education, systolic BP, cholesterol level, BMI, smoking status, and diabetes) and a term for race to evaluate whether there was an excess relative risk in black participants compared with white participants. Analyses were conducted using Statistical Analysis Software version 9 (SAS Institute, Cary, NC).

All statistical analyses were stratified by sex. The distribution of baseline risk factors was calculated by race. Age was standardized to the population age distribution using the direct method, and mortality rates were calculated per 10,000 person-years. We tested and confirmed the proportional hazards assumption using log-log survival plots. We categorized baseline age into 6 groups with roughly equal numbers of persons (18-29 years, 30-36 years, 37-43 years, 44-50 years, 51-57 years, and 58-64 years) and calculated age-specific hazard ratios using Cox proportional hazards regression. We selected this technique, which allows a separate baseline hazard in each stratum but constrains the effect of the main exposure and covariates to be the same for all levels of the stratification variable, rather than age adjustment because of the marked difference in the age range of the black and white adults.
stantly increased risk of CVD mortality in black women, whereas it was associated with a significantly increased risk in white women. However, the \( P \) value for interaction (\( P = .02 \)) did not achieve statistical significance at our Bonferroni-corrected level of statistical significance (\( P / H = .001 \)). This difference was not present for CHD mortality, for which the HRs for black and white women were similar, and all CIs overlapped to include point estimates from the other strata.

Among men, there were some differences by race in the association of cholesterol level and current smoking with cardiovascular mortality (Table 3). Among black men, neither higher cholesterol level nor smoking status (current and former) were associated with CVD mortality, and smoking status was not associated with CHD mortality, but they were associated with CVD and CHD mortality among white men. For CVD mortality, neither the interaction value for cholesterol level nor current smoking met the corrected level of statistical significance (cholesterol level, \( P = .002 \); smoking, \( P = .03 \)). Similarly, neither cholesterol level nor smoking status met the corrected level of significance in relation to CHD mortality (cholesterol level, \( P = .01 \); former smoking, \( P = .03 \); and current smoking, \( P = .003 \)). In secondary analyses, we investigated the association of pulse pressure with mortality in men and women and found no differences by race group in either sex.

In multivariable models that included all risk factors (Table 4), the findings among women were the same as those in minimally adjusted models. Among men, race differences were more pronounced for cholesterol level (interaction, \( P = .002 \) for CVD mortality) and current smoking (interaction, \( P = .003 \) for CHD mortality) but again did not achieve corrected levels of statistical significance. While many factors among black men did not achieve statistical significance in relation to CHD mortality, there was no strong evidence of heterogeneity of effect. Again, the associations between pulse pressure and mortality were similar by race across sex.

To further explore the heterogeneity by race for smoking status among men, we tested whether black men were more likely than white men to have died from cancer at...
young ages, another major smoking-related cause of death. There was no evidence that black men died from cancer more often compared with white men. We found excess mortality at younger ages from injury-related causes of death (eg, motor vehicle crashes and homicides) in black men compared with white men.

Finally, the combination of traditional risk factors was included in a single model to identify whether relative

Table 3. Adjusted* Hazard Ratios (95% Confidence Intervals) for Mortality From CVD and CHD by Sex and Race†

<table>
<thead>
<tr>
<th>Risk Factor (Per Standard Deviation)</th>
<th>Women</th>
<th>Black</th>
<th>White</th>
<th>Men</th>
<th>Black</th>
<th>White</th>
</tr>
</thead>
<tbody>
<tr>
<td>Systolic BP (19 mm Hg)</td>
<td>1.15</td>
<td>1.25</td>
<td>(0.99-1.34)</td>
<td>1.11-1.40)</td>
<td>1.34</td>
<td>(1.29-1.38)</td>
</tr>
<tr>
<td>Diastolic BP (12 mm Hg)</td>
<td>1.09</td>
<td>1.28</td>
<td>(0.93-1.29)</td>
<td>1.14-1.42)</td>
<td>1.34</td>
<td>(1.29-1.39)</td>
</tr>
<tr>
<td>Total cholesterol (40 mg/dL [1.04 mmol/L])</td>
<td>1.05</td>
<td>0.97</td>
<td>(0.88-1.26)</td>
<td>1.10-1.20)</td>
<td>1.25</td>
<td>(1.21-1.30)</td>
</tr>
<tr>
<td>BMI (4)</td>
<td>1.15</td>
<td>1.30</td>
<td>(0.99-1.34)</td>
<td>1.16-1.26)</td>
<td>1.23</td>
<td>(1.18-1.28)</td>
</tr>
<tr>
<td>Former smoking‡</td>
<td>1.33</td>
<td>0.96</td>
<td>(0.70-2.54)</td>
<td>0.89-1.21)</td>
<td>1.26</td>
<td>(1.14-1.40)</td>
</tr>
<tr>
<td>Current smoking‡</td>
<td>2.31</td>
<td>1.20</td>
<td>(1.30-3.54)</td>
<td>1.66-2.06)</td>
<td>1.86</td>
<td>(1.69-2.06)</td>
</tr>
</tbody>
</table>

Table 4. Multivariable Risk Factor Adjusted* Hazards Ratios (95% Confidence Intervals) for Mortality From CVD and CHD by Sex and Race†

<table>
<thead>
<tr>
<th>Risk Factor (Per Standard Deviation)</th>
<th>Women</th>
<th>Black</th>
<th>White</th>
<th>Men</th>
<th>Black</th>
<th>White</th>
</tr>
</thead>
<tbody>
<tr>
<td>Systolic BP (19 mm Hg)</td>
<td>1.15</td>
<td>1.19</td>
<td>(0.99-1.34)</td>
<td>1.01-1.39)</td>
<td>1.35</td>
<td>(1.30-1.41)</td>
</tr>
<tr>
<td>Diastolic BP (12 mm Hg)</td>
<td>1.18</td>
<td>1.17</td>
<td>(0.94-1.48)</td>
<td>1.18-1.46)</td>
<td>1.38</td>
<td>(1.32-1.44)</td>
</tr>
<tr>
<td>Total cholesterol (40 mg/dL [1.04 mmol/L])</td>
<td>1.09</td>
<td>1.02</td>
<td>(0.85-1.41)</td>
<td>1.12-1.26)</td>
<td>1.33</td>
<td>(1.27-1.38)</td>
</tr>
<tr>
<td>BMI (4)</td>
<td>1.20</td>
<td>1.23</td>
<td>(0.97-1.47)</td>
<td>1.18-1.32)</td>
<td>1.24</td>
<td>(1.18-1.30)</td>
</tr>
<tr>
<td>Former smoking‡</td>
<td>1.15</td>
<td>0.65</td>
<td>(0.45-2.91)</td>
<td>0.93-1.39)</td>
<td>1.25</td>
<td>(1.01-1.49)</td>
</tr>
<tr>
<td>Current smoking‡</td>
<td>2.16</td>
<td>0.92</td>
<td>(1.19-3.91)</td>
<td>1.80-2.39)</td>
<td>1.94</td>
<td>(1.72-1.99)</td>
</tr>
<tr>
<td>Diabetes§</td>
<td>3.37</td>
<td>1.76</td>
<td>(1.21-3.5-9.0)</td>
<td>1.65-3.09)</td>
<td>2.08</td>
<td>(1.78-2.42)</td>
</tr>
</tbody>
</table>

Abbreviations: BMI, body mass index (calculated as weight in kilograms divided by the square of height in meters); BP, blood pressure; CHD, coronary heart disease; CVD, cardiovascular disease.

*Age-stratified Cox proportional hazards modeling, adjusted additionally for education. Age was stratified into 6 groups: 18-29 years, 30-36 years, 37-43 years, 44-50 years, 51-57 years, and 58-64 years.
†Data are given as hazard ratio (95% confidence interval). The statistical significance of the interaction terms was assessed at a Bonferroni-corrected level of P < .001.
‡Compared with the never smoking group.
§Compared with the nondiabetes group.

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In this population-based longitudinal study, we observed largely similar patterns in the associations between traditional risk factors and mortality from CVDs in black and white men and women. For the first time, we demonstrated these patterns in relation to ischemic CHD mortality as well as total CVD. This contemporary investigation of risk factors measured between 1967 and 1973 in relation to mortality from CHD and CVD through 2002, provides evidence that differences in the association between risk factors and CVD mortality are not a likely explanation for the excess mortality experienced by black adults compared with white adults. Furthermore, we demonstrated that there was little excess mortality for blacks compared with whites when the combination of traditional risk factors was included in a single model.

Despite the preponderance of similarities, there were a few notable exceptions in the strength of associations between traditional risk factors and CVD mortality between blacks and whites. Black men and women develop hypertension at a higher rate compared with their white counterparts, even in the absence of some traditional risk factors such as overweight. There is little evidence in our study that the association between BP and CVD mortality differs between blacks and whites. One exception is that among black women, a higher diastolic BP did not confer an elevated risk for CVD mortality, whereas it did for white women. Our finding is not directly comparable with previous studies because most other studies evaluated only systolic BP in relation to CHD and not CVD mortality. In the absence of a biologically plausible explanation for diastolic, but not systolic, BP to be weakly associated with CVD mortality in black women, it is possible that other factors, including systolic BP, play a relatively larger role in CVD mortality. These results suggest that the elevated BP in blacks compared with whites may be a significant contributor to the higher rates of CVD in blacks.

In the present study, there was no association between serum cholesterol level and CHD or CVD mortality for black men. This finding was also reported in the Charleston Heart Study men and in a pooled analysis of the men in the Evans County study and Charleston Heart Study. However, in the first and second National Health and Nutrition Examination Surveys (NHANES) higher total cholesterol level was associated with an elevated risk of CHD mortality in all men and in the Atherosclerosis Risk in Communities study, the HRs were comparable by race when higher cholesterol level cut points were selected. Because the HRs reported for black men do not achieve statistical significance in a direction that would indicate a protective effect of cholesterol, we do not interpret our null association in black men compared with the positive association in white men as evidence of a clinically important difference in effect. Differences between our findings and previous studies may be attributable to dose response vs categorical analyses, and the varying cholesterol level cut points selected for categorical analyses.

Smoking was a weaker risk factor for CVD mortality in black men compared with other race-sex groups, which is consistent with reports in NHANES cohorts but was not found in other studies. Differences in biological factors are an unlikely explanation for our findings; rather, smoking behaviors or competing mortality may explain our findings. Black men and women who reported smoking cigarettes in this study smoked fewer cigarettes per day, and there is a known positive association between amount of smoking and CVD mortality risk. In a secondary analysis, we confirmed a positive association between the number of cigarettes smoked per day and CVD mortality among current smokers that was present across race and sex groups. Furthermore, as indicated by our secondary analyses investigating the associations between smoking and cancer in black and white men and the association between race and other causes of death, it is possible that black men in this study were more likely to have died from injury-related causes of death at younger ages, which precluded their risk of dying from CVDs at older ages. This association is likely independent of smoking status and other risk factors studied in this report.

Because of the importance of body mass in the development of all of the traditional CVD risk factors, BMI has not traditionally been included in CVD risk prediction models. We confirm the importance of higher BMI as a risk factor for long-term CVD mortality across race and sex groups in models that adjust for age and education. Among men and white women, there was a small residual effect of BMI when all other CVD risk factors are taken into account. However, among black women the association between BMI and CVD mortality attenuates to marginal significance in multivariable models. This may demonstrate, as previously suggested, that higher BMI is less important as a risk factor for mortality among black women compared with white women or that higher BMI is associated with increased risk through its impact on the development of other CVD risk factors. If it is the latter, the marginal significance we observed in multivariable models may represent an overadjustment.

STRENGTHS AND LIMITATIONS

To our knowledge, this is the first black-white comparison study conducted exclusively in an employed population. By studying an employed cohort, we were able to better account for socioeconomic differences between race groups compared with previous studies. However, the potential for residual confounding remains because most blacks in 1967 through 1973 were employed in lower status occupations, at lower salaries, compared with whites. Because occupational status was not available in this co-
hence, we could not confirm this hypothesis. Instead, we adjusted for education, which, unlike income or occupation, is reasonably stable across the life course in adults and applies equally to men and women.20

By conducting this study in 1 location (Chicago, Ill), we limit the potential for confounding by geography where access to health care resources, including physician offices and exercise facilities, likely differs by region and in urban vs rural communities. With the exception of the NHANES, most previous research has been conducted in the samples of blacks from the southern United States.9,11,12 Prior to this study, the substantial proportion of the US black population that lives in northern cities has been understudied. Unfortunately, however, we were unable to generate response rates to determine whether the black participants in this study were representative of the eligible population.

Black participants in this study were younger than whites owing to the demographic distribution of employed black adults in the Chicago area during the late 1960s and early 1970s.16 Consequently, comparisons across the full age range in this study (18-64 years) may result in underestimates of risk in black adults. Comparable CHD mortality rates in black and white women, when rates have been higher for black women over many decades,3 may be one reflection of this underestimate. Stratified modeling by age may not fully account for these differences, and residual confounding by age may remain. In secondary analyses not shown herein, we tested our hypotheses in the subset of adults aged 40 to 59 years and found patterns that were similar to those in the full cohort. We feel comfortable that our results are not unduly biased but urge caution in interpreting these findings.

We were unable to account for changes in risk factors over time or duration of risk factor exposure in this cohort because we only had a single risk factor measurement available that was taken up to 30 years prior to the outcome. Despite secular changes in the prevalence and treatment for risk factors, a single risk factor measurement is predictive of CVD risk in this1,21 and other22 prospective cohort studies and therefore cannot be discounted as an important measure of risk. Measures of physical activity, other health behaviors, or psychological states that may have modified these risk factors or been directly related to mortality were not available for study. Using death certificates to identify CHD mortality potentially overestimates CHD mortality,23 particularly among blacks compared with whites.24 Misclassification is considerably greater in adults older than 85 years at the time of death, and research suggests that misclassification is greater in some racial groups (ie, American Indians and Alaskan Natives).24,25

The magnitude of many associations between risk factors and CVD mortality was smaller among black participants. However, few of these differences achieved statistical significance, particularly once our P value for statistical significance was corrected for multiple comparisons. Rather than interpret these as differences in biological factors, it is plausible that small numbers of events or competing mortality from other causes has affected the strength of associations. Life expectancy in US blacks is lower than that of whites, so instead of living to the age when CVD becomes the leading cause of death, many black people died earlier as the result of violence or unintentional injuries. In addition, if death due to other causes that are also related to CVD risk factors (eg, renal disease) occurred more frequently among black adults, the likelihood of dying from CVDs is smaller and the strength of association with risk factors may have appeared weaker.

**IMPLICATIONS**

More than a third of the difference in life expectancy between black adults and white adults is attributable to CVDs.26 Despite differences in the prevalence and distribution of risk factors measured during the baseline examination of this cohort up to 30 years earlier compared with a contemporary population, this investigation provides evidence that the association between traditional risk factors and CVD mortality is generally similar in black and white men and women. Given the current large racial disparity in the prevalence of risk factors in the US population, namely higher rates of obesity, hypertension, and diabetes in black adults compared with white adults, it is important that health care professionals closely monitor and work to control the traditional CVD risk factors in all adults. Additional research is needed to explore differences in social, environmental, or biological factors that might account for disparities in CVD morbidity and mortality in black and white adults.

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Author Contributions: Dr Carnethon had full access to all the data in the study and takes responsibility for the integrity of the data and the accuracy of the data analysis.

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