Epidemiology of Incident Heart Failure in a Contemporary Elderly Cohort

The Health, Aging, and Body Composition Study

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Background: The race- and sex-specific epidemiology of incident heart failure (HF) among a contemporary elderly cohort are not well described.

Methods: We studied 2934 participants without HF enrolled in the Health, Aging, and Body Composition Study (mean [SD] age, 73.6 [2.9] years; 47.9% men; 58.6% white; and 41.4% black) and assessed the incidence of HF, population-attributable risk (PAR) of independent risk factors for HF, and outcomes of incident HF.

Results: During a median follow-up of 7.1 years, 258 participants (8.8%) developed HF (13.6 cases per 1000 person-years; 95% confidence interval, 12.1-15.4). Men and black participants were more likely to develop HF. No significant sex-based differences were observed in risk factors. Coronary heart disease (PAR, 23.9% for white participants and 29.5% for black participants) and uncontrolled blood pressure (PAR, 21.3% for white participants and 30.1% for black participants) carried the highest PAR in both races. Among black participants, 6 of 8 risk factors assessed (smoking, increased heart rate, coronary heart disease, left ventricular hypertrophy, uncontrolled blood pressure, and reduced glomerular filtration rate) had more than 5% higher PAR compared with that among white participants, leading to a higher overall proportion of HF attributable to modifiable risk factors in black participants vs white participants (67.8% vs 48.9%). Participants who developed HF had higher annual mortality (18.0% vs 2.7%). No racial difference in survival after HF was noted; however, rehospitalization rates were higher among black participants (62.1 vs 30.3 hospitalizations per 100 person-years, P < .001).

Conclusions: Incident HF is common in older persons; a large proportion of HF risk is attributed to modifiable risk factors. Racial differences in risk factors for HF and in hospitalization rates after HF need to be considered in prevention and treatment efforts.

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Heart failure (HF) is primarily a disease of older persons, with an annual incidence of 10 cases per 1000 after age 65 years, which doubles every decade thereafter. Subjects older than 65 years represent more than 75% of prevalent HF cases in the United States. In a 2004 European study, participants older than 70 years accounted for 88% of new HF cases. Although some recent data suggest relative improvement in survival after development of HF, other evidence challenges this finding, especially in older persons. Nevertheless, the absolute survival rate for these patients remains poor, and the actual number of HF deaths has increased by 20.5% during the last decade, reflecting the increasing prevalence of HF and the aging of the population. In patients older than 67 years, the median survival is generally less than 3 years after hospitalization for HF. The annual hospitalization rate for these patients now exceeds 1 million in the United States, 80% of patients hospitalized with HF are older than 65 years, and readmission rates as high as 50% within 6 months of discharge have been reported.

Risk factors and outcomes for HF in the general population have been well described. However, many of these data are old and were derived primarily from white subjects among young or middle-aged populations. The risk factor profile for cardiovascular diseases is changing with increasing prevalence of obesity, metabolic syndrome, and diabetes mellitus. Similarly, the therapeutic profile for risk factors such as hypertension or coronary heart disease (CHD) has evolved over time with increased use of angiotensin-converting enzyme inhibitors and statins. This may affect...
the development of HF and the outcomes thereafter. Recent evidence suggests substantial differences in disease development and progression among sex- and race-based subgroups.

Understanding and quantifying these differences is imperative for planning appropriate preventive and therapeutic interventions. To our knowledge, the sex- and race-related risk factor profile and population-attributable risk (PAR) for HF risk factors in contemporary older persons are unknown. In this study, we sought to assess the epidemiology of incident HF among older persons enrolled in the Health, Aging, and Body Composition (Health ABC) Study.

METHODS

STUDY POPULATION AND OUTCOMES

The Health ABC Study is a population-based study of 3075 community-dwelling men and women aged 70 to 79 years at enrollment. To be eligible, participants had to report no difficulty in walking one-quarter mile or climbing 10 stairs without resting. Participants were identified from a random sample of white Medicare beneficiaries and all age-eligible black community residents in designated zip code areas surrounding Pittsburgh, Pennsylvania, and Memphis, Tennessee. Exclusion criteria included difficulties with activities of daily living, obvious cognitive impairment, inability to communicate with the interviewer, intention of moving within 3 years, or participation in a trial involving a lifestyle intervention. All participants provided written informed consent, and the institutional review boards at both study sites approved the study protocol.

Baseline data were collected from April 1997 to June 1998. Cardiovascular disease status at baseline, including prevalent HF, was based on self-reported history, use of selected drugs, and International Classification of Diseases, Ninth Revision, Clinical Modification codes as reported by Medicare and Medicaid Services from 1995 through 1998. All participants were asked to report any hospitalizations, and every 6 months they were asked direct questions to elicit information about interim cardiovascular events. All admissions with an overnight stay were evaluated for cardiovascular events by reviewing the medical records at each site. All hospitalizations and deaths were reviewed by the Health ABC Study diagnosis and disease ascertainment committee, and underlying causes of death were determined by central adjudication.

Of 3075 subjects enrolled in the Health ABC Study, 95 had definite or possible HF at baseline, and 46 were excluded because of missing data on HF status. The final cohort analyzed for this study included 2934 participants.

STUDY DEFINITIONS

Incident HF

All first admissions with an overnight stay confirmed to be related to HF were classified as incident HF. Local adjudicators classified events as HF based on symptoms, signs, chest radiograph results, and echocardiographic findings based on criteria similar to those used in the Cardiovascular Health Study. The HF criteria required at least a HF diagnosis from a physician and treatment for HF (ie, a current prescription for a diuretic agent and either digitalis or a vasodilator); these criteria have been used in previous investigations. Because HF was not allowed as a cause of death, there were no deaths from incident HF.

Risk Factor Definitions

History of coronary revascularization, electrocardiographic evidence of myocardial infarction, or self-reported history of myocardial infarction or angina accompanied by antianginal medication use was considered definite evidence of CHD; self-reported history of CHD without medication use was considered probable CHD. Hypertension was classified as definite if there were both a history of hypertension or physician diagnosis and use of antihypertensive medications; history or medication use alone was classified as probable hypertension. Diabetes mellitus was considered present if the participant reported a history of diabetes or use of antidiabetic medications. Smoking was classified as current, past (if ≥ 100 lifetime cigarettes), or never. Cerebrovascular disease was based on a history of stroke, transient ischemic attack, or carotid intervention. Depression was defined as self-reported depression accompanied by medication use. Left ventricular hypertrophy (LVH) was determined from the baseline electrocardiogram using the following criteria: R wave exceeding 12 mm in lead aVL; R wave exceeding 26 mm in lead V1 or V6; R wave exceeding 20 mm in any of leads I, II, III, and aVF; or R wave in lead V1 exceeding 35 mm.

Risk Factors for Incident HF

Independent predictors of incident HF in the Health ABC Study were previously reported. The following 9 variables were independently associated with HF development: age, LVH, history of smoking and CHD, systolic blood pressure and heart rate, and serum levels of glucose, albumin, and creatinine; sex and race were considered, but neither was independently associated with incident HF. For the PAR calculation, continuous predictors were dichotomized using clinically relevant cutoff points. Systolic blood pressure was dichotomized as controlled vs uncontrolled at 140 mm Hg, fasting glucose level at 125 mg/dL (to convert glucose level to millimoles per liter, multiply by 0.0555), resting heart rate at 75 beats/min, and albumin level at 3.8 g/dL (to convert albumin level to grams per liter, multiply by 10). Creatinine level was converted to glomerular filtration rate (GFR) using the Modification of Diet in Renal Disease formula, and a cutoff of 60 mL/min/1.73 m² was used to define impaired GFR. Smoking (current yes or no) and CHD status (definite yes or no) were collapsed into binary predictors. We classified independent risk factors into the following 2 groups: modifiable (CHD, LVH, smoking, glucose level, and systolic blood pressure) and potentially modifiable (heart rate, albumin level, and renal function). Age was included in regression models for multivariable PAR calculation, but the PAR of age was omitted from the tables because age cannot be modified.

STATISTICAL ANALYSIS

We compared continuous variables using the Mann-Whitney rank sum test and categorical variables using the Fisher exact test. Cumulative event rates were obtained using the Kaplan-Meier method and were compared using the log-rank statistic. Raw and adjusted hazard ratios (HRs) and the corresponding confidence intervals (CIs) were obtained using Cox proportional hazards regression models. Interaction terms were fitted where appropriate.

Because of the sampling approach of the Health ABC Study, the study sample is not representative of the race distribution of older persons in the United States. Therefore, we opted to calculate only race-specific PAR estimates and did not calculate pooled PARs. Sex-specific and sex-stratified (Mantel-
Haenszel combined) rate ratio (RR) estimates for the risk factors of interest were calculated. Unadjusted (univariable) PARs were calculated using the following standard formula:

$$PAR_{unadj} = \frac{1 - \frac{R - 1}{R}}{\frac{P_1 - 1}{P_1}}$$

where $R$ is the incidence rate ratio (IRR) and $P_1$ is the prevalence of the disease in the exposed population. However, this formula is only valid when no confounding exists.$^{23}$ Therefore, we also calculated adjusted (multivariable) PARs using a Poisson regression model with incident HF as the outcome and the factors already described as predictors.$^{24}$ Briefly, the predicted number of cases is calculated for the full model ($N_{full}$), which equals the actual number of cases. Next, the effect of the risk factor of interest is “removed” by setting the value of the covariable to zero, and the predicted number of events is calculated ($N_{removed}$). The adjusted PAR for the risk factor then becomes $PAR_{adj} = 1 - \frac{N_{removed}}{N_{full}}$. Similarly, it is possible to calculate the total preventable fraction (ie, the proportion of cases that could be prevented if these risk factors were eliminated) by setting all covariables to zero; notably, the sum of the individual PARs does not add up to the total preventable fraction. Also, although maximum likelihood estimators have been shown to produce reliable point estimates and CIs of PAR in simulation studies,$^{24}$ no formal method to compare PAR for a risk factor between groups has been developed to date.

Hospitalizations were analyzed as count data over time at risk; RR and CIs were obtained by Poisson regression analysis. All analyses were performed using commercially available statistical software (STATA 10.0; StataCorp LP, College Station, Texas). Adjusted PARs were calculated using a STATA module (http://www.stata.com/stb/stb42/sbe21).

## RESULTS

### INCIDENT HF

The mean (SD) age of participants was 73.6 (2.9) years; 47.9% were men, and 58.6% were of white race. During a median follow-up of 7.1 years (25th-75th percentiles, 6.6-7.5 years), 258 participants (8.8%) developed HF (13.6 per 1000 person-years, 95% CI, 12.1-15.4). The baseline characteristics of participants are given in Table 1.

**Figure 1** shows the Kaplan-Meier estimates of incident HF in race-based and sex-based subgroups; men and black participants were more likely to develop HF than women and white participants. The rates were 16.3 per 1000 person-years in black participants vs 11.9 per 1000 person-years in white participants (age-adjusted HR, 1.41; 95% CI, 1.11-1.80; $P = .006$) and 15.8 per 1000 person-years in men vs 11.7 per 1000 persons-years in women (age-adjusted HR, 1.34; 95% CI, 1.05-1.71; $P = .02$). The sex × race interaction term was not statistically significant ($P = .47$).

**POPULATION-ATTRIBUTABLE RISKS**

Table 2 and Table 3 give the prevalences and the unadjusted (univariable) and adjusted (multivariable) PARs for the independent risk factors. Smoking, LVH, decreased albumin levels, and increased heart rate, fasting glucose levels, and systolic blood pressure were more prevalent in black participants than in white participants ($P < .001$ for all except heart rate [$P = .005$] and albumin level [$P = .01$]). In contrast, decreased GFR was more prevalent among white participants ($P = .001$). Prevalence of CHD was comparable across race ($P = .48$). The preventable fractions owing to modifiable risk factors were 48.9% (95% CI, 35.1%-59.8%) in white participants vs 67.8% (95% CI, 55.1%-76.8%) in black participants. Uncontrolled systolic blood pressure and CHD had the highest PARs (>20%) in both race groups in unadjusted and adjusted analyses. Adjusted PARs for all independent risk factors except serum glucose level were higher in black participants than in white participants, with smoking, LVH, and GFR less than 60 mL/min/1.73 m² being approximately 10% higher.

### OUTCOMES AFTER INCIDENT HF

During a median of 2.1 years (25th-75th percentiles, 0.7-4.4 years) after initial hospitalization for HF, 121 of 258 participants (46.9%) with HF died, representing 18.0% (95% CI, 15.0%-21.5%) annual mortality. In addition, 312 all-cause readmissions were recorded (46.3 per 100 person-years); of these, 252 (80.8%) (37.4 per 100 person-years) were related to HF. In comparison, participants who remained HF-free throughout the study had an annual mortality of 2.72% (95% CI, 2.49%-2.97%) and a hospitalization rate of 21.9 per 100 person-years ($RR = 2.11$; 95% CI, 1.88-2.37; $P < .001$).

**Table 4** summarizes the outcomes after incident HF in the race-based and sex-based subgroups. Overall survival did not differ between white participants and black participants (log-rank $X^2 = 0.00$, $P = 98$); however, there was a significant sex × race interaction (white male vs female HR, 0.70; 95% CI, 0.41-1.21; $P = .20$ and black male vs female HR, 2.10; 95% CI, 1.23-3.60; $P = 0.02$ for interaction) (Figure 2). Rates of all-cause hospitalization were 2-fold higher in black participants compared with white participants (62.1 vs 30.3 per 100 person-years; RR, 2.05; 95% CI, 1.62-2.60; $P < .001$). This effect was mainly because of HF-related readmissions: black participants had 53.2 HF-related hospitalizations per 100 person-years compared with 21.3 for white participants (RR, 2.50; 95% CI, 1.90-3.30; $P < .001$). The differences in hospitalization rates between race-based subgroups persisted after controlling for age and number of concomitant conditions (data not shown). No sex × race interaction was observed for all-cause and HF rehospitalization rates ($P = .36$ and $P = .71$, respectively, for the interaction terms).

Data on ventricular function during hospitalization for HF were not prospectively collected in the Health ABC Study; therefore, the available data are based on medical record reviews. Data on left ventricular ejection fraction (LVEF), obtained from echocardiography or left ventriculography reports during the index HF hospitalization, were available in 197 of 258 participants (76.4%). The median LVEF was 40% (interquartile range, 26%-55%), without difference between white and black participants (median LVEF, 40% for both groups). However, LVEF was higher in female participants (median LVEF, 46% vs 35% in men; $P = .01$); no sex × race interaction was observed. Participants with LVEF greater than 40% (n = 97 [49.2%]) had subsequent age-adjusted mortality 14.9 per 100 person-years, all-cause hospitalization rate 51.3 per 100 person-years, and HF rehospitalization rate 38.6 per 100 person-years. In comparison, participants with LVEF of 40% or less (n = 100 [50.8%])
had age-adjusted mortality 23.2 per 100 person-years (HR, 1.46; 95% CI, 0.98-2.18; P = .06), all-cause hospitalization rate 53.5 per 100 person-years (RR, 1.04; 95% CI, 0.82-1.32; P = .74) and HF rehospitalization rate 46.3 per 100 person-years (RR, 1.20, 95% CI, 0.92-1.57, P = .18).

In the present study, we found a high rate of incident HF in older persons, poor prognosis for these participants once they developed HF, and race-related differences in

**Comment**

In the present study, we found a high rate of incident HF in older persons, poor prognosis for these participants once they developed HF, and race-related differences in
risk factor profiles for incident HF. Considering the worsening risk factor profile for HF in the population (eg, diabetes and hypertension), aging of the population, and increasing HF prevalence, our study underscores the need for focused HF prevention efforts.

**HF INCIDENCE**

The rate of incident HF in our study was similar to a recent study composed of an elderly population\(^5\); however, these rates are lower compared with data from administrative databases.\(^5\)\(^6\)\(^7\)\(^20\) Varying rates for incident HF have also been reported in the Framingham Heart Study, in the Cardiovascular Health Study, and from Olmsted County, Minnesota.\(^4\)\(^5\)\(^10\) These variations are likely related to different age groups, racial mix, geographic variation, and other variables. Our study most likely underestimated the HF rate because the definition of incident HF required hospitalization and some patients with new-onset HF may not require hospitalization. Also, diagnosing HF is challenging, and investigations have primarily used signs and symptoms, medication profile review, and cardiac imaging–based assessment; some investigators have used medical record reviews, whereas others have used administrative databases. This becomes more complicated when assessing HF with preserved ejection fraction because its signs and symptoms are nonspecific, there is no standard medication profile, and imaging characteristics are complicated.

Irrespective of the precise quantitative assessment, several themes are similar across these studies, including a high incidence rate, worsening profile with aging, and stagnant or increasing incidence rates during the past several decades. These trends have tremendous implications as the population demographics are changing. With the aging of 78 million baby boomers, 1 in 5 Americans is expected to be older than 65 years by 2050.\(^27\) This trend is projected to significantly affect health care and health care economics because the use of formal and informal services strongly correlates with age. Without effective prevention efforts, the current HF epidemic can be expected to substantially worsen in the near future.

**RISK FACTORS FOR HF**

Although risk factors for HF have been described,\(^10\) those data have limitations because many are based on older studies primarily among younger white participants. Many investigations have assessed risk factors in isolation rather than assessing their multivariable independent association. The risk factor profile for cardiovascular diseases is changing with increasing obesity prevalence, and sex- and race-based differences in risk factors and outcomes for incident HF among older persons were not evaluated in some studies.\(^12\)\(^13\)\(^14\) In the present study, most modifiable risk factors were significantly more prevalent among black participants (compared with their white counterparts) and constituted a major driving force for the higher incidence of HF among black participants. Last, although the literature on HF risk factors is rich, data on PAR are limited.\(^28\)\(^29\) and only the Cardiovascular Health Study\(^10\) has specifically addressed this issue.

In the Cardiovascular Health Study,\(^10\) the investigators assessed PAR for all significant associations with HF. Several of these may be collinear and may “dilute” the true effect of modifiable risk factors. For example, stroke, CHD, ankle-arm index, carotid intima medial thickness, and electrocardiographic evidence of ST-T wave changes were included in the analysis because of their statistical significance. However, they may be related to a common domain of vascular atherosclerosis as a risk factor. Indeed, the CHD-attributable risk was only 13%.

Assessment of PAR requires binary categorization of continuous variables; such categorization in the Cardiovascular Health Study was not based on clinically used cutoffs (eg, C-reactive protein level, \(>7\) mg/dL). Finally, sex- and race-based analyses were not performed, and we found significant differences in this regard.

Coronary heart disease and uncontrolled blood pressure were the leading causes of HF in sex- and race-based subgroups. We also observed that a substantial proportion of HF is attributed to metabolic and cardiorenal factors, including glucose level and renal abnormalities as described previously\(^30\)\(^31\) and low albumin levels. It is unclear whether a low albumin level signifies cachexia, inflammation, or comorbidity burden or whether hypoalbuminemia precipitates symptomatic HF due to fluid extravasation.\(^32\) Increased heart rate has been reported as an HF risk factor and may represent a surrogate of vagovagal imbalance or a physiologic response to worsening cardiac function.\(^33\) Although much work is needed to assess which of these risk factors can be modified and how much (if any) HF risk reduction may be achieved, it is still notable that most risk factors were modifiable or amenable to potential intervention.

Two observations regarding racial differences merit special comment. First, LVH seems to affect mainly blacks, which is consistent with the high prevalence of uncontrolled blood pressure among persons of black race. However, the risk associated with LVH in our study was additive to and independent of that associated with uncontrolled blood pressure; in fact, LVH was encountered in 8.6% of participants with systolic blood pressure of less than 140 mm Hg. Second, the higher incidence of HF observed among black participants in our

![Figure 1.](image-url)
study is accompanied by a higher prevalence of modifiable risk factors and a higher preventable fraction of incident HF. Except for increased fasting glucose level, PAR for all risk factors was higher in black participants, with 6 of 8 risk factors assessed (CHD, LVH, smoking, reduced GFR, increased heart rate, and uncontrolled blood pressure) having more than 5% higher PAR.

**OUTCOMES AFTER HF DEVELOPMENT**

Despite the selection bias resulting in lower comorbidity burden compared with that in the general population, mortality after hospitalization for HF in the Health ABC Study was high (annual rate, 18.0%), similar to that reported in recent community-based studies. In fact, investigations assessing temporal trends in mortality after HF development demonstrate none or only modest improvements during the past few decades. These results suggest that the benefit from recent advances in HF therapy observed in “real-life” patient populations is significantly less than that in the clinical trial setting, that recent advances may not have been translated yet into routine clinical practice, or that HF characteristics at the community level are different from those in clinical trials. In concordance with a retrospective cohort study, we observed significantly higher all-cause and HF read-

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### Table 2. Prevalence and Unadjusted Rate Ratios (RRs) and Population-Attributable Risk (PAR) of Modifiable Risk Factors for Incident Heart Failure

<table>
<thead>
<tr>
<th>Risk Factor</th>
<th>Prevalence, %</th>
<th>RR (95% CI)</th>
<th>Prevalence, %</th>
<th>RR (95% CI)</th>
<th>P Value</th>
<th>Prevalence, %</th>
<th>RR (95% CI)</th>
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<tbody>
<tr>
<td></td>
<td>White Race (886 Men and 834 Woman)</td>
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<td>Modifiable</td>
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<tr>
<td>Systolic blood pressure &gt;140 mm Hg</td>
<td>32.5</td>
<td>1.91 (1.21-3.02)</td>
<td>32.7</td>
<td>1.78 (0.99-3.17)</td>
<td>.84</td>
<td>32.6</td>
<td>1.86 (1.33-2.61)</td>
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<tr>
<td>Coronary heart disease</td>
<td>23.3</td>
<td>2.66 (1.66-4.22)</td>
<td>10.1</td>
<td>2.67 (1.24-5.29)</td>
<td>.99</td>
<td>16.9</td>
<td>2.66 (1.84-3.84)</td>
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<tr>
<td>Fasting glucose level &gt;125 mg/dL</td>
<td>14.6</td>
<td>2.30 (1.34-3.82)</td>
<td>5.5</td>
<td>1.43 (0.38-3.90)</td>
<td>.41</td>
<td>10.2</td>
<td>2.09 (1.34-3.25)</td>
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<tr>
<td>Left ventricular hypertrophy</td>
<td>7.0</td>
<td>0.74 (0.20-1.96)</td>
<td>6.5</td>
<td>1.22 (0.32-3.31)</td>
<td>.49</td>
<td>6.7</td>
<td>0.92 (0.45-1.87)</td>
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<tr>
<td>Current smoking</td>
<td>5.1</td>
<td>1.32 (0.42-3.21)</td>
<td>7.7</td>
<td>2.72 (1.17-5.64)</td>
<td>.22</td>
<td>6.3</td>
<td>1.96 (1.12-3.42)</td>
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<tr>
<td>GFR &lt;60 mL/min/1.73 m²</td>
<td>20.5</td>
<td>1.18 (0.65-2.01)</td>
<td>28.6</td>
<td>1.69 (0.93-3.03)</td>
<td>.35</td>
<td>24.4</td>
<td>1.39 (0.96-2.04)</td>
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<tr>
<td>Albumin level &lt;3.8 g/dL</td>
<td>19.5</td>
<td>1.32 (0.74-2.33)</td>
<td>22.6</td>
<td>1.40 (0.71-2.60)</td>
<td>.88</td>
<td>21.0</td>
<td>1.35 (0.91-1.99)</td>
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<tr>
<td>Heart rate &gt;75 beats/min</td>
<td>14.5</td>
<td>1.32 (0.68-2.37)</td>
<td>15.1</td>
<td>1.98 (0.97-3.77)</td>
<td>.35</td>
<td>14.8</td>
<td>1.57 (1.03-2.39)</td>
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</table>

<table>
<thead>
<tr>
<th>Risk Factor</th>
<th>Prevalence, %</th>
<th>RR (95% CI)</th>
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<th>P Value</th>
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<td>Black Race (520 Men and 694 Women)</td>
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<tr>
<td>Systolic blood pressure &gt;140 mm Hg</td>
<td>42.3</td>
<td>1.90 (1.10-3.34)</td>
<td>42.1</td>
<td>2.02 (1.20-3.44)</td>
<td>.87</td>
<td>42.2</td>
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<td>Coronary heart disease</td>
<td>17.6</td>
<td>4.77 (2.72-8.30)</td>
<td>14.6</td>
<td>2.82 (1.56-4.93)</td>
<td>.17</td>
<td>15.9</td>
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<td>Fasting glucose level &gt;125 mg/dL</td>
<td>18.9</td>
<td>1.67 (0.86-3.08)</td>
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<td>1.73 (0.90-3.14)</td>
<td>.94</td>
<td>17.3</td>
<td>1.70 (1.13-2.57)</td>
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<tr>
<td>Left ventricular hypertrophy</td>
<td>18.3</td>
<td>2.31 (1.25-4.13)</td>
<td>19.6</td>
<td>1.90 (1.06-3.28)</td>
<td>.61</td>
<td>19.0</td>
<td>2.08 (1.42-3.04)</td>
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<td>Current smoking</td>
<td>21.0</td>
<td>1.88 (1.01-3.35)</td>
<td>13.0</td>
<td>1.75 (0.85-3.32)</td>
<td>.87</td>
<td>16.4</td>
<td>1.82 (1.20-2.76)</td>
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<td>Potentially modifiable</td>
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<tr>
<td>GFR &lt;60 mL/min/1.73 m²</td>
<td>14.8</td>
<td>2.57 (1.35-4.68)</td>
<td>14.8</td>
<td>2.25 (1.19-4.03)</td>
<td>.73</td>
<td>14.8</td>
<td>2.40 (1.60-3.59)</td>
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<tr>
<td>Albumin level &lt;3.8 g/dL</td>
<td>26.4</td>
<td>1.35 (0.73-2.42)</td>
<td>22.6</td>
<td>1.40 (0.71-2.60)</td>
<td>.88</td>
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<td>Heart rate &gt;75 beats/min</td>
<td>18.9</td>
<td>1.93 (1.03-3.47)</td>
<td>18.7</td>
<td>1.74 (0.95-3.04)</td>
<td>.79</td>
<td>18.8</td>
<td>1.82 (1.23-2.70)</td>
</tr>
</tbody>
</table>

**Table 3. Adjusted Rate Ratios (RRs) and Population-Attributable Risk (PAR) of Modifiable Risk Factors for Incident Heart Failure**

<table>
<thead>
<tr>
<th>Risk Factor</th>
<th>White Participants (n=1686)</th>
<th>PAR, %</th>
<th>Black Participants (n=1167)</th>
<th>PAR, %</th>
</tr>
</thead>
<tbody>
<tr>
<td>Modifiable</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Systolic blood pressure &gt;140 mm Hg</td>
<td>1.80 (1.27-2.55)</td>
<td>21.3</td>
<td>1.95 (1.33-2.84)</td>
<td>30.1</td>
</tr>
<tr>
<td>Coronary heart disease</td>
<td>2.72 (1.89-3.90)</td>
<td>23.9</td>
<td>3.31 (2.26-4.85)</td>
<td>29.5</td>
</tr>
<tr>
<td>Fasting glucose level &gt;125 mg/dL</td>
<td>2.08 (1.35-3.22)</td>
<td>11.3</td>
<td>1.37 (0.88-2.14)</td>
<td>7.3</td>
</tr>
<tr>
<td>Left ventricular hypertrophy</td>
<td>0.90 (0.44-1.84)</td>
<td>...</td>
<td>2.29 (1.47-3.30)</td>
<td>19.5</td>
</tr>
<tr>
<td>Current smoking</td>
<td>2.04 (1.15-3.64)</td>
<td>5.5</td>
<td>2.08 (1.37-3.16)</td>
<td>15.0</td>
</tr>
<tr>
<td>Preventable fraction</td>
<td>...</td>
<td>48.9</td>
<td>...</td>
<td>67.8</td>
</tr>
<tr>
<td>Potentially modifiable</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>GFR &lt;60 mL/min/1.73 m²</td>
<td>1.29 (0.88-1.87)</td>
<td>6.8</td>
<td>2.14 (1.42-3.24)</td>
<td>16.2</td>
</tr>
<tr>
<td>Albumin level &lt;3.8 g/dL</td>
<td>1.46 (0.98-2.16)</td>
<td>8.5</td>
<td>1.63 (1.09-2.44)</td>
<td>12.7</td>
</tr>
<tr>
<td>Heart rate &gt;75 beats/min</td>
<td>1.45 (0.94-2.23)</td>
<td>6.7</td>
<td>1.97 (1.30-2.99)</td>
<td>15.7</td>
</tr>
<tr>
<td>Potentially preventable fraction</td>
<td>...</td>
<td>20.5</td>
<td>...</td>
<td>38.6</td>
</tr>
</tbody>
</table>

**Abbreviations:** CI, confidence interval; ellipsis, not applicable; GFR, glomerular filtration rate.

SI conversion factors: To convert albumin to grams per liter, multiply by 10; to convert glucose to millimoles per liter, multiply by 0.0555.

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Table 4. Mortality and Rehospitalization Rates After Incident Heart Failurea

<table>
<thead>
<tr>
<th>Variable</th>
<th>Rate (per 100 person-years)</th>
<th>Hazard Ratio (95% CI)</th>
<th>P Value</th>
<th>Rate (per 100 person-years)</th>
<th>RR (95% CI)</th>
<th>P Value</th>
<th>Rate (per 100 person-years)</th>
<th>RR (95% CI)</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>White race (n=135)</td>
<td>18.0</td>
<td>..</td>
<td>..</td>
<td>30.3</td>
<td>1.02 (0.68-1.52)</td>
<td>.94</td>
<td>21.3</td>
<td>1.05 (0.80-1.38)</td>
<td>.73</td>
</tr>
<tr>
<td>Male sex (n=82)</td>
<td>16.7</td>
<td>0.70 (0.41-1.21)</td>
<td>.20</td>
<td>30.4</td>
<td>1.02 (0.68-1.52)</td>
<td>.94</td>
<td>19.6</td>
<td>0.82 (0.52-1.32)</td>
<td>.42</td>
</tr>
<tr>
<td>Female sex (n=53)</td>
<td>20.0</td>
<td>1 [Reference]</td>
<td>..</td>
<td>30.0</td>
<td>1 [Reference]</td>
<td>..</td>
<td>23.8</td>
<td>1 [Reference]</td>
<td>..</td>
</tr>
<tr>
<td>Black race (n=123)</td>
<td>18.0</td>
<td>..</td>
<td>..</td>
<td>62.1</td>
<td>..</td>
<td>..</td>
<td>53.2</td>
<td>..</td>
<td>..</td>
</tr>
<tr>
<td>Male sex (n=58)</td>
<td>26.3</td>
<td>2.10 (1.23-3.60)</td>
<td>.007</td>
<td>63.9</td>
<td>1.05 (0.80-1.38)</td>
<td>.73</td>
<td>57.2</td>
<td>1.26 (0.84-1.51)</td>
<td>.43</td>
</tr>
<tr>
<td>Female sex (n=65)</td>
<td>12.3</td>
<td>1 [Reference]</td>
<td>..</td>
<td>60.9</td>
<td>1 [Reference]</td>
<td>..</td>
<td>50.8</td>
<td>1 [Reference]</td>
<td>..</td>
</tr>
</tbody>
</table>

Abbreviations: CI, confidence interval; ellipsis, not applicable; RR, rate ratio.

a Rates refer to events per 100 person-years.

b Rate ratio, 2.05; 95% CI, 1.62-2.60; P < .001 compared with white participants.

c Rate ratio, 2.50; 95% CI, 1.90-3.30; P < .001 compared with white participants.

Figure 2. Mortality after incident heart failure by sex and race. Overall survival did not differ between white and black participants; however, there was a statistically significant sex × race interaction, with black men demonstrating higher mortality than white men compared with their female counterparts.

VALUE OF POPULATION-ATTRIBUTABLE RISK

The PAR for a risk factor represents the proportional reduction in disease risk that would be achieved by eliminating the risk factor from the population, assuming a causal relation. The relative importance of risk factors in the population as defined by PAR can help guide policymakers in planning public health interventions. However, the absolute PAR estimates are dependent on the definition of risk factors and on the presence or absence of other risk factors in the multivariable setting.

LIMITATIONS

Diagnosis of incident HF in our study was based on HF hospitalization; therefore, we likely underestimated the true incidence. Echocardiography was not performed at baseline. Therefore, participants with subclinical prevalent structural heart disease may have been included in the analysis. The Health ABC Study did not collect detailed data on valvular heart disease; however, it is unlikely that a large proportion of participants had significant subclinical valvular heart disease that would affect the results overall. Also, because ventricular function during hospitalization for HF was not prospectively assessed, we could not reliably assess the differential effect of risk factors on development of HF with preserved vs reduced LVEF. The available data on LVEF are based on medical record reviews and do not refer to a single modality. Therefore, we cannot be confident that the distribution of LVEF is representative of older persons hospitalized with new-onset HF. Finally, the causes of the observed differences in outcomes between groups cannot be ascertained in this study. These differences may represent sex, race, severity of illness, or therapy-related differences and need further study.

In this study, we observed a high incidence of HF among older persons. Race-based differences in risk factors were noted. Outcomes after development of HF remain poor. However, most risk factors for HF are modifiable or potentially amenable to interventions. Therefore, HF prevention efforts may succeed in reducing the community-based burden of HF. Considering the worsening risk factor profile and aging of the population, such efforts should be considered a public health priority.

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