Race and Renal Impairment in Heart Failure
Mortality in Blacks Versus Whites

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Background—Renal impairment is an emerging prognostic indicator in heart failure (HF) patients. Despite known racial differences in the progression of both HF and renal disease, it is unclear whether the prognosis for renal impairment in HF patients differs by race. We sought to determine in HF patients the 1-year mortality risks associated with elevated creatinine and impaired estimated glomerular filtration rate (eGFR) and to quantify racial differences in mortality.

Methods and Results—We retrospectively evaluated the National Heart Care Project nationally representative cohort of 53,640 Medicare patients hospitalized with HF. Among 5,669 black patients, mean creatinine was 1.6±0.9 mg/dL, and 54% had an eGFR ≤60, compared with creatinine 1.5±0.7 mg/dL and 68% eGFR ≤60 in 47,971 white patients. Higher creatinine predicted increased mortality risk, although the magnitude of risk differed by race (interaction \( P=0.0001 \)).

Every increase in creatinine of 0.5 mg/dL was associated with a >10% increased risk in adjusted mortality for blacks, compared with >15% increased risk in whites (interaction \( P=0.0001 \)), with the most striking racial disparities at the highest levels of renal impairment. Depressed eGFR showed similar racial differences (interaction \( P=0.0001 \)).

Conclusions—Impaired renal function predicts increased mortality in elderly HF patients, although risks are more pronounced in whites. Distinct morbidity and mortality burdens in black versus white patients underscore the importance of improving patient risk-stratification, defining optimal therapies, and exploring physiological underpinnings of racial differences. (Circulation. 2005;111:1270-1277.)

Key Words: ethnic groups ■ kidney ■ heart failure ■ mortality

Renal impairment has emerged as a critical risk factor for mortality in patients with heart failure (HF), with impaired renal function conferring nearly twice the risks for death and hospitalization in published studies.1–10 No large study of HF patients has determined how race modifies these risks, although previous literature offers evidence for racial differences in the epidemiology and progression of both HF and renal disease. Specifically, evidence suggests that black patients with hypertension and HF progress earlier to worsening systolic dysfunction,11–17 and black patients with chronic kidney disease have a higher incidence of end-stage renal disease (ESRD).18,19 These findings may reflect, in part, underlying differences by race in adaptations of the renin-angiotensin and sympathetic nervous systems or other neurohormonal factors,20–22

We sought to determine whether the mortality risks associated with various levels of renal impairment in HF differ by race. To address this question, we used data from the National Heart Care (NHC) Project, a community-based cohort of elderly patients who were initially hospitalized with HF. The NHC cohort is ideal for addressing this question, because it not only is a nationally representative sample of elderly, hospitalized HF patients but also includes patients free of ESRD at baseline with the spectrum of mild to severe renal impairment, unlike many study populations that exclude patients with clinically significant renal disease at baseline.

Methods

NHC Project

The NHC project is a Centers for Medicare and Medicaid Services quality improvement initiative for fee-for-service Medicare beneficiaries hospitalized with HF. Patients hospitalized between March 1998 to April 1999 and July 2000 to June 2001 with a principal discharge diagnosis of HF (International Classification of Diseases,
Ninth Revision, Clinical Modification codes 402.01, 402.11, 402.91, 404.01, 404.91, or 428) were identified, with codes shown to be >95% specific for HF in a previous study.21 Patients were included if this was the first admission for HF within the study period, they had valid social security numbers, they did not transfer from hospital or leave against medical advice, and they were not on long-term hemodialysis. Eight hundred records (or all records, in states with <800 total discharges) were randomly sampled from each state for each period, after sorting by age, race, sex, and treating hospital. Data were abstracted from medical records, with quality assurance through trained reviewers, medical record abstraction software, and random record reabstraction. Hospital data were derived from the American Hospital Association annual survey, and physician data were derived from the American Medical Association Physician Masterfile.

Study Cohort
Of 78,882 hospitalizations, excluded were second visits of patients in the sample appearing more than once (3732); patients <65 years of age (6558); transfers from other hospitals (2419); no clinical evidence of HF on admission (5003); patients with aortic stenosis (5493) and mitral stenosis (243) to exclude valvular HF; and patients on chronic dialysis (549). Because we were primarily interested in examining differences between black and white patients, patients defined as other race by the medical record (3216) were excluded. Patients with missing admission creatinine (1746) or error in death information (969) were excluded from analysis, for a total sample of 53,640.

Renal Function
Renal function was determined from initial serum creatinine level (mg/dL) on hospital admission. Creatinine was characterized as a categorical variable a priori by tertiles in bivariate analyses (<1.0; >1.0 to 1.5; >1.5), representing normal, mild, and moderate-to-severe impairment of renal function. Creatinine was also characterized continuously in multivariable analyses on the basis of linearity with outcomes. Hazards ratios were calculated for increases in creatinine per 0.5 mg/dL.

As a secondary characterization of renal function, we estimated glomerular filtration rate (eGFR) using the simplified Modification of Diet in Renal Disease (MDRD) prediction formula24,25: eGFR = 186 x (serum creatinine $^{-1.154}$) x (age $^{-0.203}$) x (1.212 (if black) x 0.742 (if female)) (mL · min $^{-1}$ · 1.73 m$^{-2}$). Categorical characterization of eGFR (<30; 30 to 60; >60) was based on National Kidney Foundation definitions for severe, moderate, and normal to mildly decreased GFR.26,27; in addition, a previous study found the association of eGFR with clinical outcomes to be non-linear.8 An additional subsidiary analysis used blood urea nitrogen (BUN) as a continuous variable (per 10 mg/dL).

Outcomes
All-cause mortality was assessed using the Medicare Enrollment database and Medicare Part A database. Follow-up time of up to 1 year was calculated from initial hospital admission date. In a subsidiary analysis, follow-up time was calculated from hospital discharge date, excluding patients who died in hospital.

Covariates
Our analyses adjusted for covariates selected a priori on the basis of previous studies and clinical relevance, including age and sex; severity of disease variables, including history of HF and presentation with peripheral edema; other clinical characteristics, including ejection fraction (normal, mild, moderate, severe, and not documented), admission serum sodium, potassium, glucose, hematocrit, albumin, heart rate, respiratory rate, and systolic blood pressure; cardiovascular comorbidities, including hypertension, myoccardial infarction (MI), angina, and CABG or PTCA; noncardiac comorbidities, including diabetes, previous stroke, smoking, chronic obstructive pulmonary disease, urinary incontinence, mobility, dementia, cancer, liver disease, and cirrhosis; prearrival setting; preadmission cardiac medications, including ACE inhibitors, β-blockers, diuretics, digoxin, and calcium channel blockers; and health care setting, including urban versus rural hospital and treating physician specialty. Covariates lacking linear relationships with outcomes were entered into models as categorical (dummy) variables in multivariable analyses.

Statistical Analysis
Unadjusted associations between creatinine and covariates were tested by use of the Pearson $\chi^2$ test for categorical variables and the Kruskal-Wallis test for continuous variables. Adjusted mortality rates were calculated as a multiplication product of overall mortality rates and risk-standardized ratios. Multivariable Cox proportional-hazards regressions tested whether creatinine was an independent predictor of 1-year all-cause mortality. Missing values for all covariates were coded as dummy variables. To assess whether creatinine provided comparable 1-year mortality risks for black versus white patients, modification of this association by race was tested using an interaction (product) term for race and creatinine. Proportionality assumptions were tested using a time-interaction term in the model, excluded from final models if not significant. Weighted proportional-hazards models were also performed to adjust for differences in population size by state and clustering by hospital.

Subsidiary Analyses
Interaction terms for creatinine and age, ejection fraction, hypertension, MI, CABG/PTCA, angina, and diabetes were tested on the basis of bivariate analyses, and also because racial differences in comorbidity profile could confound the relationship between renal impairment and outcomes. To confirm whether alternative means of characterizing renal function altered results, subsidiary models using categories of eGFR, BUN, and discharge creatinine (excluding in-hospital deaths) were tested.

All tests for significance were 2 tailed, with an $\alpha$-level of 0.05. Statistical analyses were conducted using SAS version 8.0 and Stata version 7.0 (Stata Corp). Use of the NHC database was approved by the Yale University School of Medicine Human Investigation Committee.

Results
Study Sample
In the sample of 53,640 patients, 89% were white, 42% were men, and mean age was 79±8 years. A total of 72% had a history of HF, 64% had a history of hypertension, 30% had a history of MI, and 40% had diabetes. Mean admission creatinine was 1.5±0.8 mg/dL; mean eGFR was 53±25 mL · min$^{-1}$ · 1.73 m$^{-2}$. For white patients, 31%, 37%, and 32% had normal, mild, and moderate-to-severe renal impairment (creatinine ≤1.0; >1.0 to 1.5; >1.5), compared with 28%, 37%, and 35% in black patients ($P=0.0001$) (Figure 1).

Differences by Level of Renal Function
Patients with worse renal function were more likely to be black, older, and women and to have a previous history of HF and ischemic disease, and were less likely to have received ACE inhibitors before admission. Blacks had greater prevalence of hypertension and diabetes but were less likely to have ischemic cardiovascular disease. In addition, renal impairment was strongly associated with ischemic disease in whites compared with blacks (Table 1).

Mortality
A total of 38% of patients ($n=20,179$ of 53,640) died during 1-year follow-up, including 29%, 35%, and 52% of patients with normal, mild, and moderate-to-severe renal impairment ($P=0.0001$), and this relationship of creatinine with mortality
appeared to be relatively linear (Figure 2). Elevated creatinine was associated with increased risk of death after adjustment for all covariates, including age, sex, severity of disease, comorbidities, clinical presentation, admission medications, and health care setting (Table 2).

Racial Differences in Mortality
Unadjusted and adjusted mortality risks showed that black patients had lower risks than white patients at every level of creatinine, and this difference increased in magnitude with worse creatinine (Figure 2). In multivariable analysis, the interaction between creatinine and race was highly significant (P=0.0001). Hazard estimates associated with increasing creatinine differed when stratified by race: in white patients, for every increase in creatinine by 0.5 mg/dL, the relative risk of 1-year mortality increased by approximately 15% (HR=1.16; 95% CI, 1.15, 1.17; P=0.0001); and in black patients, for every increase in creatinine by 0.5 mg/dL, the relative risk of 1-year mortality increased by approximately 10% (HR=1.11; 95% CI, 1.08, 1.14; P=0.0001). In models adjusting for population weight by state and clustering by hospital, risks for mortality by race remained similar (Table 2).

Adjusted 1-year mortality rates ranged from 25% to 52% for black patients with creatinine ≤1.0 to >3.0, whereas comparable risks were 29% and 77% in whites (Figure 2). This increasing divergence in mortality rates by race, which became more pronounced with worsening levels of renal function, also translated into diverging hazard ratios for death. Adjusted hazard ratio for death was 1.99 (1.62, 2.45) in black patients with creatinine >3.0 (referent creatinine, ≤1.0), compared with 2.61 (2.44, 2.80) in white patients (Figure 3).

Similarly, using renal function characterized by eGFR, black patients with the worst level of renal function (eGFR <30) had approximately a 50% mortality risk at 1 year, compared with a 1-year mortality risk of 60% for white patients (Figure 4). Adjusted risks of death for patients with eGFR <30 and eGFR 30 to 60 (referent eGFR, >60) were 1.88 (1.81, 2.06) and 1.19 (1.14, 1.23) for white patients, compared with 1.74 (1.50, 2.02) and 1.14 (1.02, 1.27) for black patients (P=0.0001 for interaction).

Subsidiary Models
Multivariable models additionally adjusted for interaction terms for creatinine and age, ejection fraction, hypertension, MI, CAGB /PTCA, angina, and diabetes did not explain the primary interaction between race and GFR on the outcome of mortality. Magnitude of mortality risks and the race interaction using discharge creatinine remained essentially unchanged (10% in blacks, 17% in whites; P=0.0001 for interaction). Also, 1-year mortality risk in blacks increased 15% for every 10-mg/dL elevation in BUN compared with an 18% increase per 10 mg/dL for whites in adjusted analysis (P=0.0001 for interaction).

Discussion
In this nationally representative sample of elderly patients hospitalized with HF, 68% of white patients and 54% of black HF patients had moderate to severe renal impairment as defined by National Kidney Foundation guidelines (eGFR ≤60). Race significantly modified mortality risks associated with renal impairment, summarized by a “5, 10, 15” rule: For every increase in creatinine by 0.5 mg/dL, 1-year death risk increased by approximately 10% in black patients and approximately 15% in white patients. For both blacks and whites, the relationship between creatinine and mortality appeared to be linear, with the greatest divergence in risk at the worst levels of renal function. Race differences in mortality risks could not be explained by confounding or effect modification by age, history of MI, hypertension, diabetes, ejection fraction, anemia, or medications, including ACE inhibitors. The effect of race on the association of renal impairment with mortality was also observed when renal impairment was defined using eGFR or BUN.

Previous Literature
Other studies have explored the association between renal impairment and poor clinical outcomes in HF patients, although
no study has addressed whether this association varies by race. An increasingly diverse body of literature, including retrospective studies on clinical trial cohorts such as the Cooperative North Scandinavian Enalapril Survival Study (CONSENSUS),5 Second Prospective Randomized study of Ibopamine on Mortality and Efficacy (PRIME-II),4 Studies Of Left Ventricular Dysfunction (SOLVD),3 and (Digitalis Investigation Group) DIG trial,2,8 Medicare patients,7 hospitalized HF patients,1,6,9 and outpatients,10 have shown that even mild to moderate renal impairment is associated with mortality.

<table>
<thead>
<tr>
<th>TABLE 1. Association Between Renal Impairment and Other Covariates by Race</th>
</tr>
</thead>
<tbody>
<tr>
<td>Characteristic</td>
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<tr>
<td></td>
</tr>
<tr>
<td>Renal function</td>
</tr>
<tr>
<td>Creatinine, mg/dL, mean (SD)*</td>
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<tr>
<td>eGFR, mL/min, mean (SD)*</td>
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<tr>
<td>Demographic</td>
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<tr>
<td>Age, y, mean (SD)</td>
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<tr>
<td>Male sex*</td>
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<tr>
<td>Severity of disease</td>
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<tr>
<td>History of HF*</td>
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<tr>
<td>Peripheral edema</td>
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<tr>
<td>Comorbidities</td>
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<tr>
<td>Hypertension</td>
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<tr>
<td>History of MI*</td>
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<tr>
<td>Angina*</td>
</tr>
<tr>
<td>CABG/PTCA*</td>
</tr>
<tr>
<td>Stroke</td>
</tr>
<tr>
<td>Diabetes</td>
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<tr>
<td>Presentation</td>
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<tr>
<td>Ejection fraction*</td>
</tr>
<tr>
<td>Normal</td>
</tr>
<tr>
<td>Mild</td>
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<tr>
<td>Moderate</td>
</tr>
<tr>
<td>Severe</td>
</tr>
<tr>
<td>Missing</td>
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<tr>
<td>Sodium, mEq/L, mean (SD)</td>
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<tr>
<td>Potassium, mEq/L, mean (SD)</td>
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<tr>
<td>Hematocrit, mean (SD)</td>
</tr>
<tr>
<td>Heart rate, bpm, mean (SD)</td>
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<tr>
<td>SBP, mm Hg, mean (SD)</td>
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<tr>
<td>Transfer from SNF</td>
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<tr>
<td>Admission medications</td>
</tr>
<tr>
<td>ACE inhibitors*</td>
</tr>
<tr>
<td>β-Blockers*</td>
</tr>
<tr>
<td>Diuretics*</td>
</tr>
<tr>
<td>Digoxin*</td>
</tr>
<tr>
<td>Ca channel blockers</td>
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<tr>
<td>Healthcare setting</td>
</tr>
<tr>
<td>Urban hospital*</td>
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<tr>
<td>Cardiologist*</td>
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</tbody>
</table>

Cr indicates creatinine; GFR, glomerular filtration rate; HF, heart failure; MI, myocardial infarction; CABG, coronary artery bypass graft; PTCA, percutaneous transluminal coronary angioplasty; SBP, systolic blood pressure; and SNF, skilled nursing facility.

Sodium, potassium, hematocrit, and physician specialty had ≤3% missing values, percentages of patients without missing values.

*Overall race differences significant at \( P<0.05 \).
from HF progression, providing some evidence for a mechanism for this relationship. Other investigators reported in the Cardiovascular Health Study cohort that poor renal function predicted incident HF, giving credence to the temporality of a possible causal relationship.\(^{28,29}\) Investigators have hypothesized that renal impairment could mediate worsening HF by contributing to upregulation of the renin-angiotensin-aldosterone system, enhanced basal sympathetic nerve discharge, and increased proinflammatory factors, which could worsen left ventricular hypertrophy and poor contractility of myocytes, ultimately leading to pump failure and death.\(^{30–32}\)

**Race and Renal Impairment**

To the best of our knowledge, no other studies in the literature have explored racial differences associated with renal impairment in HF patients. Previous reports suggest that black patients with a history of chronic kidney disease in the general population have a higher incidence of ESRD and furthermore may have an accelerated trajectory when progressing from mild baseline chronic kidney disease to ESRD, compared with white patients. Paradoxically, within the subset of patients with ESRD, some evidence suggests that black patients have lower mortality rates.\(^{33–35}\)

Results from our HF population are similar, with a significantly lower mortality risk for black patients with renal impairment. Because our study represents one of the largest cohorts of HF patients with the most extensive adjustment of possible confounding covariates to date, we believe these racial differences in mortality are genuine. Our results add to the literature by showing that, at least for HF patients, this paradoxical mortality profile exists not only in ESRD but even in mild renal impairment. Our results also suggest the novel hypothesis that racial differences in progression to ESRD may be explained in part by the increased mortality of white patients with mild-to-moderate chronic kidney disease. A recent study by Weiner et al\(^ {36}\) found that chronic kidney disease was a significant risk factor for coronary heart disease in community-based cohorts of patients without cardiovascular disease at baseline. Interestingly, black patients had an increased mortality risk compared with whites; however, the majority of these patients died of MI and coronary heart disease, and the contribution of HF in this cohort was not reported, although it was presumed to be relatively small. These results underscore the fact that the trajectory for worsening HF with renal impairment is unique and may indeed reflect unique underlying physiology with respect to the renin-angiotensin-aldosterone system or other neurohormonal factors and that renal impairment is not simply a marker for worse coronary artery disease in this select population.

No physiological explanations have adequately explained racial differences in HF and renal impairment progression, although numerous investigators have explored possible mechanisms underlying apparent racial differences in out-

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**TABLE 2. Creatinine and Adjusted Risk of 1-Year Mortality**

<table>
<thead>
<tr>
<th>General Model</th>
<th>HR*</th>
<th>95% CI</th>
<th>( P )</th>
</tr>
</thead>
<tbody>
<tr>
<td>Unadjusted</td>
<td>1.18</td>
<td>1.17–1.19</td>
<td>0.0001</td>
</tr>
<tr>
<td>Adding socio-demographics</td>
<td>1.19</td>
<td>1.18–1.20</td>
<td>0.0001</td>
</tr>
<tr>
<td>Adding all covariates†</td>
<td>1.15</td>
<td>1.14–1.16</td>
<td>0.0001</td>
</tr>
<tr>
<td>All covariates in weighted model</td>
<td>1.15</td>
<td>1.14–1.16</td>
<td>0.0001</td>
</tr>
<tr>
<td>Adding interaction term in fully adjusted model</td>
<td>Creatinine\times race</td>
<td>…</td>
<td>…</td>
</tr>
</tbody>
</table>

**Stratified analysis of fully adjusted model**

<table>
<thead>
<tr>
<th></th>
<th>HR*</th>
<th>95% CI</th>
<th>( P )</th>
</tr>
</thead>
<tbody>
<tr>
<td>White patients</td>
<td>1.16</td>
<td>1.15–1.17</td>
<td>0.0001</td>
</tr>
<tr>
<td>In weighted model‡</td>
<td>1.16</td>
<td>1.15–1.20</td>
<td>0.0001</td>
</tr>
<tr>
<td>Black patients</td>
<td>1.11</td>
<td>1.08–1.14</td>
<td>0.0001</td>
</tr>
<tr>
<td>In weighted model‡</td>
<td>1.11</td>
<td>1.08–1.15</td>
<td>0.0001</td>
</tr>
</tbody>
</table>

*HR represents hazards ratio for every increase in creatinine by 0.5 mg/dL, except interaction model.
†Includes all covariates listed in Table 1 and COPD, dementia, mobility, respiratory rate, and albumin.
‡Adjusts for population weight by state and for clustering by hospital.

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**Figure 2. Adjusted mortality by creatinine and race.**
comes for HF and hypertension. Small et al.\(^\text{37}\) recently reported that polymorphisms of \(\beta\)- and \(\alpha\)-adrenergic receptors differ by race and postulated that this variation may affect risks of developing HF. Variations in sodium intake and handling, potassium and calcium intake, fasting insulin levels, plasma renin activity, and urinary kallikrein excretion have also been proposed as causal factors in outcome differences by race in hypertension.\(^\text{38,39}\) Differences in these mechanisms could potentially be relevant for identifying whether the neurohormonal milieu in HF with renal impairment may also differ by race and whether such differences could help explain mortality risks by race observed in our analysis.

The normal variation of creatinine by race did not explain the race interaction in this study, given that we found similar results using the GFR estimation equation proposed by Levey and colleagues.\(^\text{24,25}\) This equation includes an adjustment factor of approximately 1.2 for black patients, which reflects blacks typically having higher GFR levels than whites at equal levels of serum creatinine. Lewis et al.\(^\text{40}\) recently reported that the Levey GFR estimation equation was useful for approximating true GFR in the African-American Study of Kidney disease and hypertension (AASK), which included 1703 African-American patients.

Race differences in HF outcomes, and the implications of these differences for guidelines for treatment, is a highly controversial subject. In particular, several recent studies have shown conflicting evidence for possible lower effectiveness of agents such as ACE inhibitors in black HF patients.\(^\text{41–44}\) Eliciting considerable discussion in the literature over the usefulness of establishing differing HF treatment strategies by race.\(^\text{45–49}\) At the same time, Schwartz\(^\text{45}\) particularly highlights the importance of regarding race as “a social construct, not a scientific classification” and the imprecision of “attributing differences in a biological end point to race.” Although our study cannot resolve this debate, the strong

Figure 3. Adjusted hazard ratios for mortality by creatinine and race. Hazard ratios use creatinine \(\leq 1.0\) mg/dL as referent category: hazard ratio (95% CI).

Figure 4. Adjusted mortality by eGFR and race.
independent interaction effect of race in our analysis, even after adjustment for numerous clinical comorbidities, suggests that race still remains an invaluable predictor variable in evaluating outcomes, even if it probably represents a combination of other underlying clinical and social factors. Understanding the differing profiles in patient subgroups is particularly salient given recent recommendations for routine reporting of eGFR in high-risk patients for risk-stratification and outcomes prognostication.30

Study Limitations

There are several issues in our study to consider. Only patients over the age of 65 years were included our study, although the majority of HF patients are elderly; in addition, because our data do not capture differences in hospital admission practices by race, these data cannot necessarily be extrapolated to the outpatient HF population. Second, renal function was estimated with only a single assessment, which probably reflected acute and chronic disease and could not distinguish renal impairment directly associated with HF from intrinsic renal disease. Finally, although creatinine and eGFR have unique limitations for estimating renal function, subsidiary analyses with other measures, including discharge creatinine and admission BUN, confirm the strength, direction, and magnitude of the association with narrow confidence intervals and help to confirm the validity of our findings, even for patients with mild impairment.

Conclusions

A substantial proportion of all patients with HF have impaired renal function and increased mortality risk, and this risk is even more pronounced in white HF patients. The mortality risks, which require further validation, are summarized by the “5, 10, 15” rule: For every increase in creatinine by 0.5 mg/dL, the 1-year death risk increases by approximately 10% in black patients and approximately 15% in white patients. The distinct morbidity and mortality burden in black versus white patients underscores the importance of improving patient risk-stratification, defining optimal therapies, and exploring the biological and environmental underpinnings of racial differences in disease trajectory.

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