Neighborhood Socioeconomic Status, Race, and Mortality in Young Adult Dialysis Patients

Tanya S. Johns,* Michelle M. Estrella,† Deidra C. Crews,‡ Lawrence J. Appel,§§ Cheryl A.M. Anderson,‡ Patti L. Ephraim,§ Courtney Cook,| and L. Ebony Boulware‡§**

*Division of Nephrology, Department of Medicine, Albert Einstein College of Medicine, Bronx, New York; †Division of Nephrology, Department of Medicine, Johns Hopkins University School of Medicine, Baltimore, Maryland; ‡Welch Center for Prevention, Epidemiology, and Clinical Research, Johns Hopkins Medical Institutions, Baltimore, Maryland; §Department of Epidemiology, Johns Hopkins Bloomberg School of Public Health, Baltimore, Maryland; §§Division of General Internal Medicine, Department of Medicine, Johns Hopkins School of Medicine, Baltimore, Maryland; ¶Division of Preventive Medicine, Department of Family and Preventive Medicine, University of California San Diego School of Medicine, San Diego, California; and **Division of General Internal Medicine, Department of Medicine, Duke University School of Medicine, Durham, North Carolina

ABSTRACT

Young blacks receiving dialysis have an increased risk of death compared with whites in the United States. Factors influencing this disparity among the young adult dialysis population have not been well explored. Our study examined the relation of neighborhood socioeconomic status (SES) and racial differences in mortality in United States young adults receiving dialysis. We merged US Renal Data System patient-level data from 11,027 black and white patients ages 18–30 years old initiating dialysis between 2006 and 2009 with US Census data to obtain neighborhood poverty information for each patient. We defined low SES neighborhoods as those neighborhoods in US Census zip codes with ≥20% of residents living below the federal poverty level and quantified race differences in mortality risk by level of neighborhood SES. Among patients residing in low SES neighborhoods, blacks had greater mortality than whites after adjusting for baseline demographics, clinical characteristics, rurality, and access to care factors. This difference in mortality between blacks and whites was significantly attenuated in higher SES neighborhoods. In the United States, survival between young adult blacks and whites receiving dialysis differs by neighborhood SES. Additional studies are needed to identify modifiable factors contributing to the greater mortality among young adult black dialysis patients residing in low SES neighborhoods.


In the United States, the incidence of ESRD is 3.4 times higher in blacks compared with whites.1 The greater incidence of ESRD among blacks has been attributed to prevalent CKD risk factors (including hypertension,2,3 diabetes,4,5 and obesity6), genetic predisposition,7–9 low socioeconomic status (SES),10–12 and inequalities in the access and quality of kidney disease care.13,14 Despite the greater incidence of ESRD, numerous studies have shown that blacks experience paradoxically better survival on dialysis compared with whites.15–35 Although the reasons for this survival paradox are not well understood, proposed mechanisms include more favorable nutritional and/or inflammatory profiles,29 greater resilience to inflammation,36 and tolerance of lower dialysis dose.15 Others postulate that improved access to health care afforded by the US Centers for Medicare and Medicaid Services (CMS) ESRD insurance coverage program may confer a survival benefit, especially to poor black patients likely to have been uninsured before dialysis initiation.35

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Correspondence: Dr. Tanya Johns, Division of Nephrology, Albert Einstein College of Medicine, Montefiore Medical Center, 1300 Morris Park Avenue, Bronx, NY 10461. Email: tjohns@montefiore.org

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A recent study by Kucirka et al. challenged the robustness of this survival paradox by showing that the risk of death varied across age strata, with 18- to 30-year-old black dialysis patients having nearly a 2-fold increased risk of death compared with similarly aged whites. Reasons for disparate findings among younger (versus older) dialysis patients have been poorly explored. Although older adults with progressive CKD often have access to private and public (e.g., Medicare) forms of health insurance, young adults are more frequently of low SES and uninsured, and they may be particularly vulnerable to receiving poor predialysis health care.

Although income is one of the most commonly used metrics to determine an individual’s SES, area-based SES factors, such as neighborhood poverty, may capture contextual factors of importance beyond individual measures. Area-based SES measures have been shown to be reflective of SES inequalities in health. Low area-based SES has been shown to be associated with poor health outcomes and linked to poorer dialysis outcomes even in the absence of individual-level SES data. The association between neighborhood SES and race disparities in mortality among young adult dialysis patients has not been previously examined.

RESULTS

Population Characteristics

Our cohort of young adult incident ESRD patients was obtained from the US Renal Data System (USRDS). After exclusions (Figure 1), our cohort included 11,027 young adult ESRD patients. Patients’ mean (SD) age was 25.3 (±3.6) years, 42% were black, 56% were men, 22% had diabetes, and 79% had hypertension. Over 90% of young adults were on hemodialysis. Rurality was low, with the majority of patients classified as living in an urban area (82%). More than 50% of these young patients did not see a nephrologist before starting dialysis. Young blacks were nearly two times more likely to live in a category III poverty neighborhood (47% versus 28%, respectively) and three times more likely to live in a category IV severe poverty neighborhood (6% versus 2%, respectively) compared with young whites (Figure 2).

Among patients classified as residing in low SES neighborhoods, young blacks were more likely to be women, reside in urban areas, and have higher body mass indexes (BMIs) compared with young whites. AIDS nephropathy was seen almost exclusively in young black patients, and young blacks were also more likely to have systemic lupus erythematosus or FSGS listed as a cause of ESRD compared with young whites. The prevalence of potential indicators of AKI (i.e., acute interstitial nephritis and tubular necrosis) as a cause of ESRD was low (1%) and similar between whites and blacks. Young blacks were more likely to have comorbidities, such as diabetes, hypertension, and congestive heart failure. The prevalence of tobacco use as well as drug and alcohol dependence was low and similar among the groups. More than 25% of young adults were uninsured at the time of dialysis initiation. Low SES young blacks were more likely than young whites to have Medicaid insurance and less likely to have private insurance. Most young adult patients in the low SES neighborhoods were not seen or it was unknown if they were seen by a nephrologist before dialysis initiation, and there was no difference by race. We observed similar trends among young blacks and young whites in the higher neighborhood SES group; however, the proportion of young blacks not seen (or unknown if seen) by a nephrologist was significantly greater compared with young whites (Table 1).

Outcomes in Young Blacks and Young Whites by Level of Neighborhood SES

During a median follow-up of 23 months, 1242 young adult patients died (11%), and 2383 (22%) patients received a kidney transplant. Mortality was highest in low SES young blacks (16%) followed by higher SES young blacks (12%) and similar (9%) in the low and higher SES young whites (P<0.001) (Figure 3). Cardiovascular (29%) and infectious (12%) causes accounted for 41% of all deaths. Respiratory failure, diabetes complications, and chronic renal failure complications accounted for the majority of other medical deaths. There were no deaths attributable to accidents, suicides, homicides, or trauma among those patients with a known cause of death. Among patients who died from cardiovascular causes, blacks residing in low SES neighborhoods had the highest proportion of deaths (35%). Within deaths attributable to infectious causes, young adult black patients accounted for 70% of deaths (38% low SES blacks and 32% higher SES blacks). Whites living in higher SES neighborhoods had the largest proportion
of deaths (37%) from other medical causes. Low SES whites were equally likely to die from cardiovascular (16%) and other medical causes (15%) (Figure 4). Of 28% of deaths with an unknown cause, 26% occurred in young blacks, and 30% occurred in young whites. Characteristics of those patients with a known cause of death were similar to the characteristics of patients with an unknown cause of death (Supplemental Table 1).

In the Cox model adjusted for age and sex, blacks had worse survival compared with whites, and this difference in survival differed by neighborhood SES (P<0.01 for interaction). Among patients residing in low SES neighborhoods, blacks had a 65% higher risk of death compared with whites (95% confidence interval [95% CI], 1.38 to 1.97) (Table 2, model 1). This risk was attenuated in the higher SES group, in which blacks had an 18% greater risk of death compared with similarly aged whites (95% CI, 1.01 to 1.38). Additional adjustments for BMI, baseline comorbidities, cause of ESRD, dialysis modality, rurality, and access to care factors showed that young adult blacks living in low SES neighborhoods had approximately a 50% greater hazard of death compared with young whites (aHR, 1.46; 95% CI, 1.18 to 1.71). Among patients residing in higher SES neighborhoods, young blacks had similar survival to young whites (aHR, 1.13; 95% CI, 0.96 to 1.33). In the competing risk model, the mortality disparity between young adult blacks and whites persisted among patients residing in low SES neighborhoods (aSHR, 1.51; 95% CI, 1.24 to 1.82). This difference was attenuated but still statistically significant in the higher SES group (aSHR, 1.28; 95% CI, 1.08 to 1.51 for young blacks compared with young whites). The P for interaction was >0.05 in both models. Analyses excluding patients receiving peritoneal dialysis and adjusting for access type in the fully adjusted Cox proportional and competing risk models yielded similar findings.

**DISCUSSION**

In this national study of 18- to 30-year-old incident United States ESRD patients, we found that mortality was greatest among low SES young blacks. Young blacks in both low and higher SES neighborhoods had greater mortality than young whites, regardless of their neighborhood SES. Young whites had similar mortality in higher and low SES neighborhoods. In the adjusted traditional Cox models accounting for patients’ baseline comorbidities, access to care, and rurality, young blacks living in low SES neighborhoods had approximately a 50% greater hazard of death compared with young whites living in low SES neighborhoods. In contrast, the relative
The hazard of death between young blacks and young whites living in higher SES neighborhoods was not statistically significant. Competing risk analyses accounting for racial disparities in transplantation showed an even greater risk of death among young blacks compared with young whites living in low SES neighborhoods as well as worse mortality among young blacks compared with young whites living in higher SES neighborhoods. The overall mortality rate for this young adult dialysis population was high, with 1 in 10 patients dying over a median follow-up of 2 years. Notably, the deaths attributable to infection were much greater among young blacks compared with young whites and also two times greater among young blacks compared with deaths from infection in a general dialysis cohort.42

Few studies have examined the association between race, SES, and mortality in ESRD. Earlier United States studies found an association between low SES and higher mortality and indicated that this relationship may be limited to blacks.43,44 However, recent large population-based studies have not shown a consistent association. Eisenstein et al.21 found no difference in survival by income level, and blacks maintained their survival advantage across all income levels.45

Table 1. Baseline characteristics by neighborhood SES and race

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Low SES(^a) (n=4432)</th>
<th>P Value</th>
<th>Higher SES(^a) (n=6595)</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean age, yr ± SD</td>
<td>25.7 ± 3.4</td>
<td>&lt;0.001</td>
<td>25.6 ± 3.4</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Mean BMI ± SD</td>
<td>27.6 ± 7.7</td>
<td>&lt;0.001</td>
<td>26.4 ± 5</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Men</td>
<td>1194 (48.4)</td>
<td>&lt;0.001</td>
<td>1159 (54.0)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Hemodialysis</td>
<td>2324 (94.3)</td>
<td>&lt;0.001</td>
<td>2011 (93.2)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Urban(^b)</td>
<td>2045 (83.0)</td>
<td>0.03</td>
<td>1912 (89.6)</td>
<td>0.001</td>
</tr>
<tr>
<td>Primary cause of ESRD</td>
<td></td>
<td>&lt;0.001</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>549 (22.3)</td>
<td></td>
<td>435 (20.3)</td>
<td></td>
</tr>
<tr>
<td>GN(^c)</td>
<td>767 (31.1)</td>
<td></td>
<td>782 (36.4)</td>
<td></td>
</tr>
<tr>
<td>AIDS-associated nephropathy</td>
<td>169 (6.9)</td>
<td></td>
<td>95 (4.4)</td>
<td></td>
</tr>
<tr>
<td>Other</td>
<td>980 (39.7)</td>
<td></td>
<td>836 (38.9)</td>
<td></td>
</tr>
<tr>
<td>Comorbidities</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hypertension</td>
<td>2050 (83.2)</td>
<td>&lt;0.001</td>
<td>1801 (83.9)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>649 (26.3)</td>
<td>&lt;0.001</td>
<td>491 (22.2)</td>
<td></td>
</tr>
<tr>
<td>Congestive heart failure</td>
<td>267 (10.8)</td>
<td>&lt;0.001</td>
<td>205 (9.5)</td>
<td></td>
</tr>
<tr>
<td>Atherosclerotic heart disease</td>
<td>26 (1.6)</td>
<td>0.47</td>
<td>25 (1.3)</td>
<td>0.58</td>
</tr>
<tr>
<td>Other cardiac disease</td>
<td>130 (5.3)</td>
<td>&lt;0.01</td>
<td>106 (4.9)</td>
<td>0.01</td>
</tr>
<tr>
<td>Cerebrovascular disease</td>
<td>41 (1.7)</td>
<td>0.28</td>
<td>41 (1.9)</td>
<td>0.02</td>
</tr>
<tr>
<td>Chronic obstructive pulmonary disease</td>
<td>26 (1.0)</td>
<td>0.23</td>
<td>12 (0.6)</td>
<td>0.65</td>
</tr>
<tr>
<td>Peripheral vascular disease</td>
<td>52 (2.1)</td>
<td>0.30</td>
<td>41 (1.9)</td>
<td>0.18</td>
</tr>
<tr>
<td>Malignancy</td>
<td>19 (0.8)</td>
<td>0.60</td>
<td>13 (0.6)</td>
<td>0.05</td>
</tr>
<tr>
<td>Alcohol dependence</td>
<td>20 (0.9)</td>
<td>0.84</td>
<td>11 (0.5)</td>
<td>0.83</td>
</tr>
<tr>
<td>Drug dependence</td>
<td>78 (3.2)</td>
<td>0.26</td>
<td>54 (2.5)</td>
<td>0.50</td>
</tr>
<tr>
<td>Tobacco use</td>
<td>190 (7.7)</td>
<td>0.14</td>
<td>128 (6.0)</td>
<td>0.09</td>
</tr>
<tr>
<td>Needs assistance for ADL</td>
<td>115 (4.6)</td>
<td>0.76</td>
<td>74 (3.5)</td>
<td>0.75</td>
</tr>
<tr>
<td>Insurance</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Private</td>
<td>406 (16.5)</td>
<td>0.004</td>
<td>555 (25.8)</td>
<td></td>
</tr>
<tr>
<td>Medicaid</td>
<td>1162 (47.1)</td>
<td>&lt;0.001</td>
<td>808 (37.6)</td>
<td></td>
</tr>
<tr>
<td>No medical insurance</td>
<td>723 (29.3)</td>
<td>0.82</td>
<td>599 (27.9)</td>
<td>0.003</td>
</tr>
<tr>
<td>Seen by a nephrologist pre-ESRD</td>
<td></td>
<td>0.45</td>
<td></td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>No</td>
<td>1092 (44.3)</td>
<td></td>
<td>914 (42.5)</td>
<td></td>
</tr>
<tr>
<td>Unknown</td>
<td>295 (12.0)</td>
<td></td>
<td>251 (11.7)</td>
<td></td>
</tr>
<tr>
<td>Type of access (hemodialysis patients)</td>
<td></td>
<td>0.04</td>
<td></td>
<td>0.001</td>
</tr>
<tr>
<td>Arteriovenous fistula</td>
<td>151 (6.5)</td>
<td></td>
<td>137 (6.8)</td>
<td></td>
</tr>
<tr>
<td>Arteriovenous graft</td>
<td>57 (2.4)</td>
<td></td>
<td>37 (1.9)</td>
<td></td>
</tr>
<tr>
<td>Catheter</td>
<td>2096 (90.2)</td>
<td></td>
<td>1811 (90.5)</td>
<td></td>
</tr>
<tr>
<td>Unknown</td>
<td>20 (0.9)</td>
<td></td>
<td>16 (0.8)</td>
<td></td>
</tr>
</tbody>
</table>

ADL, activities of daily living.

\(^a^\)Low neighborhood SES is a neighborhood with \(\geq 20%\) below poverty. Higher neighborhood SES is a neighborhood with \(<20%\) living below poverty.

\(^b^\)Data are shown as number (%) unless otherwise specified.

\(^c^\)Patients with rural-urban communicating area information (n=10,986).

\(^d^\)Systemic lupus erythematosus: low SES: blacks, 300 (39); whites, 143 (19); higher SES: blacks, 290 (37); whites, 298 (16); FSGS: low SES: blacks, 248 (32); whites, 165 (22); higher SES: blacks, 258 (33); whites, 326 (18).
Rodriguez et al. also found no difference in survival for blacks living in zip codes where 75% or more of residents were black compared with zip codes where less than 10% of the residents were black. Kimmel et al. found that having a very low income was associated with worse survival for both blacks and whites; however, blacks maintained a survival advantage over whites. None of these studies evaluated race survival differences among younger adults. Our findings confirm the results by Kucirka et al., showing that the race survival paradox is not present among young adults initiating dialysis; we extend them by showing the substantial and differential relation of neighborhood SES with race disparities in survival among young adults.

In the setting of dialysis, social and environmental determinants may be particularly influential on the health of young blacks. In our study, baseline access to care and quality of care measures (i.e., the provision of health insurance, initiation of dialysis with appropriate vascular access, and receipt of pre-ESRD care by a nephrologist) were generally poor and similar between blacks and whites of low and higher SES. Nonetheless, these pre-ESRD measures may still contribute to the disparate findings in mortality that we observed by race and level of neighborhood SES. Although the access to care may not differ among young adult patients residing in low SES neighborhoods, challenging psychosocial circumstances for low SES young blacks (such as experiences of social stigmatization and discrimination or poor health literacy) may lead to greater distrust in medical institutions, potentially resulting in their underuse of available medical resources and lower medical adherence. These factors may contribute to uncontrolled diabetes and HIV as well as greater rates of dialysis initiations with a catheter and fewer conversions to arteriovenous access among young blacks with low SES. These factors may underlie the greater risk of deaths attributable to infections compared with young whites. Our findings of better survival in higher SES young blacks compared with low SES young blacks would suggest that improved access to care, likely better health literacy, and potentially less social stigmatization and discrimination could at least partially attenuate some of the postulated effects of low SES on the health for young blacks.

Our study has limitations. First, both low individual SES and low neighborhood SES may contribute to poor outcomes among young blacks compared with young whites. The
USRDS registry does not include individual-level SES data, and we were, therefore, not able to fully explore the potential interactions between these SES indicators. However, prior epidemiologic studies provide rationale for the use of area-based measures to reflect SES in the absence of other socioeconomic data.\textsuperscript{11,35,58} Second, the lack of data available on AKI limited our ability to fully assess it as a contributor to our findings. The prevalence of potential indicators of AKI, specifically acute interstitial nephritis and tubular necrosis as primary causes of ESRD, however, was very low and therefore, unlikely to be a major contributor to our overall results. Furthermore, to avoid misclassification of dialysis-requiring AKI cases as ESRD cases, we excluded patients with recovery at any point during follow-up. Third, although we made a distinction between non-Hispanic and Hispanic individuals (with the recognition that Hispanics also have better survival on dialysis),\textsuperscript{29} there were participants in our study with unspecified ethnicity, especially among whites. We also could not account for unmeasured confounders that could impact racial differences in ESRD risk and survival (e.g., genetic susceptibility, patients’ attitudes and beliefs, ecological toxins, medication adherence, and medical treatment). As with all observational cohort studies, there is possibility of residual confounding, and causality is difficult to establish. Notwithstanding these limitations, our study is the first national study to examine the influence of neighborhood SES on race differences in mortality among young dialysis patients. We examined a contemporary cohort of incident patients over a fairly short timeframe, which likely limited the influence of secular trends in ESRD care. We were able to adjust for many important confounders in our analyses and accounted for racial disparities in renal transplantation in this young cohort of individuals with ESRD. Finally, our data linkage to the National Death Index (NDI) facilitated a robust descriptive analysis of cause-specific mortality that has not previously been possible.

In summary, the survival differences among young adult blacks compared with young adult whites are most striking in low SES neighborhoods. In addition to practices that improve transplantation rates, predialysis access to care, and predialysis health, additional studies are needed to identify modifiable factors contributing to the higher mortality, especially among young blacks on dialysis residing in low SES neighborhoods.

### CONCISE METHODS

#### Study Population and Design
We obtained our data from the USRDS, a national registry of ESRD patients funded by the National Institute of Diabetes and Digestive and Kidney Diseases in conjunction with the CMS. Patients who develop ESRD are included in this registry and assigned a unique identifier that is linked to Medicare and a Social Security Master Death file. Information on patients’ demographics, cause of ESRD, comorbidities, Medicare Part A institutional claims, Medicare Part B physician/supplier claims, and outcomes, such as hospitalizations and mortality, are captured in this database.\textsuperscript{1} In this study, we also linked USRDS data to NDI data from the Centers for Disease Control and Prevention.\textsuperscript{59}

We conducted a retrospective cohort study that included all young adult non-Hispanic white and black patients ages 18–30 years who initiated dialysis and did not spontaneously recover renal function between January 1, 2006, and December 31, 2009. The patient time at risk was defined as day 91 after ESRD diagnosis until death, renal transplantation, or the end of the study (December 31, 2010). Patients who died or received a renal transplant within the first 90 days were excluded, which typical for most observational analyses of USRDS data on patients <65 years of age, because these patients are not Medicare-eligible within the first 90 days.\textsuperscript{35,60} We also excluded individuals without a United States residential zip code and individuals for whom socioeconomic information could not be obtained (Figure 1). Our study qualified for an exemption under the Code of Federal Regulations, Protection of Human Subjects (45 CFR 46.101[b]) by the Institutional Review Board at Johns Hopkins School of Medicine.

#### Data Sources and Study Variables

##### Patient Level
We derived patient-level characteristics from the CMS 2728 Medical Evidence Form—a federally mandated form that must be completed and signed by the supervising physician within 45 days of dialysis initiation. Our primary exposure was race (non-Hispanic white versus black). We determined covariates \textit{a priori} based on their association with the primary exposure (race) and their possible association with the primary outcome (all-cause mortality) as well as to allow for comparison with similar population-based studies.\textsuperscript{37} We collected information on the following covariates at ESRD onset (baseline): age, sex, race and ethnicity, BMI, zip code, dialysis modality, insurance status and type, pre-ESRD nephrology referral, primary cause of ESRD, type of dialysis access, and presence or absence of

### Table 2. Adjusted relative risk of death for blacks versus whites by level of neighborhood SES

<table>
<thead>
<tr>
<th>Model</th>
<th>Low SES (n=4432)</th>
<th>Higher SES (n=6595)</th>
<th>P Interaction</th>
<th>Low SES (n=4432)</th>
<th>Higher SES (n=6595)</th>
<th>P Interaction</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>1.65 (1.38 to 1.97)</td>
<td>1.18 (1.01 to 1.38)</td>
<td>&lt;0.001</td>
<td>1.79 (1.50 to 2.14)</td>
<td>1.40 (1.17 to 1.60)</td>
<td>0.03</td>
</tr>
<tr>
<td>2</td>
<td>1.44 (1.19 to 1.73)</td>
<td>1.09 (0.93 to 1.28)</td>
<td>0.02</td>
<td>1.51 (1.26 to 1.83)</td>
<td>1.24 (1.06 to 1.45)</td>
<td>0.10</td>
</tr>
<tr>
<td>3</td>
<td>1.45 (1.21 to 1.74)</td>
<td>1.09 (0.93 to 1.28)</td>
<td>0.02</td>
<td>1.53 (1.27 to 1.85)</td>
<td>1.24 (1.05 to 1.46)</td>
<td>0.09</td>
</tr>
<tr>
<td>4\textsuperscript{a}</td>
<td>1.46 (1.21 to 1.74)</td>
<td>1.11 (0.94 to 1.30)</td>
<td>0.03</td>
<td>1.53 (1.28 to 1.85)</td>
<td>1.26 (1.07 to 1.48)</td>
<td>0.10</td>
</tr>
</tbody>
</table>

\textsuperscript{Model 1, age+sex; model 2, model 1+BMI+primary cause of ESRD and baseline comorbidities; model 3, model 2+access to care factors.}

\textsuperscript{aModel 4, model 3+rurality (n=10,986).}
comorbidities (hypertension, diabetes mellitus, atherosclerotic heart disease, congestive heart failure, other cardiac disease, cerebrovascular disease, chronic obstructive pulmonary disease, peripheral vascular disease, current tobacco use, alcohol dependence, drug dependence, and need for assistance with activities of daily living).

**Zip Code Level**
We merged patient-level USRDS zip code data with the US Census data to obtain community-level SES for each patient’s zip code of residence. We obtained zip code-level poverty information from the US Census Bureau American Community Survey (ACS) 5-year estimates from 2007 to 2011. The ACS is a nationwide survey with an annual sample size of approximately 3 million addresses across the United States and Puerto Rico, and it includes both housing units and group quarters (e.g., nursing facilities and prisons). Poverty status is determined by comparing annual income with a set of dollar values termed poverty thresholds that varied by family size, the number of children, and the age of the head of the household. The poverty thresholds are updated annually to account for changes in the cost of living using the Consumer Price Index. If the total household income is less than the threshold appropriate for the family, then every member of the family is considered to be living below the poverty level. For people not living in families, poverty status is determined by comparing the individual’s income with his or her poverty threshold. For comparison with the US Census Bureau literature, we categorized zip codes into four categories: I (<13.8% living below poverty), II (13.8%–19.9% living below poverty), III (20%–39.9% living below poverty), and IV (40% or more living below poverty). We defined low neighborhood SES as a zip code with 20% or more of the residents living below the federal poverty level, consistent with the federal definition. To determine rurality, we used the rural–urban commuting area (RUCA) code version 2.0.

**Ascertainment of the Outcomes**
The primary outcome was all-cause mortality. We determined the date of death from the USRDS data, and we determined the cause of death through linked NDI International Classification of Disease (ICD-10-CM) codes. We classified causes of death into cardiovascular, infection, or other as follows: (1) cardiovascular: I05–I15, I20–I25.9, I33–I37, I42–I51, I60–I69, I70–I79, I80–I89, I90, and R02; (2) infectious: A00–B99, E06.0, G00–G08, I00–I01, I33, I38, J00–J06, J10–J18, J20–J22, J36, J39.0, J39.1, J44.0, J85–J86, K35, K63.0, K65.0, K65.9, K80.0, K80.3, K80.4, K81, K83.0, L00–L08, M00–M02, M86, N30.0, N30.8, N41.0, N41.2, N41.3, O03.0, O03.5, O04.5, O07.0, O08.0, O75.3, O85–O86, R57.2, T80.2, T81.4, T82.6–T82.7, T83.5–T83.6, T84.5–T84.7, T85.7, T87.4; and T88.0; and (3) other (all other ICD-10 codes).

**Statistical Analyses**

**Descriptive Data Analyses**
We described patients’ characteristics at dialysis initiation stratified by neighborhood SES and race. We calculated means and SDs for age and BMI and performed t tests (for continuous variables) and chi-squared tests (for categorical variables) to compare distributions between the groups. We generated Kaplan–Meier curves by race and SES status.

**Cox Proportional Hazards and Competing Risk Models**
We quantified the racial differences in mortality between patients living in low SES and higher SES neighborhoods using multivariable Cox proportional hazards models (estimating aSHRs), which adjusted for patients’ baseline demographics, clinical characteristics, access to care factors, and rurality. We estimated differences in the association of race and mortality according to neighborhood SES using interaction terms. We also performed parallel competing risk regressions (estimating aSHRs) according to the methods of Fine and Gray to assess the influence of differential censoring secondary to lower transplantation rates for blacks compared with whites. We adjusted for the presence of AIDS nephropathy in all our primary models. Although predominantly observed in blacks, the overall prevalence was low (2.5%), and, therefore, our assumption was that excluding patients with AIDS nephropathy would not significantly change our risk estimates and may decrease the power of our study to detect interaction. To test this assumption, we performed a sensitivity analysis excluding patients with a diagnosis of AIDS nephropathy. We also limited our analyses to hemodialysis patients and adjusted for patients’ hemodialysis access type to explore the influence of dialysis modality and access type (i.e., catheter versus graft or fistula for hemodialysis).

**Missing or Unknown Covariates Information**
The number of patients missing a cause of death was similar in both the Social Security Master Death file (30%) and NDI linked file (28%). Information on BMI was missing in 230 (2%) patients and imputed using Rubin’s multiple technique (n=5 iterations). For categorical covariates in which information was classified as unknown, we created a separate category for these patients and included them in our model. We excluded patients with indeterminate RUCA information (n=41 or 0.4%) from the statistical analysis.

**Model Testing and Statistical Significance**
We assessed the proportional hazards assumption both graphically and statistically using Schoenfeld residuals. We defined statistical significance as P<0.05 using two-tailed tests. We performed all analyses using multiprocessor Stata version 11.0/MP (StataCorp, College Station, TX).

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T.J. and L.E.B. had full access to all the data in the study and take responsibility for the integrity of the data and the accuracy of the data analysis. The data reported here have been supplied by the US Renal Data System. The interpretation and reporting of these data are the responsibility of the authors and in no way should be seen as an official policy or interpretation of the US Government.
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