Higher Serum Creatinine Concentrations in Black Patients with Chronic Kidney Disease: Beyond Nutritional Status and Body Composition

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Background and objectives: Serum creatinine concentrations tend to be higher in black than white individuals and people of other races or ethnicities. These differences have been assumed to be largely related to race-related differences in body composition, especially muscle mass.

Design, setting, participants, & measurements: In a diverse population of hemodialysis patients, we compared mean serum creatinine concentrations in black versus nonblack patients, adjusting for case mix (age, gender, diabetes, and dialysis vintage), body size (height, weight), laboratory parameters of nutritional status (albumin, predialysis blood urea nitrogen, transferrin, phosphorus, glucose), dialysis dosage (urea reduction ratio), and parameters of bioelectrical impedance (resistance and reactance), proxies for body composition.

Results: Adjusted mean serum creatinine concentrations were significantly higher in black versus nonblack patients (11.7 versus 10.0 mg/dl; P < 0.0001). Black patients were roughly four-fold more likely to have a serum creatinine concentration >10 mg/dl and six-fold more likely to have a serum creatinine concentration >15 mg/dl. Higher serum creatinine concentrations were associated with a lower relative risk for death (0.93; 95% confidence interval 0.88 to 0.98 per mg/dl); the association was slightly more pronounced among nonblack patients.

Conclusions: Serum creatinine concentrations are significantly higher in black compared with nonblack hemodialysis patients; these differences are not readily explained by differences in nutritional status or body composition.


Several studies have indicated that black patients with mild to moderate chronic kidney disease (CKD) are more likely to experience an increase in serum creatinine (Scr) over time than white patients (1) and that similar control of risk factors such as blood pressure does not prevent a rise in Scr in black patients as it does in white patients (2). This has led to an age- and gender-adjusted incidence of treated ESRD that is more than three-fold higher among black than white patients (3); however, black individuals have long been noted to have higher serum creatinine levels than white individuals that may be independent of kidney function. In a representative sample of the general US population, the Third National Health and Nutrition Examination Survey (NHANES III) documented that black individuals have higher Scr concentrations than white individuals for any given age and gender stratum, and racial differences in Scr persist along the entire spectrum of CKD (4).

The most commonly used equation to estimate GFR includes black race because of these pervasive and consistent differences (5). Scr concentrations are higher in black dialysis patients, a finding often attributed to differences in somatic muscle mass. In fact, the reason for these racial differences in Scr is unknown, and it raises several questions, including whether a given Scr concentration might have different clinical implications among different racial groups.

Determinants of Scr concentration include not only the GFR but also the rate of creatinine generation and tubular secretion, the dietary absorption of creatine, certain medications, and interlaboratory and intraindividual variability (4,6). Several studies have documented differences in body composition among black and white individuals (7–9) lending support to the contention that racial disparities in Scr are related to increased creatinine generation in black individuals as a result of increased muscle mass (8–9); however, few data have directly confirmed or refuted this contention. One study examining this issue used anthropometric measurements (e.g., skinfold thickness) (14), which are known to be less reliable than other measures of lean body mass (15).

Because the Scr concentration is largely influenced by kid-
ney function, studying differences in SCr concentration in a population with little or no kidney function might allow investigators to determine whether differences in SCr concentration across subpopulations were evident and potentially related to factors other than kidney function. SCr concentrations are known to be higher in black compared with nonblack patients on dialysis. We believe that many clinicians and investigators have assumed that higher SCr in black individuals was explained by the younger mean age at onset of ESRD with presumed preservation of muscle mass. Nonetheless, we have observed a similar association between race and SCr even among frail elderly patients with obvious sarcopenia.

We conducted this study to determine whether we could account for race-related differences in SCr concentration adjusting not only for age, gender, and parameters of nutritional status but also for body composition (and creatinine generation) using bioelectrical impedance analysis. We hypothesized that adjustment for body composition would eliminate or attenuate the large racial differences in SCr observed among hemodialysis patients.

Materials and Methods

Study participants were 3009 prevalent adult hemodialysis patients from 101 free-standing Fresenius Medical Care North America (FMCNA) dialysis units across the United States. Inclusion criteria were age ≥18 yr and thrice-weekly in-center hemodialysis for ≥3 mo. Patients with an amputation above the transmetatarsal site were excluded from participation. Bioelectrical impedance analysis (BIA; BIA Quantum, RJL Systems, Inc., Clinton Twp., MI) was performed before and after a midweek dialysis session during the first 6 mo of 1995. Briefly, an inner electrode was attached to the dorsal surface of the wrist on the arm without an arteriovenous fistula or graft. An outer electrode was placed on the dorsal surface of the third metacarpal bone. A second pair of electrodes was positioned on the anterior surface of the ipsilateral ankle and the dorsal surface of the third metatarsal bone. A single-frequency low-amplitude imperceptible current (800 μA at 50 kHz) was introduced via the electrodes on the hand and the foot. The electrodes at the wrist and the ankle detected the voltage decrease. Phase angle (the arc tangent of the reactance to resistance ratio) was calculated in radians using bioelectrical impedance analysis. We hypothesized that adjustment for body composition would eliminate or attenuate the large racial differences in SCr observed among hemodialysis patients.

Materials and Methods

Continuous variables were described as mean ± SD or median and interquartile range and compared using t test or the Wilcoxon rank sum test, where appropriate. Categorical variables were described as proportions. Differences in the mean SCr concentration by race (black versus nonblack) and gender were determined using general linear models. Sequential linear regression models were fitted adjusting for case mix (age, gender, diabetes, and dialysis vintage), body size (height, predialysis weight), laboratory parameters of nutritional status (albumin, predialysis blood urea nitrogen, transferrin, phosphorus, glucose), dialysis dosage (urea reduction ratio), and parameters of bioelectrical impedance (resistance and reactance), proxies for body composition. We used logistic regression to determine the likelihood of discrete, high values for SCr, again focusing on race as the key independent variable. Odds ratios (OR) and 95% confidence intervals (95% CI) were calculated from model parameter coefficients and standard error, respectively. For logistic regression, model discrimination was determined using the area under the receiver operating characteristic (ROC) curve. Calibration was assessed using the Hosmer-Lemeshow goodness-of-fit test.

Finally, we evaluated the association between SCr and survival using proportional hazards regression, and fitted a race × SCr interaction term to determine whether the prognostic significance of the SCr concentration was race dependent. Patients were followed for up to 18 mo after BIA testing. Patients who underwent kidney transplantation (n = 82; 2.7%), recovered kidney function (n = 18; 0.6%), transferred dialysis facilities (n = 287; 9.7%), withdrew from dialysis (n = 42; 1.3%), or were lost to follow-up for unknown reasons (n = 8; 0.3%) were censored.

Two-tailed P < 0.05 was considered statistically significant. Statistical analyses were conducted using SAS 9.1 (SAS Institute, Cary, NC). The project was approved by the institutional review board at FMCNA. All patients provided written informed consent.

Results

Race and SCr Concentrations

Patient characteristics stratified by race (black versus nonblack) are presented in Table 1. Black patients were younger and had significantly higher reactance, phase angle, and calculated total body water and body cell mass compared with nonblack patients. The mean SCr was 10.8 ± 3.5 mg/dl; the median SCr was 10.6 mg/dl with 10 to 90% range of 6.6 to 15.5 mg/dl. Mean SCr concentrations were significantly higher in black patients (12.1 ± 3.6 versus 9.6 ± 3.0 mg/dl in nonblack patients; P < 0.0001) and remained significantly higher after adjustment for case mix (age, gender, diabetes, and vintage, 11.9 versus 9.9 mg/dl; P < 0.0001) and after adjustment for nutritional variables (including serum albumin, phosphorus, glucose, predialysis blood urea nitrogen, and transferrin), body weight, and reactance and resistance by BIA (11.7 versus 10.0 mg/dl; P < 0.0001). Table 2 shows linear regression results including all variables. Adjusted mean SCr for Hispanic patients (n = 190; 10.8 mg/dl) and patients of other race or ethnicity (n = 34; 10.3 mg/dl) were intermediate between black (n = 1371; 11.7 mg/dl) and white patients (n = 1304; 9.9 mg/dl). The results were identical when adjusted further for the dosage of dialysis, whether expressed as the urea reduction ratio, Kt/Vurea, or Kt urea. When we restricted the analytic sample to patients who were unlikely to have significant residual kidney function (n = 1780 with vintage ≥2 yr), race-related differences in adjusted mean SCr concentration were still evident and of similar magnitude (12.5 versus 10.7 mg/dl; P < 0.0001).

Black patients were significantly more likely to have a SCr >10 mg/dl (adjusted OR 3.7; 95% CI 3.0 to 4.6). The area under the model’s ROC curve was 0.88, indicating excellent discrimination, and was well calibrated (Hosmer-Lemeshow$\chi^2$, P = 0.69). Only 66 (4.3%) of the 1528 nonblack patients had SCr >15 mg/dl, in contrast to 292 (21.3%) of the 1371 black patients (P <
Adjusted for case mix, nutritional variables, and body composition, black patients were six-fold more likely to have SCr concentrations $\geq 15$ mg/dl (adjusted OR 6.0; 95% CI 4.2 to 8.5). The area under the second model’s ROC curve was 0.93 and was also well calibrated (Hosmer-Lemeshow $\chi^2$, $P = 0.30$).

SCr and Mortality

There were 342 deaths, corresponding to 1-yr mortality rates of 9% among black patients and 14% among nonblack patients. Higher SCr concentrations were associated with a lower relative risk (RR) for death (RR 0.93; 95% CI 0.88 to 0.98 per mg/dl), adjusted for age, gender, race, diabetes, vintage, and the laboratory proxies of nutritional status and bioimpedance parameters included in Table 2 ($P = 0.008$). The association between SCr and mortality was race-dependent; the RR per mg/dl SCr was 0.91 (95% CI 0.84 to 0.98 per mg/dl) in nonblack patients and 0.93 (0.86 to 1.00 mg/dl) in black patients (race $\times$ SCr interaction $P = 0.03$). The association between SCr and mortality remained significant among patients with vintage $\geq$2 yr (RR 0.89; 95% CI 0.84 to 0.96 per mg/dl).

### Table 1. Baseline patient characteristics by race

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Black ($n = 1371$)</th>
<th>Nonblack ($n = 1528$)</th>
<th>$P$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (yr; mean ± SD)</td>
<td>56.8 ± 14.8</td>
<td>63.4 ± 15.4</td>
<td>$&lt;0.0001$</td>
</tr>
<tr>
<td>Female gender (%)</td>
<td>50</td>
<td>44</td>
<td>0.0020</td>
</tr>
<tr>
<td>Vintage (yr; median [IQR])</td>
<td>2.9 (1.5 to 5.4)</td>
<td>2.4 (1.2 to 4.5)</td>
<td>$&lt;0.0001$</td>
</tr>
<tr>
<td>Diabetes (%)</td>
<td>38</td>
<td>35</td>
<td>0.1400</td>
</tr>
<tr>
<td>Creatinine (mg/dl; mean ± SD)</td>
<td>12.1 ± 3.6</td>
<td>9.6 ± 3.0</td>
<td>$&lt;0.0001$</td>
</tr>
<tr>
<td>Albumin (g/dl; mean ± SD)</td>
<td>3.88 ± 0.37</td>
<td>3.83 ± 0.36</td>
<td>$&lt;0.0001$</td>
</tr>
<tr>
<td>Predialysis BUN (mg/dl; mean ± SD)</td>
<td>68 ± 17</td>
<td>68 ± 17</td>
<td>0.4700</td>
</tr>
<tr>
<td>Cholesterol (mg/dl; mean ± SD)</td>
<td>178 ± 45</td>
<td>175 ± 46</td>
<td>0.0400</td>
</tr>
<tr>
<td>Glucose (mg/dl; mean ± SD)</td>
<td>140 ± 68</td>
<td>150 ± 73</td>
<td>0.0001</td>
</tr>
<tr>
<td>Hemoglobin (g/dl; mean ± SD)</td>
<td>10.1 ± 1.2</td>
<td>10.5 ± 1.0</td>
<td>$&lt;0.0001$</td>
</tr>
<tr>
<td>URR (%; mean ± SD)</td>
<td>64.0 ± 7.2</td>
<td>66.0 ± 6.5</td>
<td>$&lt;0.0001$</td>
</tr>
<tr>
<td>Weight (kg; mean ± SD)</td>
<td>76.8 ± 19.5</td>
<td>72.0 ± 17.4</td>
<td>$&lt;0.0001$</td>
</tr>
<tr>
<td>Quetelet’s index (kg/m²; mean ± SD)</td>
<td>27.2 ± 2.1</td>
<td>27.2 ± 1.8</td>
<td>0.0200</td>
</tr>
<tr>
<td>Resistance (Ω; mean ± SD)</td>
<td>490 ± 98</td>
<td>504 ± 100</td>
<td>0.0004</td>
</tr>
<tr>
<td>Reactance (Ω; mean ± SD)</td>
<td>45 ± 15</td>
<td>38 ± 12</td>
<td>$&lt;0.0001$</td>
</tr>
<tr>
<td>Phase angle (°; mean ± SD)</td>
<td>5.3 ± 1.8</td>
<td>4.4 ± 1.6</td>
<td>$&lt;0.0001$</td>
</tr>
<tr>
<td>Total body water (kg; mean ± SD)</td>
<td>41.6 ± 9.5</td>
<td>40.1 ± 9.0</td>
<td>$&lt;0.0001$</td>
</tr>
<tr>
<td>Body cell mass (kg; mean ± SD)</td>
<td>27.1 ± 6.2</td>
<td>25.2 ± 5.4</td>
<td>$&lt;0.0001$</td>
</tr>
</tbody>
</table>

$^a$BUN, blood urea nitrogen; IQR, interquartile range; URR, urea reduction ratio.

### Table 2. Linear regression with SCr as the dependent variable

<table>
<thead>
<tr>
<th>Variable</th>
<th>Parameter Estimate</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Black</td>
<td>1.73500</td>
<td>1.56000 to 1.91000</td>
</tr>
<tr>
<td>Age</td>
<td>-0.04480</td>
<td>-0.05080 to -0.03870</td>
</tr>
<tr>
<td>Male</td>
<td>0.936</td>
<td>0.75500 to 1.11600</td>
</tr>
<tr>
<td>Diabetes</td>
<td>-0.90000</td>
<td>-1.10600 to -0.69400</td>
</tr>
<tr>
<td>Vintage</td>
<td>0.12600</td>
<td>0.10300 to 0.14900</td>
</tr>
<tr>
<td>Albumin</td>
<td>1.44700</td>
<td>1.20200 to 1.69200</td>
</tr>
<tr>
<td>Phosphorus</td>
<td>0.29000</td>
<td>0.23200 to 0.34400</td>
</tr>
<tr>
<td>Glucose</td>
<td>-0.00530</td>
<td>-0.00671 to -0.00389</td>
</tr>
<tr>
<td>BUN</td>
<td>0.051300</td>
<td>0.04620 to 0.05640</td>
</tr>
<tr>
<td>Transferrin</td>
<td>-0.00423</td>
<td>-0.00612 to -0.00234</td>
</tr>
<tr>
<td>Weight</td>
<td>0.01810</td>
<td>0.01270 to 0.02350</td>
</tr>
<tr>
<td>Reactance</td>
<td>0.03340</td>
<td>0.02660 to 0.04020</td>
</tr>
<tr>
<td>Resistance</td>
<td>-0.00293</td>
<td>-0.00399 to -0.00134</td>
</tr>
</tbody>
</table>

$^a$Effects estimated as age (per year), vintage (per year), albumin (per g/dl), phosphorus (per mg/dl), glucose (per mg/dl), BUN (per mg/dl), transferrin (per mg/dl), weight (per kg), reactance (per Ω), and resistance (per Ω). CI, confidence interval; SCr, serum creatinine.
**Discussion**

The Institute of Medicine’s 2002 report on racial and ethnic disparities in health care was an exhortation to continue—and, indeed, amplify—efforts to eliminate these disparities (16). Racial disparities in kidney disease have been well documented: Black patients are disproportionately overrepresented in the treated ESRD population, (3), and although the prevalence of mild to moderate CKD is roughly equivalent in black and white patients (1,2), black patients seem more likely to progress to ESRD, even after accounting for comorbid conditions (e.g., diabetes, hypertension, obesity) and differences in competing mortality risks. Black patients are less likely to receive kidney transplantation than white patients (3,16,17) and fare worse after transplantation (18); however, black patients with ESRD have a lower overall risk for death than their white counterparts (3,17,19–21) despite many factors that are associated with higher risk, including fewer transplants (3,16,17), adverse socioeconomic factors, a higher prevalence of anemia (21–23), fewer arteriovenous fistulas (24–26), and lower dialysis dosages (19–21). Some have attributed the race survival paradox in hemodialysis patients to better nutrition among black patients (10,11,19,22,27–29), as evidenced in part by higher SCR concentrations, which are assumed to be a proxy for higher muscle mass (10,11,29); however, few studies have examined whether higher SCR concentrations in black patients can be explained by differences in muscle mass alone.

Although the reason(s) for this racial difference in SCr remains unclear, possible explanations fall into two major categories: Either black individuals generate more creatinine, or they eliminate less creatinine. The hypothesis that black individuals generate more creatinine is supported by reports of increased muscle mass and metabolic activity in black individuals (7,9,30). Furthermore, evidence of higher renal creatinine excretion per kilogram body weight in black compared with white individuals suggests that greater creatinine generation per kilogram body weight may contribute to the higher SCR observed (10,31). Avenues of decreased creatinine elimination in black individuals include a lower GFR, decreased renal tubular secretion of creatinine, or diminished extrarenal creatinine elimination. Evidence for a lower GFR in black individuals includes the parallel between the 3:1 ratio of the percentage of non-Hispanic black and non-Hispanic white individuals with SCR values of ≥2.0 mg/dl estimated in NHANES III (4) and the 3:1 ratio of the incidence rates of ESRD of black and white individuals in the United States (3). The possibility that higher SCR levels in black individuals reflect lower tubular secretion of creatinine, or diminished extrarenal creatinine elimination. Evidence for a lower GFR in black individuals includes the parallel between the 3:1 ratio of the percentage of non-Hispanic black and non-Hispanic white individuals with SCR values of ≥2.0 mg/dl estimated in NHANES III (4) and the 3:1 ratio of the incidence rates of ESRD of black and white individuals in the United States (3). The possibility that higher SCR levels in black individuals reflect lower tubular secretion of creatinine, or diminished extrarenal creatinine elimination. 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(>10 and >15 mg/dl). There are also important limitations. In general, healthier patients tend to participate in research studies, even those that require minimal effort or incur minimal risk; thus, we cannot rule out the possibility of selection effects. Although most hemodialysis patients have relatively little residual kidney function, our conclusions would be more definitive had we been able to adjust additionally for residual creatinine clearance. Proxies of inflammation, such as C-reactive protein, have also been shown to influence SCr concentrations and were not adjusted for. Additional measures of body composition, such as dual-energy x-ray absorptiometry, could confirm our contention that differences in muscle mass did not fully account for observed differences in SCr; however, BIA is the best method to estimate body composition in a free-standing dialysis unit, particularly when addressing a large population.

Conclusions
Among a diverse cohort of hemodialysis patients, we showed significantly higher SCr concentrations in black compared with nonblack individuals; differences that could not be extinguished by adjustment for laboratory proxies of nutritional status, body size, dialysis dosage, or estimates of body composition. In contrast to the non-ESRD population, where higher SCr concentrations (and lower SCr-derived estimates of GFR) are associated with increased risks for death and cardiovascular disease, (45–50), higher SCr concentrations are associated with a lower risk for death in hemodialysis patients. Understanding the reason(s) for race-related differences in SCr in the ESRD and CKD populations may help to clarify findings from epidemiologic studies showing major race-related differences in ESRD- and CKD-related outcomes.

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Disclosures
None.

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