# 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.**

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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 (10–14); 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-

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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 $\mu$A 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 and multiplied by $180/\pi$ (approximately 3.14159265) to covert radians to degrees. Reactance, resistance, phase angle, and the derived estimates of total body water and body cell mass were merged with the Patient Statistical Profile, a database with selected demographic, historic, and laboratory information on patients cared for at FMCNA-affiliated dialysis facilities. Predialysis BIA and laboratory values were used in the analysis. Laboratory values were means of the three months proceeding BIA testing. Patients with missing SCr concentrations ($n$ = 93; 3.1%) or whose survival time was unknown or uninterpretable ($n$ = 19; 0.6%) were excluded from the analysis. The final analytic sample included 2899 patients (1371 black and 1528 nonblack).

### *Statistical Analysis*

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, $Kt/V_{urea}$, 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 ($P <$

*Table 1.* Baseline patient characteristics by race[a]

| Characteristic | Black ($n$ = 1371) | Nonblack ($n$ = 1528) | $P$ |
|---|---|---|---|
| Age (yr; mean ± SD) | 56.8 ± 14.8 | 63.4 ± 15.4 | <0.0001 |
| Female gender (%) | 50 | 44 | 0.0020 |
| Vintage (yr; median [IQR]) | 2.9 (1.5 to 5.4) | 2.4 (1.2 to 4.5) | <0.0001 |
| Diabetes (%) | 38 | 35 | 0.1400 |
| Creatinine (mg/dl; mean ± SD) | 12.1 ± 3.6 | 9.6 ± 3.0 | <0.0001 |
| Albumin (g/dl; mean ± SD) | 3.88 ± 0.37 | 3.83 ± 0.36 | <0.0001 |
| Predialysis BUN (mg/dl; mean ± SD) | 68 ± 17 | 68 ± 17 | 0.4700 |
| Cholesterol (mg/dl; mean ± SD) | 178 ± 45 | 175 ± 46 | 0.0400 |
| Glucose (mg/dl; mean ± SD) | 140 ± 68 | 150 ± 73 | 0.0001 |
| Hemoglobin (g/dl; mean ± SD) | 10.1 ± 1.2 | 10.5 ± 1.0 | <0.0001 |
| URR (%; mean ± SD) | 64.0 ± 7.2 | 66.0 ± 6.5 | <0.0001 |
| Weight (kg; mean ± SD) | 76.8 ± 19.5 | 72.0 ± 17.4 | <0.0001 |
| Quetelet's index (kg/m$^2$; mean ± SD) | 27.2 ± 2.1 | 27.2 ± 1.8 | 0.0200 |
| Resistance (Ω; mean ± SD) | 490 ± 98 | 504 ± 100 | 0.0004 |
| Reactance (Ω; mean ± SD) | 45 ± 15 | 38 ± 12 | <0.0001 |
| Phase angle (°; mean ± SD) | 5.3 ± 1.8 | 4.4 ± 1.6 | <0.0001 |
| Total body water (kg; mean ± SD) | 41.6 ± 9.5 | 40.1 ± 9.0 | <0.0001 |
| Body cell mass (kg; mean ± SD) | 27.1 ± 6.2 | 25.2 ± 5.4 | <0.0001 |

[a]BUN, blood urea nitrogen; IQR, interquartile range; URR, urea reduction ratio.

0.0001). Adjusted for case mix, nutritional variables, and body composition, black patients were six-fold more likely to have SCr concentrations >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 × SCr interaction $P$ = 0.03). The association between SCr and mortality remained significant among patients with vintage ≥2 yr (RR 0.89; 95% CI 0.84 to 0.96 per mg/dl).

*Table 2.* Linear regression with SCr as the dependent variable[a]

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

[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 is supported by reports that creatinine clearance overestimates GFR in predominantly white populations (32–35) but does not seem to overestimate GFR in black individuals to nearly the same degree (32). Finally, there is some evidence that extrarenal metabolism of creatinine contributes significantly to creatinine elimination in patients with CKD. Mitch *et al.* (36) demonstrated that as SCr rose in individuals with CKD, an increasing fraction of creatinine was metabolized rather than excreted; the authors suggested that extrarenal clearance is usually "an undetectably small fraction of normal renal clearance . . . , [which] could explain the absence of demonstrable creatinine metabolism in normal subjects," whereas as much as two thirds of total daily creatinine excretion can occur by extrarenal excretion in patients with advanced CKD. We are not aware of studies that have examined racial differences in extrarenal creatinine metabolism. In summary, one (or several) of the reasons enumerated must be responsible for elevated SCr concentrations in black individuals; in this study, we tested the hypothesis that differences in body composition—and, by extension, creatinine generation—would account entirely for racial differences in SCr concentrations. Our results suggest that mechanisms other than increased creatinine production on the basis of larger muscle mass contribute to the higher SCr in black individuals. Moreover, although it is indisputable that higher SCr concentrations are associated with survival in dialysis patients, the reasons behind this association are unclear. Although many experts had previously assumed that higher SCr concentrations in dialysis patients indicated more robust musculature and improved nutritional status, thus explaining the association with survival, the link between higher SCr and survival may be due to other factors.

We used BIA rather than other methods to estimate body composition when examining the relation between race and SCr. Agamah *et al.* (14) used anthropometric measures (including height, weight, body mass index, and triceps and subscapular skinfold thicknesses) to estimate lean body mass in a cross-sectional study of 3983 people from the general population aged 5 to 26 yr. These investigators found that higher SCr concentrations in black compared with white individuals disappeared after accounting for differences in body composition. This contrasts directly with our finding that racial differences in SCr concentrations persisted after adjustment for differences in body composition as measured by BIA. We believe that there are two possible explanations for this discrepancy. First, the marked difference between the young, generally healthy individuals in the study by Agamah *et al.* and the older, maintenance hemodialysis patients in our study may be responsible for these conflicting results. Alternatively (and we believe more likely), differences in the precision and accuracy of BIA *versus* anthropometry account for the discrepant findings. Several studies have shown that in maintenance hemodialysis patients as well as in the general population, BIA and anthropometric measurements do not correlate well (37–39). Anthropometry is highly operator dependent, and estimates of body composition by caliper methods are even less reliable in populations with variable tissue turgor or edema, including obese, young, elderly, and ESRD patients (40–43). Several reports have indicated BIA to be more precise and accurate in estimating body composition than anthropometry when compared with a "gold standard" such as hydrodensitometry or dual-energy x-ray absorptiometry (39,44).

This study has several strengths, including a relatively large sample size, and adjustment for biochemical proxies of nutritional status as well as valid proxies of body composition. In addition to comparing mean SCr concentrations, we demonstrated a multifold higher likelihood of black individuals' demonstrating high and very high SCr concentrations

(>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.

## References

1. Shulman NB, Ford CE, Hall WD, Blaufox MD, Simon D, Langford HG, Schneider KA: Prognostic value of serum creatinine and effect of treatment of hypertension on renal function: Results from the Hypertension Detection and Follow-up Program. *Hypertension* 13[Suppl I]: I80–I93, 1989
2. Walker WG, Neaton JD, Cutler JA, Neuwirth R, Cohen JD: Renal function change in hypertensive members of the Multiple Risk Factor Intervention Trial: Racial and treatment effects. *JAMA* 268: 3085–3091, 1992
3. United States Renal Data System: *USRDS 2004 Annual Data Report: Atlas of End-Stage Renal Disease in the United States*, Bethesda, National Institutes of Health, National Institute of Diabetes and Digestive and Kidney Diseases, 2004
4. Jones CA, McQuillan GM, Kusek JW, Eberhardt MS, Herman WH, Coresh J, Salive M, Jones CP, Agodoa LY: Serum creatinine levels in the US population: Third National Health and Nutrition Examination Survey. *Am J Kidney Dis* 32: 992–999, 1998
5. Levey AS, Bosch JP, Lewis JB, Greene T, Rogers N, Roth D: A more accurate method to estimate glomerular filtration rate from serum creatinine: A new prediction equation. Modification of Diet in Renal Disease Study Group. *Ann Intern Med* 130: 461–470, 1999
6. Hsu CY, Chertow GM, Curhan GC: Methodological issues in studying the epidemiology of mild to moderate chronic renal insufficiency. *Kidney Int* 61: 1567–1576, 2002
7. Ortiz O, Russell MR, Daley TL, Baumgartner RN, Waki M, Lichtman S, Wang J, Pierson RN Jr, Heymsfield SB: Differences in skeletal muscle and bone mineral mass between black and white females and their relevance to estimates of body composition. *Am J Clin Nutr* 55: 8–13, 1992
8. Harsha DW, Frerichs RR, Berenson GS: Densitometry and anthropometry of Black and white children. *Hum Biol* 50: 261–280, 1978
9. Cohn SH, Abesamis C, Zanzi I, Aloia JF, Yasumura S, Ellis KJ: Body elemental composition: Comparison between black and white adults. *Am J Physiol* 232: E419–E422, 1977
10. Goldwasser P, Aboul-Magd A, Maru M: Race and creatinine excretion in chronic renal insufficiency. *Am J Kidney Dis* 30: 16–22, 1997
11. Lowrie EG, Lew NL, Huang WH: Race and diabetes as death risk predictors in hemodialysis patients. *Kidney Int* 42[Suppl 28]: S22–S31, 1992
12. Abuelo JG, Shemin D, Chazan JA: Serum creatinine concentration at the onset of uremia: Higher levels in black males. *Clin Nephrol* 37: 303–307, 1992
13. Jungers P, Lacoux B, Vassauly A, Barthélémy A, Vivien JN, Delons S: Serum creatinine concentration in chronic hemodialysis patients: Higher predialytic levels in black males [Letter]. *Clin Nephrol* 40: 118–119, 1993
14. Agamah ES, Webber LS, Lawrence M, Wattigney W, Berenson GS: Serum creatinine and its relation to cardiovascular disease risk variables in children and young adults from a biracial community. The Bogalusa Heart Study. *J Lab Clin Med* 116: 327–334, 1990
15. Lukaski HC, Johnson PE, Bolonchuk WW, Lykken GI: Assessment of fat-free mass using bioelectrical impedance measurements of the human body. *Am J Clin Nutr* 41: 810–817, 1985
16. Institute of Medicine: *Unequal Treatment: Confronting Racial and Ethnic Disparities in Health Care*, Washington DC, National Academy Press, 2002
17. Bleyer AF, Tell GS, Evans GW, Ettinger WH Jr, Burkart JM: Survival of patients undergoing renal replacement therapy in one center with special emphasis on racial differences. *Am J Kidney Dis* 28: 72–81, 1996
18. Chakkera HA, O'Hare AM, Johansen KL, Hynes D, Stroupe K, Colin PM, Chertow GM: Influence of race on kidney transplant outcomes within and outside the Department of Veterans Affairs. *J Am Soc Nephrol* 16: 269–277, 2005
19. Owen WF Jr, Chertow GM, Lazarus JM, Lowrie EG: Dose of hemodialysis and survival: Differences by race and sex. *JAMA* 280: 1764–1768, 1998
20. Leggat JE Jr, Orzol SM, Hulbert-Shearon TE, Golper TA, Jones CA, Held PJ, Port FK: Noncompliance in hemodial-

ysis: Predictors and survival analysis. *Am J Kidney Dis* 32: 139–145, 1998
21. Sehgal AR: Impact of quality improvement efforts on race and sex disparities in hemodialysis. *JAMA* 289: 996–1000, 2003
22. Frankenfield DL, Rocco MV, Frederick PR, Pugh J, McClellan WM, Owen WF Jr: Racial/ethnic analysis of selected intermediate outcomes for hemodialysis patients: Results from the 1997 ESRD Core Indicators Project. *Am J Kidney Dis* 34: 721–730, 1999
23. Ifudu O, Dawood M, Iofel Y, Valcourt JS, Friedman EA: Delayed referral of Black, Hispanic, and older patients with chronic renal failure. *Am J Kidney Dis* 33: 728–733, 1999
24. Allon M, Ornt DB, Schwab SJ, Rasmussen C, Delmez JA, Greene T, Kusek JW, Martin AA, Minda S: Factors associated with the prevalence of arteriovenous fistulas in hemodialysis patients in the HEMO Study. *Kidney Int* 58: 2178–2185, 2000
25. Stehman-Breen CO, Sherrard DJ, Gillen D, Caps M: Determinants of type and timing of initial permanent hemodialysis vascular access. *Kidney Int* 57: 639–645, 2000
26. Reddan D, Klassen P, Frankenfield DL, Szczech L, Schwab S, Coladonato J, Rocco M, Lowrie EG, Owen WF Jr: National profile of practice patterns for hemodialysis vascular access in the United States. *J Am Soc Nephrol* 13: 2117–2124, 2002
27. Fleischmann E, Teal N, Dudley J, May W, Bower JD, Salahudeen AK: Influence of excess weight on mortality and hospital stay in 1346 hemodialysis patients. *Kidney Int* 55: 1560–1567, 1999
28. Morris D, Samore MH, Pappas LM, Ramkummar M, Beddhu S: Nutrition and racial differences in cardiovascular events and survival in elderly dialysis patients. *Am J Med* 118: 671–675, 2005
29. Tanna MM, Vonesh EF, Korbet SM: Patient survival among incident peritoneal dialysis and hemodialysis patients in an urban setting. *Am J Kidney Dis* 36: 1175–1182, 2000
30. Ama PF, Simoneau JA, Boulay MR, Serresse O, Thériault G, Bouchard C: Skeletal muscle characteristics in sedentary black and Caucasian males. *Am J Physiol* 61: 1758–1761, 1986
31. James GD, Sealey JE, Alderman M, Ljungman S, Mueller FB, Pecker MS, Laragh JH: A longitudinal study of urinary creatinine and creatinine clearance in normal subjects: Race, sex, and age differences. *Am J Hypertens* 1: 124–131, 1988
32. Coresh J, Toto RD, Kirk KA, Whelton PK, Massry S, Jones C, Aogodoa L, Van Lente F: Creatinine clearance as a measure of GFR in screenees for the African-American Study of Kidney Disease and Hypertension pilot study. *Am J Kidney Dis* 32: 32–42, 1998
33. MDRD Study Group: Effects of diet and antihypertensive therapy on creatinine clearance and serum creatinine concentration in the Modification of Diet in Renal Disease Study. *J Am Soc Nephrol* 7: 556–566, 1996
34. Giovannetti S, Barsotti G: In defense of creatinine clearance. *Nephron* 59: 11–14, 1991
35. Shemesh O, Golbetz H, Kriss JP, Myers BD: Limitations of creatinine as a filtration marker in glomerulopathic patients. *Kidney Int* 28: 830–838, 1985
36. Mitch WE, Collier VU, Walser M: Creatinine metabolism in chronic renal failure. *Clin Sci* 58: 327–335, 1980
37. Chertow GM, Lazarus JM, Ma L, Lowrie EG: Bioimpedance norms for the hemodialysis population. *Kidney Int* 52: 1617–1621, 1997
38. Rammohan M, Aplasca EC: Caliper method vs bioelectrical impedance analysis for determining body fat in patients undergoing chronic dialysis and in healthy individuals. *J Am Diet Assoc* 92: 1395–1397, 1992
39. Roubenoff R: Applications of bioelectrical impedance analysis for body composition to epidemiologic studies. *Am J Clin Nutr* 64: 459S–462S, 1996
40. Kuczmarski RJ, Fanelli MT, Koch GG: Ultrasonic assessment of body composition in obese adults: Overcoming the limitations of the skinfold caliper. *Am J Clin Nutr* 45: 717–724, 1987
41. Himes JH, Roche AF, Siervogel RM: Compressibility of skinfolds and the measurement of subcutaneous fatness. *Am J Clin Nutr* 32: 1734–1740, 1979
42. Sullivan DH, Patch GA, Baden AL, Lipschitz DA: An approach to assessing the reliability of anthropometrics in elderly patients. *J Am Geriatr Soc* 37: 607–613, 1989
43. Jensen MD: Research techniques for body composition assessment. *J Am Diet Assoc* 92: 454–460, 1992
44. Lukaski HC, Bolonchuk WW, Hall CB, Siders WA: Validation of tetrapolar bioelectrical impedance method to assess human body composition. *J Appl Physiol* 60: 1327–1332, 1986
45. Astor BC, Coresh J, Heiss G, Pettitt D, Sarnak MJ: Kidney function and anemia as risk factors for coronary heart disease and mortality: The Atherosclerosis Risk in Communities (ARIC) Study. *Am Heart J* 151: 492–500, 2006
46. Langston RD, Presley R, Flanders WD, McClellan WM: Renal insufficiency and anemia are independent risk factors for death among patients with acute myocardial infarction. *Kidney Int* 64: 1398–1405, 2003
47. Beddhu S, Allen-Brady K, Cheung AK, Home BD, Bair T, Muhlestein JB, Anderson JL: Impact of renal failure on the risk of myocardial infarction and death. *Kidney Int* 62: 1776–1783, 2002
48. Manjunath G, Tighiouart H, Ibrahim H, MacLeod B, Salem DN, Griffith JL, Coresh J, Levey AS, Sarnak MJ: Level of kidney function as a risk factor for atherosclerotic cardiovascular outcomes in the community. *J Am Coll Cardiol* 41: 47–55, 2003
49. Muntner P, He J, Hamm L, Loria C, Whelton PK: Renal insufficiency and subsequent death resulting from cardiovascular disease in the United States. *J Am Soc Nephrol* 13: 745–753. 2002
50. Henry RM, Kostense PJ, Bos G, Dekker JM, Nijpels G, Heine RJ, Bouter LM, Stehouwer CD: Mild renal insufficiency is associated with increased cardiovascular mortality: The Hoorn Study. *Kidney Int* 62: 1402–1407, 2002