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# Role of Nutritional Status and Inflammation in Higher Survival of African American and Hispanic Hemodialysis Patients

**Elani Streja, MPH**[1,4], **Csaba P Kovesdy, MD**[5], **Miklos Z Molnar, MD, PhD**[1,9], **Keith C. Norris, MD**[3,7], **Sander Greenland, DrPH**[4], **Allen R. Nissenson, MD**[3,6], **Joel D. Kopple, MD**[1,2,3,4], and **Kamyar Kalantar-Zadeh, MD, MPH, PhD**[1,2,3,4]

[1] Harold Simmons Center for Chronic Disease Research and Epidemiology, Los Angeles Biomedical Research Institute at Harbor-UCLA, Torrance, CA

[2] Division of Nephrology and Hypertension, Los Angeles Biomedical Research Institute at Harbor-UCLA, Torrance, CA

[3] UCLA David Geffen School of Medicine, Los Angeles, CA

[4] Department of Family Health or Epidemiology, UCLA School of Public Health, Los Angeles, CA

[5] Salem Veteran Administration Medical Center, Salem, VA

[6] DaVita, El Segundo, CA

[7] Charles Drew University, Los Angeles, CA

[8] Research Department, Kaiser Permanente of Southern California, Pasadena, CA

[9] Institute of Pathophysiology, Semmelweis University, Budapest, Hungary

## Abstract

**Background—**Observational studies indicate greater survival in African American and Hispanic maintenance hemodialysis (HD) patients compared to their Non-Hispanic White counterparts, even though Blacks have shorter life expectancy than Whites in the general population. We hypothesized that this apparent survival advantage is due to a more favorable nutritional/inflammatory profile in minority HD patients.

**Study Design—**We examined the association between race/ethnicity and 5-year survival before and after adjustment for case-mix and surrogates of the malnutrition-inflammation complex syndrome (MICS), using Cox regression with or without matched sampling in a large cohort of adult HD patients.

**Setting and Participants—**124,029 adult HD patients including 16% Hispanics, 49% non-Hispanic whites, and 35% African Americans.

Address correspondence to Csaba P Kovesdy, MD FASN, Division of Nephrology, Salem VA Medical Center, 1970 Roanoke Blvd., Salem, VA 24153, Phone: 540-982 2463 Fax: 540-224 1963 csaba.kovesdy@va.gov.
Reprint requests to Kamyar Kalantar-Zadeh, MD, MPH, PhD, Harold Simmons Center for Chronic Disease Research and Epidemiology, kamkal@ucla.edu.

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**Predictors—**Race/ethnicity before and after adjustment for MICS including BMI, serum albumin, TIBC, ferritin; creatinine, phosphorus, calcium, bicarbonate, white blood cell count, lymphocyte percentage, hemoglobin and protein intake.

**Outcomes—**5-year (7/2001–6/2006) survival

**Results—**Among dialysis patients, Blacks and Hispanics had lower mortality overall than Non-Hispanic Whites after traditional case-mix adjustment. However, after additional control for MICS, Hispanics had mortality similar to non-Hispanic whites and African American had ever higher mortality. The unadjusted, case-mix and MICS adjusted hazard ratios (and 95% confidence intervals) of African American vs. Whites in the unmatched cohort were 0.68 (0.66–0.69), 0.89 (0.86–0.91) and 1.06 (1.03–1.09), and in the matched cohort 0.95 (0.90–0.99), 0.89 (0.84–0.94) and 1.16 (1.07–1.26), respectively; and for Hispanics vs. Whites in the unmatched cohort were 0.66 (0.64–0.69), 0.84 (0.81–0.87) and 0.97 (0.94–1.00), and in the matched cohort 0.89 (0.84–0.95), 0.88 (0.83–0.95) and 0.98 (0.91–1.06), respectively.

**Limitations—**Unmeasured confounders cannot be adjusted for.

**Conclusions—**Survival advantages of African American and Hispanic HD patients may be related to differences in nutritional and inflammatory status. Further studies are required to explore these differences.

## Keywords

Race; Hispanic paradox-within-paradox; malnutrition-inflammation-complex syndrome; racial disparities

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Chronic kidney disease (CKD) is common and shows major racial and ethnic disparities in the United States.[1–3] Although African Americans make up just 14% of the US total population, about one-third of the 400,000 US dialysis patients are African American.[4, 5] Hispanics compose close to 1/5 of the dialysis population.[4, 5] Such factors as disparities in income, education, diet, and lifestyle, co-morbid conditions and in healthcare have been implicated in the increased mortality of Blacks compared with Whites.[6] The racial and ethnic discrepancies in CKD prevalence have persisted over the past decades. The incidence rates for dialysis patients for 2006 in the African American and Hispanic population continued to be 3.6 and 1.5 times, respectively, greater than among non-Hispanic whites.[4, 5]

In addition to the racial and ethnic differences in CKD rates, there are trends pertaining to interaction of health outcomes and race/ethnicity. Although approximately two-thirds of all US dialysis patients die within 5 years of initiating maintenance dialysis treatment (higher than most cancers[6]), African American and Hispanic patients with end-stage renal disease (ESRD) have consistently greater survival over the past several decades than non-Hispanic Whites, with an death rate of 187 and 180 per 1000 patient-years at risk, respectively, compared to 207 per 1000 patient-years at risk for non-Hispanic Whites.[4, 5] The causes of these disparities remain largely unknown.

Discovering the factors responsible for survival advantages of African American and Hispanic dialysis patients may have major clinical and public health implications, not only for CKD patients but also for other populations with chronic disease states and poor survival. Understanding these factors might lead to methods for improving clinical outcomes in other groups of dialysis patients as well as in populations with other chronic disease states associated with poor survival.[7] These issues are particularly time-sensitive for ESRD patients, since the imminent *Bundling Payment* for provision of medical care to dialysis patients in the US currently does not include an adjustor for race.[8–10] Some nutritional surrogates such as BMI are associated with both race and survival in maintenance

hemodialysis patients, in whom higher BMI is associated with greater survival.[11–14] Glanton *et al.* reported 22% higher adjusted risk of obesity in African-Americans than in White-Americans.[15] The same study also reported slightly higher hazard ratio for mortality in non-Caucasian than in Caucasian maintenance hemodialsis patients with BMI<22 kg/m$^2$.[15] Hence, we hypothesized that the survival advantage of African American and Hispanic dialysis patients are due to distinct clinical conditions. In particular, we hypothesized that better nutritional status can largely explain the greater survival of minorities. Hence, we examined the survival differences across the three mutually exclusive racial and ethnic groups, i.e., Hispanics, non-Hispanic Whites and non-Hispanic African Americans, in a large (over 120,000 subjects) and contemporary (7/2001–6/2006) cohort of long-term hemodialysis patients from a large dialysis organization (DaVita) with continuous treatment records, survival data, and monthly to quarterly blood tests over the 5 years of observation.

## Methods

### Patients

We extracted, refined, and examined data from all individuals with CKD stage 5 who underwent hemodialysis treatment from July 2001 to June 2006, i.e., for 5 consecutive years, in one of the outpatient dialysis facilities of a United States based large dialysis organization, i.e., DaVita (prior to its acquisition of the former Gambro dialysis facilities). The study was approved by the Institutional Review Committees of both Los Angeles Biomedical Research Institute at Harbor-UCLA and DaVita Clinical Research. Because of the large sample size studied, the anonymity of the patients studied, and the non-intrusive nature of the research, the requirement for a written consent form was exempted.

### Clinical and Demographic Measures

The creation of the cohort has been described previously [16–22]. To minimize measurement variability, all repeated measures for each patient during any given calendar quarter, i.e., over a 13-week or 3-month interval, were averaged and the quarterly means in each of the 20 calendar quarters were used in time-dependent analyses. In addition to quarterly laboratory values, post-hemodialysis dry weight (to calculate averaged body mass index [BMI]), was also calculated. Dialysis vintage was defined as the duration of time between the first day of dialysis treatment and the first day that the patient entered the cohort. The first (baseline) study quarter for each patient was the calendar quarter in which patient's vintage was >45 days during at least half of the time of that quarter.

### Laboratory Measures

Blood samples were drawn using uniform techniques in all of the DaVita dialysis clinics and were transported to the DaVita Laboratory in Deland, Florida, typically within 24 hrs. All laboratory values were measured by automated and standardized methods in the DaVita Laboratory. Most laboratory values were measured monthly, including serum urea, creatinine, albumin, calcium, phosphorus, bicarbonate, and total iron binding capacity (TIBC). Serum ferritin and intact PTH were measured at least quarterly. Hemoglobin was measured at least monthly in essentially all patients and weekly to bi-weekly in most patients. Most blood samples were collected pre-dialysis with the exception of the post-dialysis serum urea nitrogen that was obtained to calculate urea kinetics. The Kt/V (single pool) was calculated using urea kinetic modeling equations as described elsewhere.[16] Albumin-corrected calcium was calculated by subtracting 0.8 mg/dL for each g/dL serum albumin below 4.0 g/dL.[23]

### Statistical and Epidemiologic Methods

Survival analyses included time-dependent Cox proportional hazards regression modeling quarterly averaged (over each 13-week or 3-month calendar quarter) values. For each analysis, three models were examined based on the level of multivariate adjustment:

**I.** An unadjusted model that included mortality data or hemodialysis treatment time (in either continuous or ordinal format) and entry calendar quarter (q1 through q20);

**II.** Case-mix adjusted models that included all of the above plus age, gender, diabetes mellitus, categories of dialysis vintage (<6 months, 6 months to 2 years, 2–5 years and ≥5 years), primary insurance (Medicare, Medicaid, private and others), marital status (married, single, divorced, widowed and other or unknown), the standardized mortality ratio of the dialysis clinic during entry quarter, dialysis dose as indicated by Kt/V (single pool), presence or absence of a dialysis catheter, and residual kidney function during the entry quarter, i.e. urinary urea clearance; and

**III.** Malnutrition-inflammation-complex syndrome (MICS), adjusted models which included all of the covariates in the case-mix model as well as 12 surrogates of nutritional status and inflammation, including BMI, and 11 laboratory variables as surrogates of the nutritional state or inflammation or minerals having known association with clinical outcomes in hemodialysis patients: (1) serum albumin, (2) serum TIBC, (3) serum ferritin; (4) serum creatinine, (5) serum phosphorus, (5) serum calcium, (7) serum bicarbonate, (8) peripheral white blood cell count (WBC), (9) lymphocyte percentage, (10) hemoglobin, and (11) nPCR as an indicator of daily protein intake, also known as the normalized protein nitrogen appearance (nPNA). Patients who were transplanted, switched to peritoneal dialysis or left DaVita clinics were censored at the time of the event. Plots of log [−log (survival rate)] against log (survival time) were used to check the proportionality assumption.

All analyses were performed on the cohort of the entire national database. To better control confounding, we also conducted subanalyses that matched most African Americans or Hispanic patients to non-Hispanic white patients in a 1:1 format to create two separate matched cohorts. We also created a 3$^{rd}$ matched cohort by matching each Hispanic patient to an African American patient. We matched randomly and without replacement, using gender, age (±5 years), diabetes, entry calendar quarter, and State of patients' residence (one of the 50 US States, the District of Columbia, or US territories Guam, Puerto Rico, and the Virgin Islands). Matching procedures were completed by SAS macro *Gmatch* based on the Greedy Algorithm created by Kosanke and Bergstralh.[24]

Missing covariate data (under 1% for most laboratory and demographic variables) were imputed by the mean or median of the existing values. Most analyses were carried out with the SAS, version 9.1, SAS Institute, Inc (www.sas.com).

## Results

### Study Participants and Demographics

The original 5-year (7/2001–6/2006) national database of all DaVita hemodialysis patients (prior to Gambro acquisition) included 164,801 subjects, as shown in Figure 1. After deleting those patients who did not receive in-center hemodialysis (such peritoneal dialysis patients) or those whose reported age was either not within the 16 to 99 year old range or not otherwise verifiable, 139,328 hemodialysis patients remained. Out of the latter population, we identified 124,029 hemodialysis patients with consistently reported race/ethnicity

categories in all calendar quarters in one of the 3 mutually exclusive racial/ethnic categories, i.e., Hispanics (16%), non-Hispanic whites (49%), and non-Hispanic African Americans (35%) (Figure 1); in this manuscript the latter 2 groups are referred to as Whites and African Americans, respectively. Hispanic ethnicity included diverse races. Patients who belonged to other races had similar basic characteristics (Table S1, available as online supplementary material).

Table 1 shows demographic, clinical and laboratory characteristics of the three mutually exclusive racial/ethnic cohorts. African American and Hispanic patients were 6 to 7 years younger than the Whites but included larger proportions of patients who were treated for more than 5 years with hemodialysis. Hispanic patients were substantially more likely to be diabetic or have Medicaid as their primary insurance. African Americans had higher serum creatinine, PTH, and ferritin levels.

## Mortality across Race

As shown in Figures 2 and Table 2, survival analyses in the naive (unmatched) cohort showed that, compared to the Whites, African Americans had 32% greater survival in the unadjusted model, and this racial survival benefit fell to 11% after controlling for demographics and other case-mix variables. However, upon adjustment for the MICS panel, African American exhibited a 6% higher death rate (HR, 1.06; 95% confidence interval, 1.03–1.09). In the matched analyses (Table S2 and Figure S1), a similar pattern was observed, with even slightly higher (16%) death rate for African Americans as compared to Whites. As shown in Figure 3, a similar trend was noticed when Hispanics were compared to Whites, in that demographic and nutritional differences could explain essentially the entire survival advantage of the Hispanic ethnicity; however, a reversal of death risk was not observed for the Hispanics. Similar results were found in matched analysis (Figure S2).

## Comparing Mortality of African Americans to Hispanics

When African Americans were compared to Hispanics (Figure 4), the apparently similar death rates of the two groups in the unadjusted models became 11% to 17% higher death rate for African Americans in the case-mix and MICS adjusted models in the unmatched and matched cohort analyses, respectively. Similar results were found in matched analysis (Figure S3). Stepwise analyses of the MICS surrogates showed that serum creatinine, percent of lymphocyte and WBC were the 3 covariates with the largest contribution to the MICS adjustment. Similar associations were found for cardiovascular mortality in the 3-year (7/2001–6/2004) cohort of patients, where the records of cause specific mortality were available (Figures S4–S6).

## Subgroup Analyses

Figure 5 shows analyses in the unmatched cohorts comparing the mortality hazard ratios of African Americans or Hispanics to Whites. When survival models were adjusted for both case-mix and MICS, in most (12 out of 22) strata, the originally lower mortality of African American race compared to Whites reversed to higher mortality risk, while in the remaining strata the same survival status as in Whites was observed. However, this mortality risk reversal was less prominent for the older (age>=65 yrs), hypoalbuminemic (albumin <3.8 g/dL), anemic (hemoglobin<12 g/dL), hyperferritinemic (ferritin>800 ng/ml) and incident (vintage <6 months) patients. Additional sensitivity analyses for early vintage patients (<9 months, <12 months, and <18 months) showed gradual increase in the HR contrast before and after adjustment (data not shown). Hispanic patients showed survival similar to Whites in all but three strata. With the latter three strata, the survival superiority of Hispanics as compared to Whites persisted despite multivariate adjustment (age <65 years, vintage 2 to 5 years, hemoglobin 10 to 11 g/dL).

## Discussion

In a contemporary cohort of 124,029 adult hemodialysis patients composed of three mutually exclusive racial/ethnic categories involving 16% Hispanics, 49% non-Hispanic Whites, and 35% African Americans, we found that African American and Hispanic patients had better survival than Whites even after controlling for such demographic variables as age, gender and diabetes. However, after additional multivariate adjustment for surrogates of nutrition and inflammation, together known as the MICS, African Americans had poorer survival than Hispanics or Whites. After removing the contribution of the nutritional status, higher hemoglobin concentrations were associated with lower survival in African Americans as compared to Whites. Hispanic hemodialysis patients showed a similar reduction of their survival advantage, compared to Whites, after controlling for the MICS, although Hispanic patients younger than 65 years maintained their better survival even after extensive multivariate adjustment.

These findings, if verified in additional studies, may have important clinical and public health implications, since they may indicate that a more favorable nutritional and inflammatory status is the main condition that confers survival benefit to these minorities in the hemodialysis patient populations.[25–27] The incidence and prevalence of ESRD have been rising in the United States and in most countries in the world.[28] Racial discrepancies in CKD have persisted for the past 20 years.[3, 5, 29] The annual ESRD incidence for African Americans and Hispanics in 2006 reached 1,010 and 520 per million population, respectively, which is 3.6 and 1.5 times greater than the incidence in non-Hispanic Whites.[28] The prevalence of ESRD continues to be highest for African Americans and Hispanics, at 5,004 and 2,326 per million population, respectively, as compared to 1,194 per million among Whites.[28] In many maintenance dialysis clinics in inner-city areas, over one-half to two-thirds of dialysis patients are African American, whereas in many other dialysis centers, especially in California or Texas, Hispanics predominate. [28] As shown in Table 1, in our national cohort, over one-third of the dialysis patients were African Americans and 16% Hispanic, as compared to their proportions of 14% and 13% in the general U.S. population, respectively.[28]

About one out of every five American dialysis patients dies each year, and the 5-yr survival is only around 35%,[5, 29] which is worse than survival for most cancers.[5, 7, 29] At any given age group, mortality of dialysis patients is 10–100 times higher than that of non-dialyzed Medicare patients.[4, 28, 30] Nearly half of the deaths are considered to be caused by cardiovascular disease.[29, 31, 32] African American and Hispanic dialysis patients have higher survival than their non-Hispanic White counterparts, a phenomenon that has persisted over the past two decades.[4, 5] The higher dialysis survival of these minorities is rather consistent and independent of demographic or residency status and also of the modality of dialysis treatment (thrice-weekly hemodialysis vs. daily peritoneal dialysis), the dialysis dose, and other factors related to dialysis treatment or techniques..[28, 30] In the U.S. general population, disparities in income, education, and health have been implicated in the increased total mortality and shorter life expectancy of African Americans compared with Whites,[33–35] even though as dialysis patients they have a much lower annual mortality rate (18%) than Whites (28%).[29] A similar phenomenon is observed in preterm African American infants, who are more likely to survive than white infants born after pregnancies of comparable duration,[36] even though within the ‘normal range’ of gestation/birth weight (≥37 weeks/≥2,500–3,000 g) African American infants are substantially more likely to die than white infants.[36, 37] Similar results were found in Netherland, van den Beukel et al reported better survival in immigrant compare to native dialysis patients,[38] and also in Israel, where Arab have higher end-stage renal disease but exhibit greater survival than Jewish Israelis.[39] Our findings indicate that a more favorable nutritional and inflammatory

profile, captured under the MICS adjustor, can explain the bulk of dialysis survival advantage of African Americans, in whom BMI and serum creatinine (an indicator of muscle mass as well as ingestion of skeletal muscle or meat).[40] are substantially higher than in other groups (see Table 1). Indeed, without this favorable nutritional profile, we found that African Americans had higher mortality than others. We also found that after nutritional-status adjustment, higher hemoglobin levels were associated with higher mortality in African Americans, as compared to white hemodialysis patients (Figure 5). Similarly, some but not all previous studies have shown that hemoglobin levels above 13 g/dL are associated with increased mortality as compared to a Hb of 11.5 to 12 g/dL.[41, 42] The survival superiority of African American hemodialysis patients has been treated as confounding that is to be adjusted away.[43] Nonetheless, because race is determined long before nutritional status, the latter functions more as an intermediate rather than confounder, and thus can be considered an integral part of the effect of race.[6] African American dialysis patients exhibit other differences from other racial/ethnic entities. In a 3-year contemporary cohort of 15,859 hemodialysis patients, African Americans were the only patients in whom a high serum LDL (>100 mg/ml) was associated with increase in cardiovascular death risk, whereas LDL-hypercholesterolemia was paradoxically protective in other races.[44] A recent study in 9,303 incident hemodialysis patients, including 3,214 African Americans, suggested that therapy with active forms of vitamin D could explain the greater survival of African American hemodialysis patients. [45] Considering our current findings and the aforementioned studies together, there appear to be altered risk factor patterns in individuals from certain racial/ethnic backgrounds, but not from others, who have chronic health conditions that are associated with increased mortality.

We observed that African American patients tended to have lower WBC and higher serum creatinine levels. Further studies are required to explore these differences. We found that African Americans had better survival than Whites even after controlling for case-mix variables. However, after additional multivariate adjustment for surrogates of nutrition and inflammation, African Americans had indeed even poorer survival than Whites similar to the general population. African-American patients have higher creatinine level (Table 1) which is mainly correlated with muscle mass.[46] Low serum creatinine is a potential marker for the protein-energy wasting (PEW) in maintenance hemodialsis patients.[47] PEW is a strong predictor of mortality in this population.[48] Additionally, African-American patients had lower WBC and higher percentage of lymphocytes representing the lowest level of inflammation. This profile suggests that PEW is less frequent in African Americans than in Whites.

We also examined survival features of Hispanic hemodialysis patients, the second largest and fastest growing US dialysis population. For the past two decades, evidence has accumulated of a tendency for U.S. Hispanics to have lower than average rates of most chronic illnesses, and better than expected health and mortality outcomes, despite the fact that many Hispanics live in relatively poor economic conditions.[49–51] Proposed explanations include under-reporting of Hispanic deaths, ‘salmon bias’ (return of aged Hispanics to their homeland prior to death) and healthy-migrant effects[52, 53] but may not fully account for this so-called “Hispanic Paradox”.[6]

Despite the decreased likelihood of Hispanics developing most chronic diseases, they are, nearly twice as likely to develop ESRD than non-Hispanic whites.[54–56] This striking ‘paradox-within-a-paradox’ might be a function of the increased incidence and prevalence of diabetes mellitus, confirmed in our study (see Table 1). Mexican Americans with type 2 diabetes mellitus are more likely to develop proteinuria,[57] and six times more likely to progress to CKD-5,[55] than non-Hispanic whites. Hispanic patients with CKD almost

inevitably progress to CKD-5.[55] The prevalence of type 2 diabetes mellitus is 2–5 times higher in Hispanics than in non-Hispanic whites.[58–60]

Mexican Americans, who are at highest risk of developing CKD, might share a common genetic background and 'thrifty genotype' with Native American Indians, themselves a group at high risk of developing diabetes mellitus and CKD.[54, 61] When diabetic Mexican Americans undergo maintenance dialysis, however, they are more likely to survive than non-Hispanic whites;[57, 62] which is yet another example of a paradox-within-a-paradox. In our current study we found that the survival advantage of Hispanic hemodialysis patients was mostly explainable by the MICS adjustor, which may indicate that, similar to African Americans, a more favorable nutritional/inflammatory status is also the potential biology behind the survival superiority of this ethnic group.

Our study is limited by the lack of detailed, up-to-date data of comorbid states or measurements of markers of inflammation such as C-reactive protein. However, as indicated previously, we believe that adjustment for the MICS, by including a number of indicators of malnutrition and inflammation, was adequately inclusive, as it could explain the preponderance of survival differences across the racial and ethnic groups. The strengths of this study include the recency of the patient data (from 2001–2006); uniformity in laboratory measurements (performed at a single facility); large sample size; use of time averaged Kt/V and laboratory data, with most values representing means of up to 3 monthly measurements; and analysis of 5-year survival.

In conclusion, in a large cohort of 124,029 adult hemodialysis patients, who were observed for up to 5 years during the first decade of the 21st century, African American and Hispanic patients had greater survival than Whites even after case-mix adjustment. After additional multivariate adjustment for surrogates of nutrition and inflammation, Hispanics had essentially the same mortality as White, whereas African Americans had a greater death risk compared to Hispanics or Whites. Our findings suggest that a healthier nutritional and inflammatory status is the main cause of the survival advantages of minorities who undergo hemodialysis treatment. Trials of nutritional and anti-inflammatory interventions are warranted to examine whether longevity can be improved in dialysis patients and other populations with chronic disease states and the wasting syndrome.

## Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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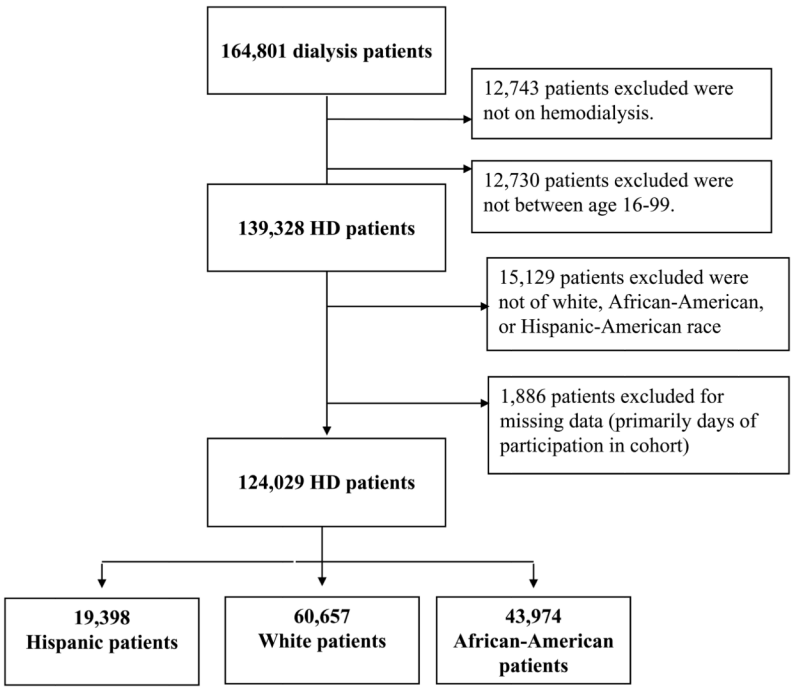


**Figure 1.**
Algorithm (flow chart) of the cohort creation.

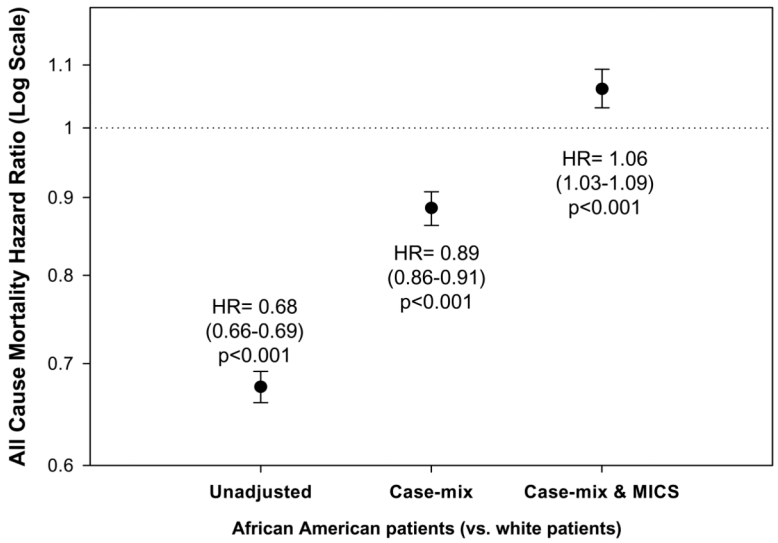


**Figure 2.**

Mortality hazard ratios (from Cox regression) from the unmatched cohort comparing 43,974 African American to 60,657 non-Hispanic White HD patients over a 5-year observation period (7/2001–6/2006). Cox regression based hazard ratios of death are represented by filled circles for unadjusted model, unfilled circles for case-mix-adjusted model, and filled triangles for case-mix and MICS adjusted models. Error bars represent 95% confidence intervals.

Matched cohort was created by 1:1 matching each African American to a non-Hispanic White, matching on gender, age (±5 years), diabetes, entry calendar quarter, and patient residence location (one of the 50 States or the District of Columbia).

Case-mix adjusted models include adjustment for age, gender, diabetes mellitus, standardized mortality ratio, race, vintage, primary insurance, marital status, dialysis dose, dialysis catheter, and baseline comorbid states. Malnutrition-inflammation complex syndrome (MICS) model covariate include all case-mix covariates (see above) plus urea kinetics calculated protein catabolic rate (nPNA or nPCR), serum levels of albumin, creatinine, total iron binding capacity (TIBC), calcium, phosphorous, bicarbonate, ferritin; blood white blood cells (WBC) and lymphocyte percentage.

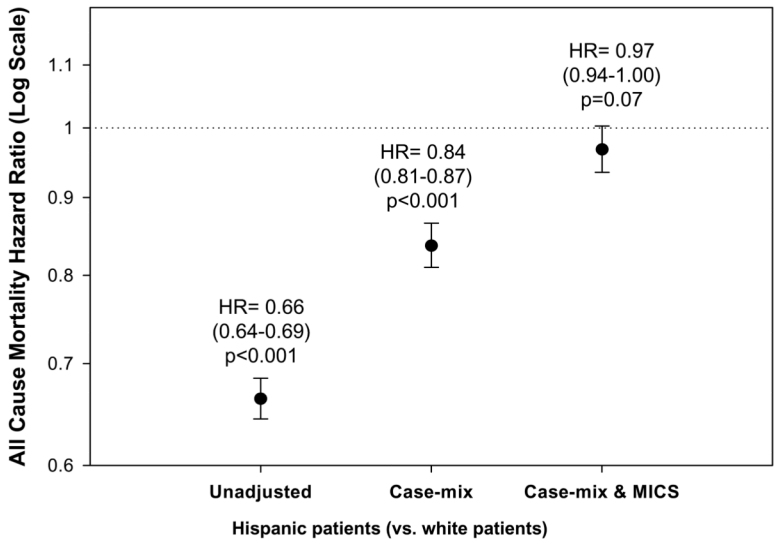


**Figure 3.**

Cox Mortality hazard ratios (from Cox regression) from the unmatched cohort comparing 19,398 Hispanic to 60,657 non-Hispanic White HD patients over a 5-year observation period (7/2001–6/2006). Cox regression based hazard ratios of death are represented by filled circles for unadjusted model, unfilled circles for case-mix-adjusted model, and filled triangles for case-mix and MICS adjusted models. Error bars represent 95% confidence intervals.

Matched cohort was created by 1:1 matching each Hispanic patient to a non-Hispanic White, matching on gender, age (±5 years), diabetes, entry calendar quarter, and State of patients' residence (one of the 50 States or the District of Columbia).

Case-mix adjusted models include adjustment for age, gender, diabetes mellitus, standardized mortality ratio, race, vintage, primary insurance, marital status, dialysis dose, dialysis catheter, and baseline comorbid states. Malnutrition-inflammation complex syndrome (MICS) model covariate include all case-mix covariates (see above) plus urea kinetics calculated protein catabolic rate (nPNA or nPCR), serum levels of albumin, creatinine, total iron binding capacity (TIBC), calcium, phosphorous, bicarbonate, ferritin; blood white blood cells (WBC) and lymphocyte percentage.

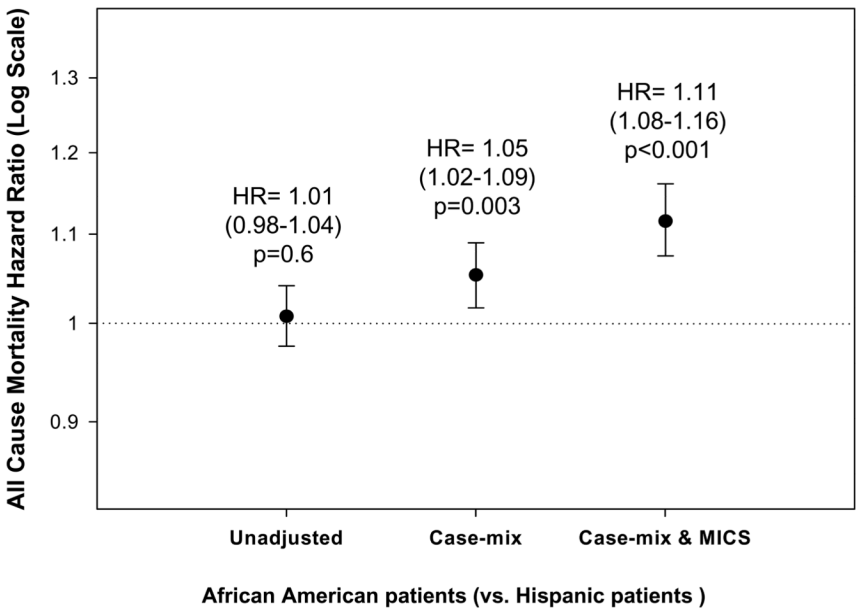


**Figure 4.**

Mortality hazard ratios (from Cox regression) from the unmatched cohort comparing 43,974 African American to 19,398 non-Hispanic White HD patients over a 5-year observation period (7/2001–6/2006). Cox regression based hazard ratios of death are represented by filled circles for unadjusted model, unfilled circles for case-mix-adjusted model, and filled triangles for case-mix and MICS adjusted models. Error bars represent 95% confidence intervals.

Matched cohort was created by 1:1 matching each Hispanic to an African American, matching on gender, age (±5 years), diabetes, entry calendar quarter, and State of patients' residence (one of the 50 States or the District of Columbia).

Case-mix adjusted models include adjustment for age, gender, diabetes mellitus, standardized mortality ratio, race, vintage, primary insurance, marital status, dialysis dose, dialysis catheter, and baseline comorbid states. Malnutrition-inflammation complex syndrome (MICS) model covariate include all case-mix covariates (see above) plus urea kinetics calculated protein catabolic rate (nPNA or nPCR), serum levels of albumin, creatinine, total iron binding capacity (TIBC), calcium, phosphorous, bicarbonate, ferritin; blood white blood cells (WBC) and lymphocyte percentage.

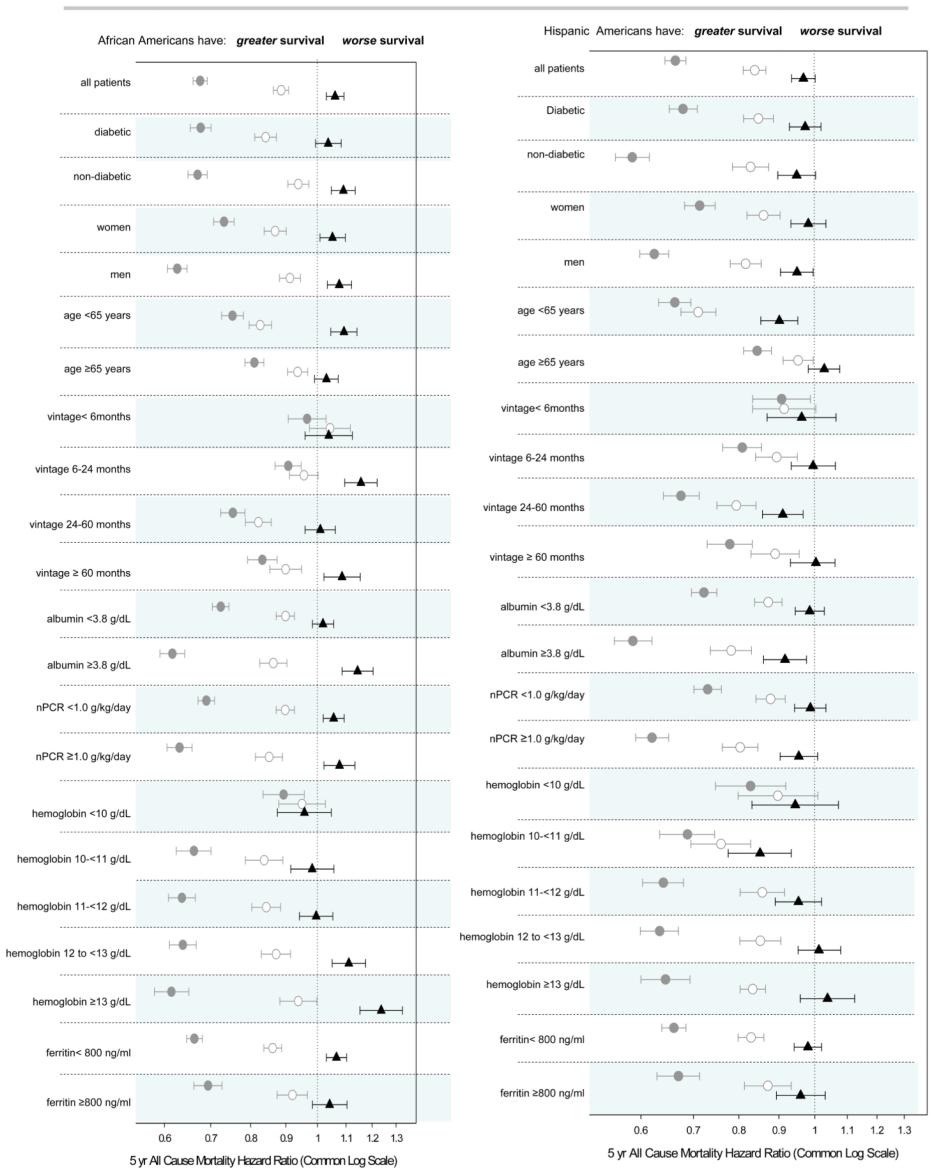


**Figure 5.**

Subgroup survival analyses in the unmatched cohort comparing the mortality hazard ratios for African Americans (Right Panel) or Hispanics (Left panel) to Non-Hispanic Whites in 124,029 long-term HD patients (including 16% Hispanics, 49% non-Hispanic Whites and 35% non-Hispanic African Americans) over a 5-year observation period (7/2001–6/2006). Cox regression based hazard ratios of death are represented by filled circles for unadjusted model, unfilled circles for case-mix-adjusted model, and filled triangles for case-mix and MICS adjusted models. Error bars represent 95% confidence intervals. Case-mix adjusted models include adjustment for age, gender, diabetes mellitus, standardized mortality ratio, race, vintage, primary insurance, marital status, dialysis dose, dialysis catheter, and baseline comorbid states. Malnutrition-inflammation complex syndrome (MICS) model covariate include all case-mix covariates (see above) plus urea kinetics calculated protein catabolic rate (nPNA or nPCR), serum levels of albumin, creatinine, total iron binding capacity (TIBC), calcium, phosphorous, bicarbonate, ferritin; blood white blood cells (WBC) and lymphocyte percentage.

## Table 1

Demographic, clinical and laboratory characteristics of the 3 mutually exclusive racial/ethnic cohorts

| Variable | All Patients | White | African Americans | Hispanic |
|---|---|---|---|---|
| No. of patients | 124,029 (100) | 60,657 (49) | 43,974 (35) | 19,398 (15) |
| No. of deaths | 41,664 (30) | 19,135 (32) | 12,598 (29) | 5,496 (28) |
| Age (years) | 62±15 | 65±15 | 58±15 | 59±15 |
| Gender (% women) | 45 | 42 | 49 | 45 |
| Diabetes mellitus (%) | 44 | 42 | 43 | 56 |
| Dialysis Vintage Category(%): | | | | |
| <6 mo | 18 | 21 | 14 | 15 |
| 6–24 mo | 30 | 33 | 26 | 29 |
| 2–5 y | 32 | 31 | 32 | 34 |
| >5 y | 20 | 14 | 28 | 22 |
| Primary insurance (%) | | | | |
| Medicare | 63 | 65 | 64 | 56 |
| Medicaid | 5 | 2 | 6 | 13 |
| Private Insurance | 9 | 9 | 8 | 10 |
| Other | 15 | 17 | 14 | 13 |
| Marital Status (%) | | | | |
| Married | 48 | 46 | 30 | 43 |
| Divorced | 8 | 7 | 8 | 6 |
| Single | 28 | 16 | 34 | 22 |
| Widowed | 16 | 14 | 12 | 10 |
| Kt/V (single-pool) | 1.51 | 1.54 | 1.46 | 1.57 |
| BMI ($kg/m^2$) | 27.1±7.2 | 26.8±6.8 | 27.4±7.6 | 26.6±6.3 |
| nPNA (nPCR) (g/kg/day) | 0.94±0.25 | 0.93±0.26 | 0.92±0.24 | 1.01±0.26 |
| Biochemical measurements | | | | |
| Serum albumin (g/dL) | 3.65±0.48 | 3.63±0.47 | 3.68±0.48 | 3.68±0.48 |
| Serum creatinine (mg/dL) | 7.9±3.3 | 6.8±2.7 | 9.3±3.6 | 8.0±3.2 |
| TIBC (mg/dL) | 208±47 | 212±48 | 202±45 | 209±45 |
| Serum bicarbonate (mg/dL) | 22.4±3.1 | 22.4±3.1 | 22.5±3.0 | 22.1±3.0 |
| Serum phosphorus (mg/dL) | 5.5±1.5 | 5.5±1.5 | 5.6±1.5 | 5.7±1.5 |
| Serum calcium (mg/dL) | 9.2±0.7 | 9.2±0.7 | 9.2±0.8 | 9.1±0.7 |
| intact PTH (pg/mL) | 408±408 | 339±350 | 505±468 | 385±370 |
| alkaline phosphatase (U/L) | 120±93 | 116±90 | 124±100 | 127±88 |
| Serum ferritin (ng/mL) | 510±492 | 479±459 | 558±536 | 493±467 |
| hemoglobin (g/dL) | 12.0±1.4 | 12.0±1.4 | 11.9±1.5 | 12.1±1.4 |
| WBC ($\times 10^3$/microliter) | 7.5±2.6 | 7.9±2.8 | 7.0±2.4 | 7.6±2.3 |
| lymphocyte (% of total WBC) | 20±8 | 18±7 | 23±8 | 21±7 |

Data are for the base calendar quarter in 124,029 long-term hemodialysis patients. Values are given as number (percentage), percentage, or mean ± SD.

p-value <0.001 ¥ median (IQR)

TIBC: total iron binding capacity. nPNA: normalized protein nitrogen appearance; nPCR, normalized protein catabolic rate; BMI, body mass index; PTH, parathyroid hormone; WBC, white blood cell;

Conversion factors for units: serum creatinine in mg/dL to μmol/L, ×88.4; serum albumin in mg/dL to g/L, ×10; hemoglobin in g/dL to g/L, ×10

**Table 2**

Hazard ratios of death using Cox regression analyses

| Race Categories* | Unadjusted | | Case-mix adjusted** | | Case-mix & MICS adjusted*** | |
|---|---|---|---|---|---|---|
| | HR (95% CI) | P-value | HR (95% CI) | P-value | HR (95% CI) | P-value |
| African Americans vs. Whites | 0.68 (0.66–0.69) | <0.001 | 0.89 (0.86–0.91) | <0.001 | 1.06 (1.03–1.09) | <0.001 |
| Hispanics vs. Whites | 0.66 (0.64–0.69) | <0.001 | 0.84 (0.81–0.87) | <0.001 | 0.97 (0.94–1.00) | 0.07 |
| African Americans vs. Hispanics | 1.01 (0.98–1.04) | 0.6 | 1.05 (1.02–1.09) | 0.003 | 1.11 (1.08–1.16) | <0.001 |

Note: analyses (all unmatched) performed in the naive cohorts of 124,029 long-term HD patients (including 16% Hispanics, 49% non-Hispanic Whites and 35% non-Hispanic African Americans) over a 5-year observation period (7/2001–6/2006).

* Each models includes HD patients of 2 out of the 3 mutually exclusive racial and ethnic categories

** case-mix adjusted models include adjustment for age, gender, diabetes mellitus, standardized mortality ratio, vintage, primary insurance, marital status, dialysis dose, dialysis catheter, and baseline comorbid states

*** Malnutrition-inflammation complex syndrome (MICS) model covariate include all case-mix covariates (see above) plus urea kinetics calculated protein catabolic rate (nPNA or nPCR), serum levels of albumin, creatinine, total iron binding capacity (TIBC), calcium, phosphorous, bicarbonate, ferritin; blood white blood cells (WBC) and lymphocyte percentage.

HR, hazard ratio; CI, confidence interval; MICS, Malnutrition-inflammation complex syndrome

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