Racial and ethnic disparities in pediatric renal allograft survival in the United States

Rachel E Patzer, PhD, MPH1,2, Sumit Mohan, MD, MPH7, Nancy Kutner, PhD3, William McClellan, MD, MPH1,4, and Sandra Amaral, MD, MHS5,6

1Emory University Department of Surgery, Division of Transplantation, Atlanta, GA
2Rollins School of Public Health, Department of Epidemiology, Emory University, Atlanta, GA
3Emory University, Department of Rehabilitation Medicine, WMB, Room 338, 1639 Pierce Dr., Atlanta, GA 30322
4Emory University, Division of Nephrology, WMB, Room 338, 1639 Pierce Dr., Atlanta, GA 30322
5Department of Pediatrics, The Children’s Hospital of Philadelphia, 34th and Civic Center Blvd, Philadelphia, PA 19104
6Department of Biostatistics and Epidemiology, Perelman School of Medicine, University of Pennsylvania, Philadelphia, PA
7Department of Medicine, Division of Nephrology, Columbia University Medical Center, 622 W 168th St, PH4-124, New York, NY 10032

Abstract

This study was undertaken to describe the association of patient race/ethnicity and renal allograft survival among the national cohort of pediatric renal allograft recipients. Additionally, we determined whether racial and ethnic differences in graft survival exist among individuals living in low or high poverty neighborhoods and those with private or public insurance. Among 6,216 incident, pediatric End Stage Renal Disease patients in the United States Renal Data System (kidney transplant from 2000 through September, 2011) 14.4% experienced graft failure, with a median follow-up time of 4.5 years. After controlling for multiple covariates, black race, but not Hispanic ethnicity, was significantly associated with a higher rate of graft failure for both deceased and living donor transplant recipients. Disparities were particularly stark by 5 years post-transplant, when black living donor transplant recipients experienced only 63.0% graft survival compared with 82.8% and 80.8% for Hispanics and whites, respectively. These disparities persisted among high and low poverty neighborhoods and among both privately- and publicly-insured patients. Notably profound declines in both deceased and living donor graft survival rates for black, compared to white and Hispanic, children preceded the 3-year mark when transplant
Medicare eligibility ends. Further research is needed to identify the unique barriers to long-term graft success among black pediatric transplant recipients.

Keywords
Kidney transplantation; end-stage renal disease; access to healthcare; racial disparities; transplant outcomes; United States Renal Data System; clinical epidemiology

Introduction

Kidney transplantation is the preferred treatment for End-Stage Renal Disease (ESRD) patients due to its improved patient survival, quality of life, reduced morbidity, and economic savings compared to dialysis. In pediatric ESRD, children who receive kidney transplants also show improved growth. However, transplantation is not a cure; for children with ESRD, conditional on surviving the first year with a functioning transplant, the current graft half-life is estimated at 12 years for deceased donor (DD) transplants and 15 years for living donor (LD) transplants. Transplant maintenance requires diligent medication adherence, frequent laboratory monitoring and clinic visits.

In the U.S., racial, sociocultural, and socioeconomic differences have been shown to compound the challenges of maintaining long-term function of a kidney transplant. Among renal allograft recipients, studies have reported worse short- and long-term allograft survival for African American patients in both adults and children. In the pediatric population, the rate of graft failure among black patients has been reported as nearly twice the rate of graft failure of white patients. Several studies have documented racial disparities in access to kidney transplantation among blacks and Hispanics vs. white pediatric patients, including decreased rates of waitlisting and reduced rates of preemptive transplantation, lower living donor rates and poorer HLA matches. Pediatric ESRD patients who receive a preemptive transplant or LD (vs. DD) kidney transplant have improved graft survival.

The reasons for these disparities are likely multifactorial in nature, and low socioeconomic status (SES) is an important risk factor for poor health outcomes among pediatric ESRD patients. While prior studies have adjusted for some SES factors, the presence of racial disparities in renal allograft survival across levels of SES has not been previously described among the U.S. pediatric kidney allograft recipient population. Furthermore, Hispanics have often been overlooked in pediatric studies of renal allograft survival, although they comprise a growing proportion of the pediatric ESRD population. One single center study by Muneeruddin et al. suggested that Hispanics had improved DD graft survival but similar LD graft survival compared with African Americans. Notably, in the Muneeruddin et al. study, Hispanics were of similar SES to whites. Finally, previous research examining racial and ethnic differences in pediatric allograft survival has not examined interactions between SES and donor source. The purpose of our study was to describe the association of patient race/ethnicity and renal allograft survival among the national cohort of pediatric renal allograft recipients, and to determine whether racial and ethnic differences in LD and DD allograft survival exist among individuals living in low vs. high poverty neighborhoods and those with private vs. public insurance.
Results

Demographic and Clinical Characteristics of Study Population

Among the 6,216 pediatric DD or LD recipients included in this analysis, 893 patients (14.4%) experienced graft failure due to any cause over a median follow-up period of 4.5 years and an additional 307 pediatric transplant recipients (4.9%) died with a functioning graft. Demographic and clinical characteristics of the study population by race/ethnicity are presented in Table 1. The mean age at the time of transplant was 10.9 ± 5.2 yrs, 58.5% were male, 21.1% were black, and 26.7% were Hispanic. Racial differences in demographic and clinical characteristics of the pediatric transplant recipients were apparent. On average, white patients were younger (10.3 years) compared to Hispanic (11.3 years) and black patients (11.8 years). Compared to white patients, blacks were more likely to have a body mass index (BMI) >85th percentile (19.0% vs. 11.6%). Compared to whites, a greater proportion of both Hispanic and black patients had public insurance (71.5% and 68.8% vs. 42.5%, respectively) and lived in impoverished neighborhoods (32.9% and 38.2% vs. 13.6%) (Table 1).

Transplant and Donor Characteristics of Study Population by Race

Both Hispanic and black patients were less likely to receive a LD transplant (33.3% and 25.5% vs. 58.6%), to be preemptively transplanted (14.2% and 8.7% vs. 27.4%, respectively) and more likely to spend ≥ear on dialysis before transplant (59.4% and 56.5% vs. 36.5%, respectively) vs. whites. Longer donor cold ischemia time (>24 hours) was more common among black DD recipients compared to whites and Hispanics (11.8% vs. 9.0% vs. 8.8%, respectively). Donor age was higher among whites compared to minorities, and whites had fewer HLA mismatches. The majority of patients (77.5%) were prescribed a tacrolimus-based immunosuppression regimen at discharge, followed by a cyclosporine-based regimen (11.7%), and a higher proportion of black and Hispanic patients were prescribed tacrolimus- vs. cyclosporine-based regimens vs. whites (81.7% and 79.9% vs. 74.9%, p<0.0001). Delayed graft function was more common among black DD transplant recipients (11.2%), vs. whites (5.4%) and Hispanics (5.9%) (Table 1).

Graft Survival (Crude Analyses)

Among pediatric kidney transplant recipients in the U.S., overall one-year graft survival was 95.2% (95% CI: 94.4–95.9) among DD transplant recipients and 97.9% (95% CI: 97.3–98.3) among LD transplant recipients (Figure 1). Overall graft survival rates were lower for blacks compared to both Hispanics and whites throughout the follow-up period (p<0.0001), with racial/ethnic differences more pronounced in long-term graft survival (Figure 1). Among DD transplant recipients, the 2-year graft survival was 91.5% for whites and 93.7% for Hispanics, but only 86.4% for blacks. Among LD transplant recipients, the 2-year graft survival was 96.8% for whites, 96.7% for Hispanics, and 93.0% for blacks. These racial differences were notably greater in 5-year graft survival. At 5-years, black LD recipient overall graft survival was 78.9% (vs. 90.8% for Hispanics; 92.2% for whites) and black DD recipient graft survival was 63.0% (vs. 82.8% for Hispanics; 80.8% for whites).
In crude Cox analyses for DD transplant recipients, Hispanics had similar rates of graft failure at any given time during follow-up vs. white DD recipients (HR=0.90; 95% CI: 0.74–1.09) and rate of graft failure for blacks was twice as high as whites (HR=2.00; 95% CI: 1.70–2.36). Among LD transplant recipients, the disparity was higher among black vs. whites (HR=2.65; 95% CI: 2.03–3.45), and similar among Hispanics vs. white (HR=1.06; 95% CI: 0.78–1.43) (Table 2).

Of known causes of graft failure (71% of graft failures), the most common reasons included chronic rejection (37.7% of graft failures), acute rejection (23.3%), recurrent disease (10.8%), other (10.4%), and noncompliance (9.5%). Less common reasons included primary failure (3.0%), graft thrombosis (2.1%), infection (1.7%), BK Virus (1.3%), and urologic complications (0.5%). Racial/ethnic differences in causes of graft failure did exist, where a greater proportion of black patients (27.0%) had acute rejection compared to whites (21.7%) and Hispanics (19.0%). Whites also had a higher rate of recurrent disease (14.5%) vs. Hispanics (10.4%) and blacks (7.5%). Additionally, noncompliance was reported as the cause of graft failure more commonly for black (11.4%) vs. whites (8.3%) and Hispanic (7.9%) patients.

**Multivariable-adjusted Graft Failure**

The final, multivariable, donor type-stratified, Cox models examining the effect of race/ethnicity on graft survival adjusted for age, sex, insurance status, neighborhood poverty, etiology of ESRD, peak panel reactive antibody, BMI >85%, blood type, receipt of a preemptive transplant, immunosuppression regimen at discharge, induction therapy, Share 35 cohort era (i.e. post-2005 implementation of the Share 35 allocation policy that preferentially allocated donors < 35 years to pediatric patients < 18 years), and Organ Procurement Organization (OPO) allocation region (1–11). Models adjusting for other demographic and clinical characteristics were examined, but differences were not meaningfully or statistically different from the final model. In final multivariable models, Hispanics had similar or lower rates of overall graft failure over the study period vs. white patients for both DD (HR=0.84; 95% CI: 0.68–1.03) and LD (HR=0.76; 95% CI: 0.54–1.06) transplants (Table 2). In contrast, the rate of overall graft failure among blacks was nearly twice that of whites for both DD (HR=1.82; 95% CI: 1.52–2.18) and LD (HR=1.82; 95% CI: 1.34–2.47) transplants (Table 2).

Estimates of the effect of race/ethnicity on graft failure within strata of neighborhood poverty and health insurance status are presented in Table 3a (DD transplant recipients) and Table 3b (LD transplant recipients). The effect of race/ethnicity across various SES levels was consistent, i.e. we did not observe statistically significant interaction between race/ethnicity and either health insurance or neighborhood poverty for both LD and DD transplant recipients (p > 0.05). Across donor source, poverty level and insurance status, black (vs. white) children experienced a significantly higher rate of graft failure, whereas Hispanic (vs. white) children experienced a similar to lower rate of graft failure.
**Subanalyses**

In a sensitivity analysis excluding patients with ‘other’ insurance (n=116; 1.9% of study population), multivariable results for the effect of race/ethnicity on graft failure for either LD or DD transplant recipients did not differ significantly from main analyses.

Analyses that excluded patients with Focal Segmental Glomerulosclerosis (FSGS) were similar to main analyses for DD transplant recipients; among LD transplant recipients, the multivariable-adjusted effect of black vs. white race/ethnicity on graft failure was more pronounced (HR=2.03; 95% CI: 1.46–2.83).

Since death following kidney transplantation is a fairly rare event in children (4.9% of study population), multivariable modeling results considering death-censored graft failure as the outcome were similar to main results (results not shown).

**Discussion**

In this cohort of U.S. pediatric kidney transplant recipients, black (vs. white or Hispanic) race was associated with worse graft survival, particularly at 3 and 5 years post-transplant, regardless of donor source. These racial disparities persisted in low neighborhood poverty areas and among those who were privately insured. This disparity persisted despite adjustment for a broad array of demographic, clinical and transplant characteristics, such that black children were nearly twice as likely to experience graft failure at any given time when compared with white children (HR 1.82; 95%CI: 1.52–2.18 for DD; HR 1.82; 95%CI: 1.34–2.47 for LD).

For recipients of LD kidney transplants, white and Hispanic children experienced 5-year overall graft survival rates of 92.2% and 90.8%, respectively. Black children who received LD kidney transplants, however, experienced a 5-year overall graft survival rate of just 78.9%. For comparison, the 5-year graft survival rate for adult LD kidney transplant recipients is 83%\(^{19}\). Thus, among pediatric recipients of LD kidneys, most children fare well and experience outcomes superior to adults; however, black children who receive living donor kidneys actually fare worse than adult living donor kidney recipients at 5-years.

When we compared unadjusted allograft outcomes by race/ethnicity among pediatric recipients of DD kidneys, racial disparity was even more striking. In fact, black pediatric DD kidney recipients experienced only a 63% five-year graft survival rate (vs. 80.8% for whites and 82.8% for Hispanics.) Again, in comparison, 5-year graft survival for adults who receive DD is 71%\(^{19}\). Thus, black pediatric DD kidney recipients appear to experience substantially poorer 5-year outcomes than adult DD kidney recipients. After adjusting for various demographic, clinical, and SES factors, the racial/ethnic disparities in allograft survival were similar for both LD and DD transplant recipients.

Hispanics had equivalent (or better) graft survival compared to whites, across donor source, poverty levels and insurance status. Notably, both Hispanics and blacks had similarly large proportions of patients with public insurance and patients living in the poorest neighborhoods. We were unable to examine whether change in insurance status, such as loss
of insurance coverage after three years post-transplant, influenced outcomes. One study comparing European and U.S. graft survival rates suggested that insurance differences might influence differences in international transplant outcomes\(^{21}\). However, several U.S. studies have found that while loss of Medicare coverage in the U.S. influences graft survival, it does not explain the substantial differences in racial disparities observed in adolescent and adult kidney transplantation access\(^{13,15}\) and outcomes\(^{22,23}\). If loss of insurance at 3 years was the driving force behind disparities in longer term allograft survival, poor outcomes would be expected among Hispanic children as well as black children, which was not observed in our study. Further, our Kaplan-Meier graft survival curves demonstrated a divergence in graft survival (LD and DD) among black (vs. white and Hispanic) children before three years.

Hispanics and blacks were also more likely to be adolescents, have poorer HLA histocompatibility matching, have higher cold ischemia time, and delayed graft function, and less likely to receive preemptive transplantation compared with white children. All of these factors are considered risk factors for poorer long-term allograft survival\(^{11,24–27}\). Thus, we had expected comparable graft survival rates between blacks and Hispanics. Several prior studies have demonstrated that Hispanics in the United States tend to have better health and survival outcomes, despite more limited access to healthcare, lower incomes and education levels, and higher prevalence of hypertension, diabetes, and obesity compared to whites\(^{28,29}\). This “Hispanic Paradox” is hypothesized to reflect social and cultural factors that promote health, strong ethnic identity, the healthy migrant effect, acculturation, and/or study bias (e.g. misclassification bias, selection bias, etc)\(^{30–32}\). There may be other factors that are influencing graft survival among this group of Hispanic patients that are unaccounted for in our analyses. For example, Hispanic ethnicity comprises a variety of different ethnic backgrounds. Thus, we may be masking differences within subgroups by collectively assigning the label “Hispanic” to a heterogeneous population.

Why are black children faring worse in longer-term graft survival, across donor source, poverty level and insurance status? There may be unmeasured immunologic or biologic barriers to long-term graft survival which are unique to blacks.\(^{33}\) For example, blacks have higher rates of FSGS which can recur post-transplant. We attempted to examine this possibility by excluding patients with FSGS in a sensitivity analysis; however, this approach did not change our study results.

Blacks also tend to experience longer time on dialysis before transplant and may thus incur greater burdens from longer exposure to the comorbidities of chronic kidney disease, including cardiovascular disease\(^{34}\). We examined eGFR at listing and found that whites had a statistically significant eGFR at listing that was higher than Hispanics and blacks (white mean GFR 13.1 vs. Hispanic mean GFR 11.8 and black mean GFR 11.4, \(p<0.0001\)). Whether this small difference in GFR is clinically significant is unclear and does not explain why poorer long-term allograft outcomes were observed for blacks but not Hispanics.

Additionally, there may be immunocompatibility differences by race which increased risk for sub-acute rejection and chronic allograft nephropathy\(^{33,35,36}\). There are known racial differences in the metabolism of certain immunosuppressants\(^{37,38}\) and thus biologic differences may play a role. Black children in our data set were significantly more likely to
have acute rejection reported as their primary etiology of graft failure. We also observed that blacks were significantly more likely to be labeled as non-adherent as the primary cause for graft failure. Whether this reflects reporting bias or true non-adherence is unclear, since a patient may be labeled noncompliant at any time either before or after graft failure. Adherence has been shown to decline with greater time post-transplant\(^3\). Nonadherence is also significantly associated with graft failure among pediatric renal transplant recipients\(^4\). Moseley and Kershaw recently hypothesized that the primary cause of disparities in pediatric kidney transplantation for black (vs. white) children is “a social environment that limits the availability of suitable organs and makes adherence more difficult for (black families) compared with white (families)”\(^4\). The authors posit that societal bias has led to the unequal distribution of education, wealth and employment that undermines the ability to comply with the medical regimen. We were unable to explain our observed differences in graft survival rates by our proxies of SES. However, it is possible that the measures used in our study -- insurance status and neighborhood poverty -- do not adequately capture the burdens of poverty which impair long-term adherence. Since we did not observe reduced rates of graft survival among Hispanics who also comprised a large proportion of the poor and underinsured in our cohort, it is feasible that unique cultural barriers and racially-specific (black vs. white) bias enhance the burden of poverty for blacks.

As with any observational study, residual confounding from unmeasured variables may have impacted our results. In this study, we were unable to measure changing health status during follow-up and the measures for reported noncompliance are likely incomplete. Further, immunosuppression dosing information is not available in USRDS data, although in pediatric kidney transplant recipients, immunosuppressive doses are generally based on weight or age and are not race-adjusted as standard of care\(^4\). Causes of graft failure reported in the USRDS database have not been validated, and may not reflect more complex behavioral, psychosocial, or insurance causes. Finally, our study focused on patients of race/ethnicity reported as white, black, and Hispanic. While these results may not be generalizable to other minorities, this study highlights the importance of examining biological and social constructs of minority groups that may influence poor outcomes following transplantation.

This study has many strengths. For a pediatric study, our large study population provided enough study power to examine multiple racial/ethnic groups. This is the first U.S. study to compare pediatric allograft survival among Hispanics vs. black and white patients, among both DD and LD transplant recipients. Further, this is the first study of pediatric allograft survival to consider such a broad array of clinical and demographic patient-level factors and with the interaction between SES and race/ethnicity concurrently. United States Renal Data System (USRDS) data are virtually 100% complete, so study population follow-up is >99%.

In conclusion, this study is the first to our knowledge to explicitly examine the impact of both individual and neighborhood level SES on racial/ethnic differences in allograft survival among pediatric kidney transplant recipients in the US by donor source. We report that black (vs. white) children experience significantly higher rates of graft failure, regardless of donor source, poverty level or insurance status, whereas Hispanic children experience similar allograft survival rates (vs. whites) after adjusting for differences in demographic and
clinical factors and disparate SES. Targeted studies are greatly needed to identify modifiable vs. non-modifiable barriers to long-term graft success for black children. Only by gaining a better understanding of the problem, can interventions be directed at effective solutions.

Methods

Study Population and Data Sources

Incident pediatric (age < 18 years) renal allograft recipients who received a DD or LD transplant between Jan. 2000 through Sept. 2011 identified in the USRDS were included in this analysis. Basic demographic data were obtained from USRDS via the Centers for Medicare & Medicaid Services (CMS) Medical Evidence Form (CMS-2728), which is completed at ESRD treatment start. American Community Survey (ACS) data (2005–2010) on neighborhood poverty were linked to patient’s residential zip code. ACS is an ongoing annual survey conducted by the U.S. Census Bureau on demographic, economic, social, housing and financial characteristics of nearly two million subjects. Transplant follow-up and outcome data were obtained from USRDS transplant data files.

A total of 7,878 pediatric (<18 yrs) transplant recipients were identified within USRDS from 1/1/2000 through 9/30/2011. Patients were excluded if they received a prior transplant (n=770), they received a multiple organ transplant during the study period (n=87), and those with missing donor type (n=12). In addition, due to small sample size, we excluded patients whose race or ethnicity was reported as other than white non-Hispanic, white Hispanic, or black (n=676). Patients who were either missing residential zip code or could not be linked with American Community Survey 2007–2011 zip code data (n=117) were excluded. The final study population consisted of 6,216 pediatric patients.

Study Variables

The primary outcome variable was overall graft failure during the follow-up period, where deaths were considered graft failure events. Study participants were identified at the date of transplant and followed until graft failure, death, or the end of the study (Sept 30, 2011). We also considered death-censored graft survival, in subanalyses. Follow-up time for overall graft survival was defined as time from transplant until either graft failure or death. Analyses were stratified by donor type.

Race/ethnicity was the main exposure of interest in the analysis. Neighborhood poverty and health insurance were considered proxies for SES. Neighborhood poverty was estimated by the proportion of individuals residing below the federal poverty level in each 5-digit zip code using 2007–2011 American Community Survey Census data. We defined high neighborhood poverty as areas where 20% or more of the households were assigned to below the federal poverty level. Primary health insurance at the time of transplant was categorized as private, public (Medicaid, Medicare Fee for Service, Medicare & Choice, Children’s Health Insurance Program, Department of VA, Public insurance – other government, US/State Government Agency, or Medicare Unspecified), or other (Self, Donation, Free Care, or Unknown).
Covariates considered included both donor and recipient age at transplant (years), recipient sex (male or female), pre-transplant BMI >85th percentile, and etiology of ESRD. Transplant characteristics considered included duration of dialysis (preemptive transplant, 0–6 months, 6–12 months, 12–18 months, and >18 months), number of HLA mismatches (0–6), blood type, peak panel reactive antibody (PPRA) (0–19, 20–79, vs. ≥) and cold ischemic time (0–12 hours, 12–24 hours, >24 hours, and missing). We also considered delayed graft function (dialysis within the first week post-transplant), use of induction therapy (yes/no), immunosuppressant treatment regimen (tacrolimus-based, cyclosporine-based, or other regimen), induction base therapy (thymoglobulin, Interluekin-2 receptor alpha chain, Campath, steroid only, or no induction therapy), transplant era (pre- and post-Share 35 allocation policy implementation in September 2005), and 11 OPO allocation regions. In the US, an OPO is a nonprofit organization that is responsible for the evaluation and procurement of deceased donor organs for transplantation.

Data Analysis

Chi-square tests and t-tests (or non-parametric equivalents of the t-test) were used to examine the differences between baseline characteristics, including demographic and clinical characteristics, by race/ethnicity. Crude graft survival was examined using Kaplan-Meier methods, where comparisons between groups were analyzed using the log-rank test statistic. We used a Cox proportional hazards model to examine the association between race/ethnicity and time to graft failure, stratified by donor type. Covariates were considered as potential confounders based on their association with race and SES and graft failure, or because of known clinical importance. We utilized a sequential modeling approach, modeling the crude relationship between race/ethnicity and graft failure (model 1), adjusting for demographic and clinical characteristics (model 2), and next adjusting for demographic, clinical, and SES characteristics, (model 3).

Robust sandwich variance estimators were used to account for potential correlation between patients living in the same neighborhood. In multivariable analyses, we used the Markov Chain Monte Carlo method for multiple imputation methods for missing covariate information. To examine whether racial/ethnic differences in graft survival persisted even among patients without FSGS, we conducted sensitivity analyses excluding this subgroup of patients. We also conducted secondary analyses examining death-censored graft failure. All analyses were performed with SAS software version 9.3. The Emory University Institutional Review Board approved this study.

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Abbreviations

<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Description</th>
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<tbody>
<tr>
<td>ESRD</td>
<td>End Stage Renal Disease</td>
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<td>USRDS</td>
<td>United Renal Data System</td>
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<td>HLA</td>
<td>Human Leukocyte Antigen</td>
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<td>BMI</td>
<td>Body Mass Index</td>
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<td>SES</td>
<td>socioeconomic status</td>
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References


Figure 1.
Kaplan-Meier Analysis of Time to Overall Graft Failure by Race/Ethnicity among Deceased Donor Transplant Recipients (Panel A) and Living Donor Transplant Recipients (Panel B).
Table 1

Demographic and Clinical Characteristics of Study Population at the Time of Transplant by Race/Ethnicity

<table>
<thead>
<tr>
<th>Study Population N = 6,216</th>
<th>White, Non-Hispanic n = 2922 (53.8%)</th>
<th>White, Hispanic n = 1659 (26.7%)</th>
<th>Black n = 1216 (21.1%)</th>
<th>P-value For Race Difference</th>
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<tbody>
<tr>
<td>Age at Transplant, Mean (yrs)</td>
<td>10.9 ± 5.2</td>
<td>10.3 ± 5.4</td>
<td>11.3 ± 5.0</td>
<td>11.8 ± 4.9</td>
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<tr>
<td>Age at Transplant, Group (yrs)</td>
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<td></td>
<td></td>
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<tr>
<td>0–2 yrs</td>
<td>352 (5.7%)</td>
<td>239 (7.2%)</td>
<td>74 (4.5%)</td>
<td>2 (3.2%)</td>
</tr>
<tr>
<td>3–5 yrs</td>
<td>1004 (16.2%)</td>
<td>605 (18.1%)</td>
<td>236 (14.2%)</td>
<td>163 (13.4%)</td>
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<td>6–10 yrs</td>
<td>1135 (18.3%)</td>
<td>641 (19.2%)</td>
<td>303 (18.3%)</td>
<td>191 (15.7%)</td>
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<tr>
<td>11–17 yrs</td>
<td>3725 (59.9%)</td>
<td>1856 (55.6%)</td>
<td>1046 (63.1%)</td>
<td>823 (67.7%)</td>
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<tr>
<td>Male Sex</td>
<td>3634 (58.5%)</td>
<td>1982 (59.3%)</td>
<td>903 (54.4%)</td>
<td>749 (61.6%)</td>
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<tr>
<td>Cause of ESRD</td>
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<td>&lt; 0.0001</td>
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<td>GN²</td>
<td>566 (9.1%)</td>
<td>261 (7.8%)</td>
<td>191 (11.5%)</td>
<td>114 (9.4%)</td>
</tr>
<tr>
<td>Secondary GN</td>
<td>380 (6.1%)</td>
<td>245 (7.3%)</td>
<td>84 (5.1%)</td>
<td>51 (4.2%)</td>
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<td>Cystic/Hereditary Disease</td>
<td>2794 (45.0%)</td>
<td>1679 (50.3%)</td>
<td>667 (40.2%)</td>
<td>448 (36.9%)</td>
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<tr>
<td>FSGS³</td>
<td>825 (13.3%)</td>
<td>335 (10.0%)</td>
<td>204 (12.3%)</td>
<td>286 (23.5%)</td>
</tr>
<tr>
<td>Lupus Nephritis</td>
<td>114 (1.8%)</td>
<td>23 (0.7%)</td>
<td>48 (2.9%)</td>
<td>43 (3.5%)</td>
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<tr>
<td>Other</td>
<td>1537 (24.7%)</td>
<td>798 (23.9%)</td>
<td>465 (28.0%)</td>
<td>274 (22.5%)</td>
</tr>
<tr>
<td>BMI &gt; 85%</td>
<td>842 (13.6%)</td>
<td>386 (11.6%)</td>
<td>225 (13.6%)</td>
<td>231 (19.0%)</td>
</tr>
<tr>
<td>eGFR⁴ at Time of Waitlisting, Mean (ml/min/1.73 m²)</td>
<td>12.4 ± 5.5</td>
<td>13.1 ± 5.6</td>
<td>11.8 ± 5.2</td>
<td>11.4 ± 5.1</td>
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<tr>
<td>Recipient Socioeconomic Characteristics N (%)</td>
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<tr>
<td>Health Insurance at time of transplant</td>
<td></td>
<td></td>
<td></td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Public</td>
<td>3442 (55.4%)</td>
<td>1419 (42.5%)</td>
<td>1186 (71.5%)</td>
<td>837 (68.8%)</td>
</tr>
<tr>
<td>Private</td>
<td>2658 (42.8%)</td>
<td>1871 (56.0%)</td>
<td>425 (25.6%)</td>
<td>362 (29.8%)</td>
</tr>
<tr>
<td>Other</td>
<td>116 (1.9%)</td>
<td>51 (1.5%)</td>
<td>48 (2.9%)</td>
<td>17 (1.4%)</td>
</tr>
<tr>
<td>High Poverty Neighborhood (&gt;20% Zip Code below Poverty)</td>
<td>1463 (23.5%)</td>
<td>453 (13.6%)</td>
<td>545 (32.9%)</td>
<td>465 (38.2%)</td>
</tr>
</tbody>
</table>

Transplant Characteristics
<table>
<thead>
<tr>
<th>Donor Type</th>
<th>Study Population N = 6,216</th>
<th>White, Non-Hispanic n = 2922 (53.8%)</th>
<th>White, Hispanic n = 1659 (26.7%)</th>
<th>Black n = 1216 (21.1%)</th>
<th>P-value For Race Difference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Deceased Donor</td>
<td>2821 (45.4%)</td>
<td>1383 (41.4%)</td>
<td>1106 (66.7%)</td>
<td>906 (74.5%)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Living Donor</td>
<td>3395 (54.6%)</td>
<td>1958 (58.6%)</td>
<td>553 (33.3%)</td>
<td>310 (25.5%)</td>
<td></td>
</tr>
<tr>
<td>Duration of Dialysis</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Preemptive</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>0-6 months</td>
<td>1033 (16.6%)</td>
<td>650 (19.5%)</td>
<td>214 (12.9%)</td>
<td>109 (13.9%)</td>
<td></td>
</tr>
<tr>
<td>6-12 months</td>
<td>1048 (16.9%)</td>
<td>554 (16.6%)</td>
<td>274 (16.5%)</td>
<td>220 (18.1%)</td>
<td></td>
</tr>
<tr>
<td>12-18 months</td>
<td>920 (14.8%)</td>
<td>432 (12.9%)</td>
<td>288 (17.4%)</td>
<td>200 (16.5%)</td>
<td></td>
</tr>
<tr>
<td>&gt; 18 months</td>
<td>1958 (31.5%)</td>
<td>789 (23.6%)</td>
<td>648 (39.1%)</td>
<td>521 (42.9%)</td>
<td></td>
</tr>
<tr>
<td>Blood Type (%)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>A</td>
<td>2096 (33.7%)</td>
<td>1298 (38.9%)</td>
<td>462 (27.9%)</td>
<td>336 (27.6%)</td>
<td></td>
</tr>
<tr>
<td>B</td>
<td>730 (11.7%)</td>
<td>354 (10.6%)</td>
<td>162 (9.8%)</td>
<td>214 (17.6%)</td>
<td></td>
</tr>
<tr>
<td>AB</td>
<td>208 (3.4%)</td>
<td>123 (3.7%)</td>
<td>37 (2.2%)</td>
<td>48 (4.0%)</td>
<td></td>
</tr>
<tr>
<td>O</td>
<td>3115 (50.1%)</td>
<td>1514 (45.3%)</td>
<td>987 (59.5%)</td>
<td>614 (50.5%)</td>
<td></td>
</tr>
<tr>
<td>Missing</td>
<td>67 (1.1%)</td>
<td>52 (1.6%)</td>
<td>11 (0.7%)</td>
<td>4 (0.3%)</td>
<td></td>
</tr>
<tr>
<td>Cold Ischemic Time (hrs) among DD recipients</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>0-12</td>
<td>1198 (35.3%)</td>
<td>508 (36.7%)</td>
<td>434 (39.2%)</td>
<td>256 (28.3%)</td>
<td></td>
</tr>
<tr>
<td>12-24</td>
<td>1426 (42.0%)</td>
<td>574 (41.5%)</td>
<td>456 (41.2%)</td>
<td>396 (43.7%)</td>
<td></td>
</tr>
<tr>
<td>&gt; 24</td>
<td>329 (9.7%)</td>
<td>125 (9.0%)</td>
<td>97 (8.8%)</td>
<td>107 (11.8%)</td>
<td></td>
</tr>
<tr>
<td>Missing</td>
<td>442 (13.0%)</td>
<td>176 (12.7%)</td>
<td>119 (10.8%)</td>
<td>147 (16.2%)</td>
<td></td>
</tr>
<tr>
<td>Donor Age, Mean (yrs)</td>
<td>29.0 ± 11.8</td>
<td>31.5 ± 11.9</td>
<td>26.4 ± 11.0</td>
<td>25.6 ± 11.0</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>HLA mismatch, N (%)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>0</td>
<td>181 (2.9%)</td>
<td>130 (3.9%)</td>
<td>40 (2.4%)</td>
<td>11 (0.9%)</td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>274 (4.4%)</td>
<td>195 (5.8%)</td>
<td>64 (3.9%)</td>
<td>15 (1.2%)</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>899 (14.5%)</td>
<td>616 (18.4%)</td>
<td>190 (11.5%)</td>
<td>93 (7.7%)</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>1520 (24.5%)</td>
<td>952 (28.5%)</td>
<td>362 (21.8%)</td>
<td>206 (16.9%)</td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>1091 (17.6%)</td>
<td>509 (15.2%)</td>
<td>317 (19.1%)</td>
<td>265 (21.8%)</td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>1352 (21.8%)</td>
<td>597 (17.9%)</td>
<td>379 (22.9%)</td>
<td>376 (30.9%)</td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>709 (11.4%)</td>
<td>273 (8.2%)</td>
<td>222 (13.9%)</td>
<td>214 (17.6%)</td>
<td></td>
</tr>
<tr>
<td>Study Population N = 6,216</td>
<td>White, Non-Hispanic n = 2922 (53.8%)</td>
<td>White, Hispanic n = 1659 (26.7%)</td>
<td>Black n = 1216 (21.1%)</td>
<td>P-value For Race Difference</td>
<td></td>
</tr>
<tr>
<td>---------------------------</td>
<td>-------------------------------------</td>
<td>-----------------------------------</td>
<td>------------------------</td>
<td>-----------------------------</td>
<td></td>
</tr>
<tr>
<td>Missing</td>
<td>190 (3.1%)</td>
<td>69 (2.1%)</td>
<td>85 (5.1%)</td>
<td>36 (3.0%)</td>
<td></td>
</tr>
<tr>
<td>Panel Reactive Antibody</td>
<td></td>
<td></td>
<td></td>
<td>&lt;0.0001</td>
<td></td>
</tr>
<tr>
<td>&lt; 20%</td>
<td>3455 (55.6%)</td>
<td>1552 (46.5%)</td>
<td>1053 (63.5%)</td>
<td>850 (69.9%)</td>
<td></td>
</tr>
<tr>
<td>20–79.9%</td>
<td>207 (3.3%)</td>
<td>95 (2.8%)</td>
<td>57 (3.4%)</td>
<td>55 (4.5%)</td>
<td></td>
</tr>
<tr>
<td>&gt; 80%</td>
<td>39 (0.6%)</td>
<td>17 (0.5%)</td>
<td>8 (0.5%)</td>
<td>14 (1.2%)</td>
<td></td>
</tr>
<tr>
<td>Missing</td>
<td>2515 (40.5%)</td>
<td>1677 (50.2%)</td>
<td>541 (32.6%)</td>
<td>297 (24.4%)</td>
<td></td>
</tr>
<tr>
<td>Immunosuppression Regimen at Discharge</td>
<td></td>
<td></td>
<td></td>
<td>&lt;0.0001</td>
<td></td>
</tr>
<tr>
<td>Tacrolimus-based regimen</td>
<td>4820 (77.5%)</td>
<td>2802 (74.9%)</td>
<td>1325 (79.9%)</td>
<td>993 (81.7%)</td>
<td></td>
</tr>
<tr>
<td>Cyclosporin-based regimen</td>
<td>724 (11.7%)</td>
<td>462 (13.8%)</td>
<td>151 (9.1%)</td>
<td>111 (9.1%)</td>
<td></td>
</tr>
<tr>
<td>Other regimen</td>
<td>672 (10.8%)</td>
<td>377 (11.3%)</td>
<td>183 (11.0%)</td>
<td>112 (9.2%)</td>
<td></td>
</tr>
<tr>
<td>Induction Base Therapy&lt;sup&gt;6&lt;/sup&gt;</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No induction therapy</td>
<td>882 (14.6%)</td>
<td>436 (13.4%)</td>
<td>238 (14.7%)</td>
<td>208 (17.5%)</td>
<td>0.0032</td>
</tr>
<tr>
<td>Thymoglobulin/ATG</td>
<td>1748 (28.1%)</td>
<td>959 (28.7%)</td>
<td>386 (23.3%)</td>
<td>403 (33.1%)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>IL2-RA</td>
<td>2801 (45.1%)</td>
<td>1484 (42.9%)</td>
<td>864 (52.1%)</td>
<td>503 (41.4%)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Campath</td>
<td>259 (4.2%)</td>
<td>182 (5.5%)</td>
<td>46 (2.8%)</td>
<td>31 (2.6%)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Steroid Only</td>
<td>576 (9.3%)</td>
<td>316 (9.5%)</td>
<td>150 (9.0%)</td>
<td>110 (9.1%)</td>
<td>0.8538</td>
</tr>
<tr>
<td>Delayed Graft Function</td>
<td>408 (6.7%)</td>
<td>177 (5.4%)</td>
<td>96 (5.9%)</td>
<td>135 (11.2%)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Share 35 Policy Era&lt;sup&gt;5&lt;/sup&gt;</td>
<td>2762 (44.4%)</td>
<td>1998 (47.8%)</td>
<td>616 (37.1%)</td>
<td>548 (45.1%)</td>
<td>&lt;0.0001</td>
</tr>
</tbody>
</table>

<sup>1</sup> Body Mass Index  
<sup>2</sup> Glomerulonephritis  
<sup>3</sup> Focal Segmental Glomerulosclerosis  
<sup>4</sup> Estimated Glomerular Filtration Rate  
<sup>5</sup> Post-Sept. 2005 vs. Pre-Sept. 2005  
<sup>6</sup> Numbers may exceed 100% because some patients have more than 1 type of therapy. ATG (Atgam, Anti-thymocyte Globulin); IL2-RA (Interleukin-2 receptor alpha chain)  
<sup>a</sup> p-values < 0.05 for each variable indicate that at least one variable level is significantly different across race/ethnicity
Table 2

Multivariable-adjusted Hazard Ratios for Effect of Hispanic (H) vs. White (W) and Black (B) vs. White (W) Race/Ethnicity on Overall Graft Failure among Deceased and Living Donor Transplant Recipients.

<table>
<thead>
<tr>
<th></th>
<th>Hispanic vs. White (Interaction p-value = 0.6691)</th>
<th>Black vs. White (Interaction p-value = 0.3128)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Model 1 Crude H:W HR (95% CI)</td>
<td>Model 1 Crude B:W HR (95% CI)</td>
</tr>
<tr>
<td>Deceased Donor</td>
<td>0.90 (0.74–1.09)</td>
<td>2.00 (1.70–2.36)</td>
</tr>
<tr>
<td></td>
<td>0.84 (0.68–1.02)</td>
<td>0.84 (0.68–1.03)</td>
</tr>
<tr>
<td>Living Donor</td>
<td>1.06 (0.78–1.43)</td>
<td>2.65 (2.03–3.45)</td>
</tr>
</tbody>
</table>

Interaction p-value shown is for interaction between race/ethnicity and donor type. The interaction p-value was not significant for either Hispanics (vs. whites) or blacks (vs. whites), suggesting that there are no significant differences in the effect of race/ethnicity on graft survival among those who received living vs. deceased donor transplants.

1 Model 1 is crude/unadjusted model and only includes race/ethnicity

2 Model 2 is adjusted for age, sex, peak panel reactive antibody, BMI >85%, blood type, OPO region, etiology of ESRD, Share 35 era, immunosuppression regimen at discharge, induction regimen, and preemptive transplantation.

3 Model 3 is adjusted for age, sex, peak panel reactive antibody, BMI >85%, blood type, OPO region, etiology of ESRD, Share 35 era, immunosuppression regimen at discharge, induction regimen, preemptive transplantation, insurance status, and neighborhood poverty.
### Table 3a
Multivariable-adjusted\(^1\) Hazard Ratios for Effect of Hispanic (H) vs. White (W) and Black (B) vs. White (W) Race/Ethnicity on Overall Graft Failure within strata of Neighborhood Poverty among Deceased Donor Transplant Recipients

<table>
<thead>
<tr>
<th></th>
<th>Hispanic vs. White (Interaction p-value = 0.6691)</th>
<th>Black vs. White (Interaction p-value=0.3128)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Crude H:W HR (95% CI)</td>
<td>Adjusted (^1) H:W HR (95% CI)</td>
</tr>
<tr>
<td><strong>High Neighborhood Poverty</strong></td>
<td>0.77 (0.55–1.09)</td>
<td>0.77 (0.54–1.09)</td>
</tr>
<tr>
<td><strong>Low Neighborhood Poverty</strong></td>
<td>0.89 (0.70–1.13)</td>
<td>0.84 (0.65–1.09)</td>
</tr>
<tr>
<td><strong>Public or Other Health Insurance</strong></td>
<td>0.76 (0.60–0.95)</td>
<td>0.74 (0.58–0.94)</td>
</tr>
<tr>
<td><strong>Private Health Insurance</strong></td>
<td>1.21 (0.83–1.75)</td>
<td>1.10 (0.75–1.63)</td>
</tr>
</tbody>
</table>

Interaction p-value shown is for interaction between race/ethnicity and SES measures. The interaction p-values were not significant for either Hispanics (vs. whites) or blacks (vs. whites), suggesting that there are no significant differences in the effect of race/ethnicity on graft survival among those who lived in a high vs. low poverty neighborhood, and among those with public or other health insurance vs. private health insurance.\(^1\)

\(^1\)The model was adjusted for age, sex, insurance status, neighborhood poverty, peak panel reactive antibody, BMI >85%, blood type, OPO region, etiology of ESRD, Share 35 era and preemptive transplantation.

\(^*\)Interaction p-values are presented for multivariable analyses only.
Table 3b
Multivariable-adjusted Hazard Ratios for Effect of Hispanic (H) vs. White (W) and Black (B) vs. White (W) Race/Ethnicity on Overall Graft Failure within strata of Neighborhood Poverty among Living Donor Transplant Recipients

<table>
<thead>
<tr>
<th></th>
<th>Hispanic vs. White (Interaction p-value p=0.6691)</th>
<th>Black vs. White (Interaction p-value=0.1038)</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Crude H:W HR (95% CI)</td>
<td>Adjusted (^1) H:W HR (95% CI)</td>
<td>Crude B:W HR (95% CI)</td>
</tr>
<tr>
<td>High Neighborhood Poverty</td>
<td>0.98 (0.69–1.39)</td>
<td>0.93 (0.48–1.77)</td>
<td>3.27 (1.92–5.57)</td>
</tr>
<tr>
<td>Low Neighborhood Poverty</td>
<td>1.25 (0.67–2.35)</td>
<td>0.71 (0.48–1.05)</td>
<td>2.29 (1.65–3.17)</td>
</tr>
</tbody>
</table>

\(^1\) The model was adjusted for age, sex, insurance status, neighborhood poverty, peak panel reactive antibody, BMI >85%, blood type, OPO region, etiology of ESRD, Share 35 era and preemptive transplantation.