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Immunologic Benefits of 0-antigen Mismatched Transplants: No Added Boost for Racial and Ethnic Minorities

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Background. Systemic barriers to posttransplant care, including access to immunosuppressant medications, contribute to higher rates of kidney transplant failure in racial minorities. Matching donor and recipient HLA alleles reduce allorecognition, easing reliance on immunosuppression. We hypothesize that 0-antigen mismatch transplants may provide stronger protection against graft loss in racial minorities. **Methods.** We compared adult, single-organ, deceased-donor kidney transplants in the United States from 2007 to 2016 by degree of HLA mismatch (0- versus ≥1-antigen mismatch). We examined time-to-allograft failure, with death as a competing event, using multivariable Weibull models, stratified by recipient race (White versus non-White), and evaluated the interaction between mismatch and recipient race. We used Kaplan-Meier imputation to account for competing risk of death. **Results.** We analyzed 102 114 transplants (median follow-up, 5.6 y; 16 862 graft losses, 18 994 deaths). Zero-antigen mismatch was associated with improved allograft survival (adjusted subdistribution hazard ratio [sHR] 0.80; 95% confidence interval [CI], 0.75-0.85). When stratified by recipient race, the effect of 0-antigen mismatch was more pronounced in White (unadjusted sHR 0.78; 95% CI, 0.72-0.83) versus non-White recipients (sHR 0.88; 95% CI, 0.79-0.99; interaction $P = 0.04$). The differential effect was attenuated after adjusting for covariates (sHR 0.78; 95% CI, 0.73-0.84 versus sHR 0.87; 95% CI, 0.77-0.98; interaction $P = 0.10$). **Conclusions.** Zero-antigen mismatch transplants conferred a 20% risk reduction in allograft loss, which was similar between non-White and White recipients. This may reflect an increased degree of mismatch at other HLA alleles and non-HLA alleles in non-White recipients or because of the extent of systemic barriers to healthcare borne by minority recipients.

(*Transplantation Direct* 2024;10: e1653; doi: 10.1097/TXD.0000000000001653.)

Received 7 March 2024.
Accepted 21 March 2024.
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J.S.C. is supported by the American Kidney Fund (Clinical Scientist in Nephrology award). X.S.C. is supported by the National Institute of Diabetes, Digestive and Kidney Diseases (K23 DK123410) and Stanford University through the McCormick and Gabilan Faculty Award. J.C.T. and M.R.S. were supported by the John M. Sobrato Gift Fund. This work was supported in part by Health Resources and Services Administration contract HHSH250-2019-00001C.
The authors declare no conflicts of interest.
J.S.C. participated in research design, performance, data analysis, drafting the article, and revising the article. G.P., M.R.S., and X.S.C. participated in research design, data analysis, and article revisions. P.A. participated in research design and article revisions. J.C.T. participated in article revisions.
Supplemental digital content (SDC) is available for this article. Direct URL citations appear in the printed text, and links to the digital files are provided in the HTML text of this article on the journal's Web site (www.transplantationdirect.com).
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ISSN: 2373-8731

DOI: 10.1097/TXD.0000000000001653

Patients of racial and ethnic minorities with kidney transplants have a well-documented increased risk of allograft loss.[1-3] Immunologic explanations include the observation that minority patients may have more preformed antibodies against alloantigens and a higher degree of mismatch against the deceased-donor pool.[4,5] Socioeconomic factors, including a greater likelihood of public insurance status, lower income and education, and greater barriers to medication adherence and follow-up appointments, are also associated with allograft loss.[6-9] Improving transplant outcomes for minority transplant recipients is an important priority of research and public policy development.

A possible approach is to change the organ allocation priority for immunologic matching in minority transplant recipients. Transplants in which donor and recipient are fully matched at the A, B, and DR loci of the HLA alleles (known as 0-antigen mismatch) confer a particularly favorable graft survival advantage.[10-12] Opelz et al[13] demonstrated that 3 y posttransplant, 0-antigen mismatch transplants require less immunosuppression. For this reason, kidney allocation protocols in the United States have prioritized 0-antigen mismatch transplants in all recipients regardless of race. We hypothesize that immunologic matching may be more important for minority transplant recipients: as time from transplant increases, socioeconomic barriers to medication adherence may compound for minority transplant

recipients, owing to financial, health literacy, and linguistic challenges.[14-18] Zero-antigen mismatch transplants could, therefore, provide immunologic "insurance" against medication nonadherence that may be especially relevant for minority recipients.

In this article, we hypothesize that 0-antigen mismatch kidney transplants reduce the risk of rejection more for minority recipients than for nonminority recipients. In other words, these transplants mitigate vulnerable patients' obstacles to posttransplant immunosuppressants and monitoring. If true, this would argue that 0-mismatch transplants should be prioritized for minority patients over nonminority patients in the Kidney Allocation System. To explore this hypothesis, we undertake this retrospective analysis of transplant outcomes in a national database.

MATERIALS AND METHODS

Data Set

This study used data from the Scientific Registry of Transplant Recipients (SRTR). The SRTR data system includes data on all donors, waitlisted candidates, and transplant recipients in the United States, submitted by the members of the Organ Procurement and Transplantation Network (OPTN). The Health Resources and Services Administration, US Department of Health and Human Services, provides oversight to the activities of the OPTN and SRTR contractors.[19] The SRTR database also contains death information from the Social Security Death Master File. The data reported here have been supplied by the Hennepin Healthcare Research Institute as the contractor for the SRTR. The interpretation and reporting of these data are the responsibility of the authors, and in no way should they be seen as an official policy or interpretation by the SRTR or the US government.

Cohort

The study cohort comprised adult, deceased-donor, single-organ kidney transplants, that took place in the United States between January 1, 2007, and December 31, 2016 (Figure 1). We limited the cohort to deceased donors because living donation kidney transplants are frequently from related donors who often share more genetic similarity with the recipient than do transplants from deceased donors. We chose this 10-y cohort to allow at least 3 y of posttransplant follow-up data collection.

Exposures and Outcomes

The main exposure was the degree of HLA mismatch at the A, B, and DR loci, categorized as 0-antigen mismatches or ≥1-antigen mismatch. As prior studies demonstrate a dose/response relationship between the number of mismatches and graft outcomes,[20,21] and particular importance of DR matching,[22,23] we also performed separate analyses evaluating mismatch as a continuous variable (range, 0–6 mismatches), and another limiting the exposure to DR matching only (range, 0–2 mismatches). For all analyses, we categorized race as White versus non-White. As 0-antigen mismatched kidney transplants are uncommon, especially in non-White individuals, we chose to collapse all non-White races into one group to preserve statistical power. We performed additional analyses limited to Black recipients.

The primary outcome was time-to-allograft failure from the time of transplantation (death-censored graft failure); a secondary outcome was time to death.[24] We did not examine acute rejection episodes as an outcome because of the poor sensitivity of OPTN data for rejection events.[25]

Covariates

We adjusted the main models sequentially for medical factors, immunologic factors, and social determinants of health. Medical factors included recipient age, sex, dialysis vintage (length of time on dialysis), diabetes, hypertension, donor organ quality (kidney donor risk index, normalized by 2021 values[26,27]), cold ischemia time, and year of transplant (to adjust for differences in allocation policy). Immunologic factors included degree of antibody sensitization (calculated panel-reactive antibody, at time of transplant [0%–80%, 81%–98%, or 99%–100%]) and induction immunosuppression regimen (lymphocyte-depleting versus nonlymphocyte depleting). Socioeconomic factors included recipient

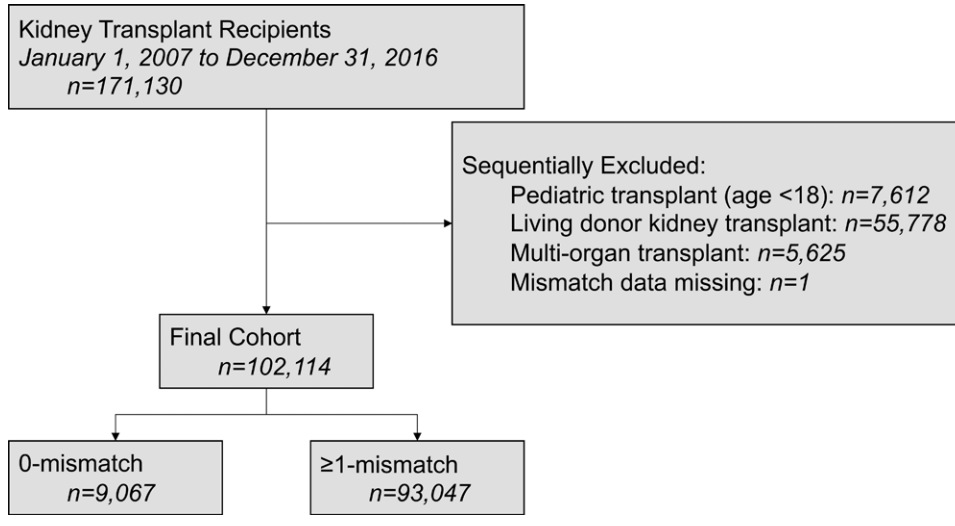

FIGURE 1. Flowchart of inclusion and exclusion criteria.

ethnicity (Latino versus non-Latino), education (some college education versus no college education), and insurance (private insurance, Medicare or Veteran's Affairs versus underinsured, defined as Medicaid, self-pay, or funded by donation). Model details are shown in Table S1 (**SDC,** http://links.lww.com/TXD/A662).

Analysis

Descriptive statistics were used to compare the baseline characteristics of the 2 groups (0- versus ≥1-antigen mismatch), stratified by recipient race. For continuous variables, means and SDs were reported for normally distributed variables and medians and interquartile ranges were reported for nonnormally distributed variables. Other variables were presented as frequencies and percentages. Missing covariates were imputed 5-fold using multiple imputation by chained equations (which uses predictive mean matching for continuous variables, logistic regression for binary variables, and multinomial logistic regression for categorical variables),[28] resulting in 5 imputed covariate data sets.

We included an interaction term for the degree of HLA mismatch by recipient race to examine effect modification by race. To interpret the association between HLA mismatch within each race category, we stratified our analysis by race. Details of each model input are included in Table S1 (**SDC,** http://links.lww.com/TXD/A662).

We used Weibull regression models to estimate the association between the degree of mismatch and time-to-allograft failure. The competing event of death was addressed using the Kaplan-Meier imputation.[29] Because each imputed covariate data set was further imputed 5-fold for the potential censoring times, there were 25 covariate outcome combination data sets. Coefficients were estimated by taking the average across the 25 data sets, and the SEs by Rubin's rule.[30] The Z-test was conducted to test whether there was an interaction between mismatch and race. We first used graphical methods to evaluate model fit and then performed statistical tests for goodness of fit using Cox-Snell residuals[31,32] on 1 of the 25 data sets. Figure S1 (**SDC,** http://links.lww.com/TXD/A662) displays calibration plots for the fitted Weibull models depicting observed versus predicted graft (**Figure S1A, SDC,** http://links.lww.com/TXD/A662) and patient survival probabilities (**Figure S1B, SDC,** http://links.lww.com/TXD/A662). Predicted and observed probabilities fall close to the diagonal line, indicating a good model fit.

Statistical significance was based on 2-sided *P* values of <0.05. Analyses were performed in SAS version 9.4 (SAS Institute Inc, Cary, NC) and R Studio version 2022.07.0 Build 548 (R Foundation for Statistical Computing, Vienna, Austria).

Ethics

The Stanford University Institutional Review Board approved this study (protocol No. 40876) in adherence with the Declaration of Helsinki. The clinical and research activities being reported are consistent with the Principles of the Declaration of Istanbul as outlined in the "Declaration of Istanbul on Organ Trafficking and Transplant Tourism." We adhered to the Strengthening the Reporting of Observational Studies in Epidemiology reporting guidelines.

RESULTS

Baseline Characteristics

We analyzed 102 114 transplants with a median follow-up time of 5.6 y, in which 16 862 graft losses and 18 994 deaths occurred. Nine thousand sixty-seven transplants had 0-antigen mismatches (7628 in White and 1439 in non-White recipients) and 93 047 transplants had ≥1-antigen mismatch (51 626 in White recipients and 41 421 in non-White recipients). The non-White recipient group was composed mainly of Black recipients (80%), with Asian-American recipients as the second largest group (16%; **Table S2, SDC,** http://links.lww.com/TXD/A662).

Table 1 shows the baseline characteristics of the cohort. In both White (58%) and non-White recipients (42%), 0-antigen mismatched transplants occurred in younger individuals (mean age 52 versus 54 y in White recipients; 51 versus 52 y in non-White recipients), who were more likely to be women (49% versus 46% in White recipients; 52% versus 40% in non-White recipients), have a shorter dialysis vintage (median 2.4 versus 3.6 y in White recipients; 3.3 versus 4.6 y in non-White recipients), and have higher levels of preformed antibodies (9% versus 3% in highest calculated panel-reactive antibody category in White recipients; 12% versus 3% in non-White recipients). Kidneys used in 0-antigen mismatched transplants had lower kidney donor risk index scores, meaning that they were from higher-quality donor organs (median [interquartile range] 0.83 [0.68–1.0] versus 0.93 [0.76–1.16] in White recipients; 0.86 [0.71–1.07] versus 0.95 [0.77–1.17] in non-White recipients).

Main Analysis: ABDR Mismatch, 0- Versus ≥1-antigen

Figure 2 illustrates the main results. In all recipients, 0-antigen mismatched transplantation was associated with improved allograft survival (subdistribution hazard ratio [sHR] 0.80; 95% confidence interval [CI], 0.75-0.85), with and without adjustment (Figure 2A). In the unadjusted analyses, the effect of 0-antigen mismatch on graft loss in White recipients (sHR 0.78; 95% CI, 0.72-0.83) was more pronounced when compared with non-White recipients, with a *P* value for interaction between mismatch and recipient race of 0.04 (sHR 0.88; 95% CI, 0.79-0.99). After adjusting for potential confounders, this differential effect was attenuated, with a *P* value for interaction no longer meeting statistical significance ($P = 0.10$; sHR 0.78; 95% CI, 0.73-0.84 versus sHR 0.87; 95% CI, 0.77-0.98; **Table S3, SDC,** http://links.lww.com/TXD/A662). A supplemental analysis limited to Black recipients revealed a similar association between 0-antigen mismatch and graft loss (adjusted sHR 0.88; 95% CI, 0.77-0.99) as was seen in the non-White recipient group (**Figure S2 and Table S4, SDC,** http://links.lww.com/TXD/A662).

Unadjusted models showed a weak association between 0-antigen mismatched transplants and death in the full cohort (unadjusted sHR 0.91; 95% CI, 0.87-0.96), White recipients (unadjusted sHR 0.90; 95% CI, 0.85-0.95), and non-White recipients (unadjusted sHR 0.99; 95% CI, 0.87-1.12). With adjustment, this effect was fully attenuated (Figure 2B).

Supplemental Analysis

Unadjusted analyses treating ABDR mismatch as a continuous variable demonstrated a 7% increase in the risk of graft loss for White recipients with each additional degree

TABLE 1.

Characteristics of kidney transplant recipients, stratified by recipient race (White vs non-White)

	White recipients (N = 59 254)		*P*	Non-White recipients (N = 42 860)		Missing data (N = 102 114)
	0-antigen mismatch (N = 7628)	≥1-antigen mismatch (N = 51 626)		0-antigen mismatch (N = 1439)	≥1-antigen mismatch (N = 41 421)	n (%)
No. of mismatches, mean (SD)	NA	4.2 (±1.2)		NA	4.6 (±1.1)	0
Age, mean (SD)	52 (±13.4)	54 (±13.6)	<0.0001	51 (±12.4)	52 (±12.7)	0 (0%)
Sex, male, n (%)	3909 (51%)	32 193 (62%)	<0.0001	692 (48%)	24 700 (60%)	0 (0%)
Dialysis vintage, y, median (IQR)	2.4 (1.3–4.2)	3.6 (2.1–5.6)	<0.0001	3.3 (1.9–5.4)	4.6 (2.8–6.8)	8600 (0.08%)
Hypertension, n (%)	5382 (86%)	36 620 (87%)	0.017	1045 (90%)	29 415 (91%)	20 374 (20%)
Diabetes, n (%)	2535 (34%)	17 954 (35%)	0.015	514 (36%)	15 046 (37%)	552 (<0.01%)
ESKD diagnosis, n (%)			<0.0001			503 (<0.01%)
Glomerulonephritis	1837 (24%)	11 545 (22%)		365 (25%)	8640 (21%)	
Diabetes	2001 (27%)	14 243 (28%)		371 (26%)	11 306 (27%)	
Hypertension	1206 (16%)	9583 (19%)		482 (34%)	15 072 (37%)	
Other	2497 (33%)	15 955 (31%)		212 (15%)	6296 (15%)	
KDRI, 2021, median (IQR)	0.83 (0.68–1.00)	0.93 (0.76–1.16)	<0.0001	0.86 (0.71–1.07)	0.95 (0.77–1.17)	23 (<0.01%)
Sensitization, n (% cPRA)			<0.0001			17 (<0.01%)
0%–80%	5616 (74%)	44 749 (87%)		1006 (70%)	35 815 (86%)	
81%–98%	1340 (17%)	5435 (10%)		256 (18%)	4362 (11%)	
99%–100%	672 (9%)	1431 (3%)		177 (12%)	1238 (3%)	
Induction (% nonlymphocyte depleting)	3483 (46%)	22 667 (45%)	0.010	644 (46%)	17 321 (43%)	1795 (0.02%)
Maintenance (% non-CNI based)	166 (3%)	892 (3%)	0.243	25 (3%)	608 (3%)	44 265 (43%)
Ethnicity, Latino, n (%)	1637 (21%)	14 610 (28%)	<0.0001	11 (0.8%)	185 (0.5%)	0 (0%)
Education, n (% no college)	3643 (51%)	26 377 (54%)	<0.0001	671 (51%)	19 659 (51%)	6495 (0.06%)
Insurance status, n (% underinsured)	385 (5%)	2742 (5%)	0.335	108 (8%)	2422 (6%)	7 (<0.01%)

Underinsured, includes Medicaid, Children's Health Insurance Program, self-pay, funded by donation, other public insurance, and free. Means and SDs reported for continuous, normally distributed variables and medians and interquartile ranges reported for continuous nonnormally distributed variables.
CNI, calcineurin inhibitor; cPRA, calculated panel-reactive antibody; ESKD, end-stage kidney disease; IQR, interquartile range; KDRI, kidney donor risk index.

of mismatch compared with 4% in non-White recipients (unadjusted sHR 1.07; 95% CI, 1.05-1.08 versus 1.04; 95% CI, 1.02-1.06; interaction P = 0.01; **Table S3, SDC,** http://links.lww.com/TXD/A662). There was a 3% increase in the risk of death in both White and non-White recipients (unadjusted sHR 1.03; 95% CI, 1.02-1.04 versus 1.03; 95% CI, 1.01-1.05; interaction P = 0.82) for each additional degree of ABDR mismatch. Each additional DR mismatch was associated with a 16% increase in graft loss in White recipients compared with an 11% increase in non-White recipients (unadjusted sHR 1.16; 95% CI, 1.13-1.20 versus 1.11; 95% CI, 1.07-1.14; interaction P = 0.01). The risk of death was increased by 9% in White versus 8% in non-White individuals (unadjusted sHR 1.09; 95% CI, 1.06-1.11 versus 1.08; 95% CI, 1.04-1.12; interaction P value = 0.75) for each additional degree of DR mismatch.

DISCUSSION

In this article, we confirmed that 0-antigen mismatched transplants were associated with a 20% reduction in risk of graft loss, but this effect was not enhanced in non-White recipients compared with that in White recipients. If anything, there was a slightly weaker protective effect of 0-antigen mismatch in non-White recipients, although this effect was not statistically significant after adjustment for possible confounders in the main analysis. This effect was similar when analyzing ABDR mismatch as a continuous variable and when limited to DR mismatch. Similar to previous studies, we did not find an association between 0-antigen mismatched transplants and death after adjustment for covariates.[22,33]

A few explanations for the absence of additional benefits in non-White recipients are possible. One is immunologic. Despite the so-called 0-antigen mismatched transplants, non-White individuals may have a greater number of mismatches at other HLA alleles (eg, HLA-C, HLA-DQ, and HLA-DP), as well as minor antigen mismatches not routinely tested for. This is plausible as individuals of more recent African ancestry have more polymorphic HLA alleles when compared with individuals of other ancestries.[5,34] Consequently, even when receiving a 0-antigen mismatched kidney transplant, non-White individuals may have a greater degree of mismatch with their donor when compared with White recipients, and this may explain the trend toward a less protective effect of 0-antigen mismatch. However, the definition of race in registry data is mixed and problematic; thus, the clustering of HLA haplotypes may not fall into the racial categories in the SRTR database.

Another explanation is medical. Non-White recipients of 0-mismatch kidney transplants received higher kidney donor profile index (KDPI; ie, worse quality) organs compared with White recipients. The KDPI is calculated from a combination of donor factors, including age, BMI, race/ethnicity, and donor history of hypertension and diabetes, among others. Non-White individuals are more likely to receive kidneys from non-White donors[4,34,35] with higher KDPI scores. The inclusion of the Black race in the KDPI calculation is controversial[36,37]; however, a subset of kidneys from Black donors may contain a high-risk APOL1 allele. The presence of these alleles has been associated with a higher risk of graft loss,[38-40] and may therefore counteract the protection afforded by better immunologic matching.

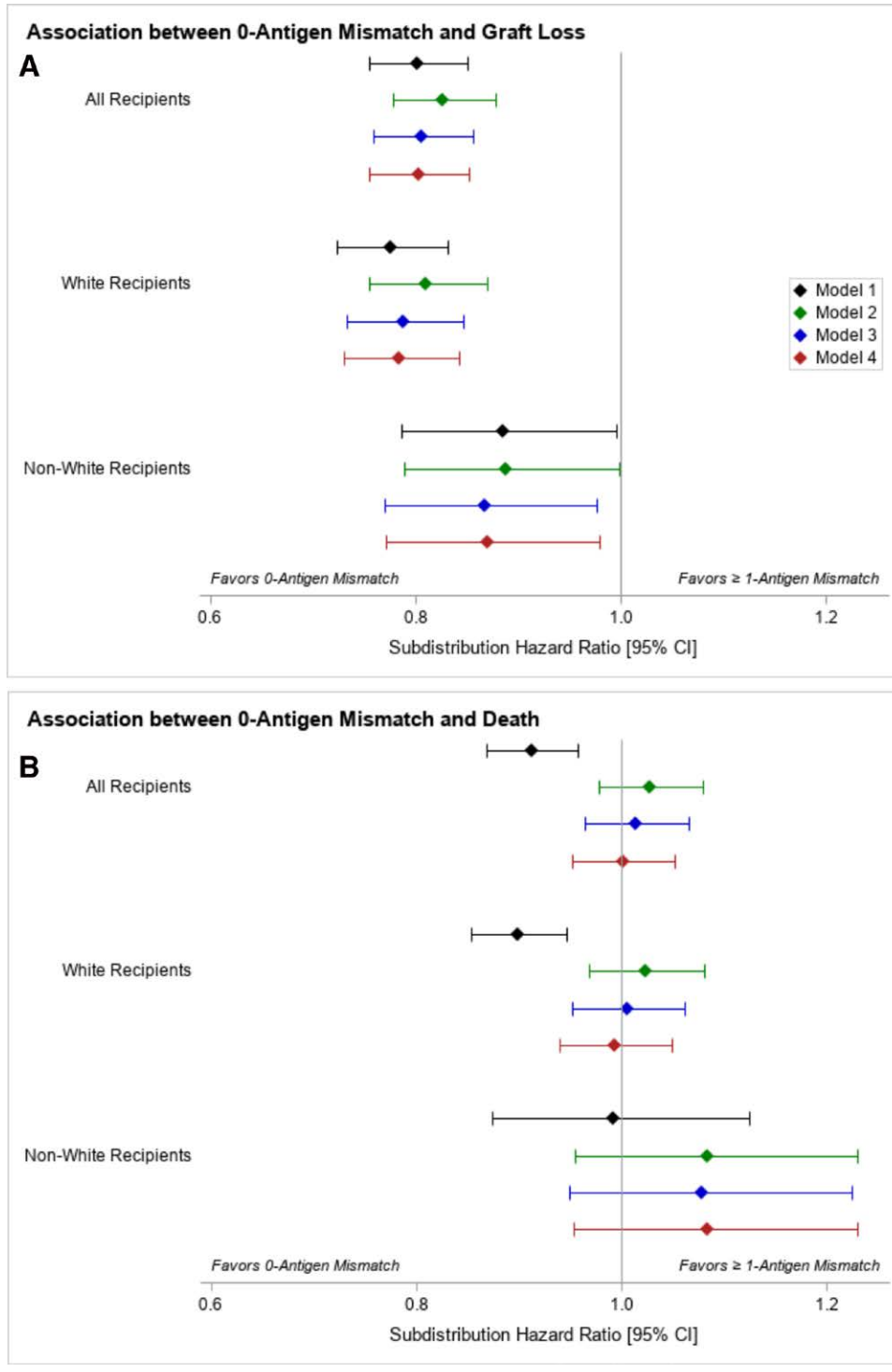

FIGURE 2. Associations between 0-antigen mismatch (reference group: ≥1-antigen mismatch) and graft loss (A) and death (B), as estimated by Weibull models, in all recipients, White recipients, and non-White recipients. The competing event of death was addressed using Kaplan-Meier imputation. Model 1: base model. Model 2: model 1 + recipient medical and transplant factors: age, sex, dialysis vintage, diabetes, hypertension, kidney donor risk index, cold ischemia time, and transplant year. Model 3: model 2 + immunologic factors: calculated panel-reactive antibody and induction immunosuppression. Model 4: model 3 + socioeconomic factors: recipient ethnicity, education, and insurance. CI, confidence interval.

Yet a third possible explanation for our findings is that the barriers to care experienced by racial minority communities are simply too great to be overcome by the benefits provided by well-matched transplants. Two plausible mediators are health literacy and insurance status. Health literacy is lower in Black patients referred for kidney transplant[14] and is associated with both education and income levels.[15,16] Individuals with lower health literacy are more likely to make "medication trade-offs" (defined as choosing to spend money on other expenses over medications), increasing the risk of medication nonadherence.[17,18] Reduced ability to navigate the complexity of posttransplant care, combined with the

challenges of receiving appropriate care (ie, insurance coverage for immunosuppressant medications), place minority patients at a serious disadvantage. Interestingly, a retrospective analysis of >5000 Canadian kidney transplants found little difference in graft survival between White and Black recipients, a finding which contrasts sharply with US outcomes.[41] In Canada, patients have broader access to immunosuppressant medications through provincial drug programs or employer-sponsored plans. This finding points to systemic, rather than biological, factors contributing to racial disparity in kidney transplant outcomes. The US Medicare's lifetime coverage of immunosuppressive medications thus serves as a step in the right direction toward mitigating these disparities.

Limitations of our analysis include the retrospective and observational nature of these data. The collection of recipient races in the SRTR database is not standardized between centers and may be inconsistent. Another important limitation is the categorization of recipients as White and non-White. Because of the rare nature of 0-antigen mismatched transplants, we were unable to study recipient groups with more granularity while maintaining statistical power; however, a supplemental analysis limited to Black recipients confirmed the results of the primary analysis. We cannot rule out the presence of unmeasured confounding variables in our study.

CONCLUSION

In an era of contemporary immunosuppression, 0-antigen mismatched transplants are associated with protection against graft loss but not against death. This protection is present in both White and non-White recipients to the same extent. As the OPTN works toward a continuous distribution system to enhance equity while maintaining optimal kidney transplant outcomes, the results of this study suggest that enhancing the priority given to 0-antigen mismatched transplants would not enhance equity for minority transplant recipients.

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