Racial Inequality in the Living Donor Kidney Transplant Opportunity Structure

Jonathan Daw

August 17, 2011

Abstract. Previous research has shown that the very large racial disparities in kidney transplantation outcomes in the U.S. are explained by differential rates of living donor kidney transplants. But what explains these disparities? This paper uses data on the attributes of the kidney transplant waiting list and population data on the distribution of biologically-informed kinship ties and health statuses to investigate the likely distribution of suitable living donors within the kinship networks of persons on the kidney transplant waiting list. The results suggest that black-white disparities in living donor kidney transplantation are not the result of group differences in the availability of suitable donors in their kinship networks. Given the sparse number of potential donors most transplant candidates have evaluated, however, it is likely that the higher probability that white kin are suitable donors is a major determinant of racial differences in living donor kidney transplantation rates.

a: Institute of Behavioral Sciences and Institute for Behavioral Genetics, University of Colorado – Boulder. Jonathan.Daw@colorado.edu.

The data reported here have been supplied by the United Network for Organ Sharing as the contractor for the Organ Procurement and Transplantation Network. The interpretation and reporting of these data are the responsibility of the author(s) and in no way should be seen as an official policy of or interpretation by the OPTN or the U.S. Government.
**Introduction**

End-stage renal disease places a large and increasing burden on the health of the U.S. populace and disproportionately affects African Americans, primarily the result of African Americans’ higher prevalence of diabetes, hypertension, and overweight (Norris and Agodoa 2005). Primary treatments for ESRD include dialysis and kidney transplantation, of which the latter is the medically preferred treatment (e.g., Danovitch and Cecka 2003). Among those receiving transplants, living donor kidney transplantation (LDKT) is associated with substantially better medical outcomes than deceased donor kidney transplants (DDKTs; Davis and Delmonico 2005; Kasiske and Bia 1995; Mange et al. 2001). The kidney transplantation system has long produced substantial racial inequalities in rates and timing of kidney transplants, particularly for LDKTs – in recent years, African Americans have been less than half as likely as whites to experience this outcome (Meier-Kriesche et al. 2000). Although the majority of LDKTs come from recipients’ kin, to date no research on the determinants of racial inequality in LDKT has examined the role of kinship structure and attributes in the production of racial differences in LDKT.

Obtaining an LDKT is a four-step process. First, one must have access to a medically suitable living donor. One’s LDKT opportunity structure – the distribution of suitable potential kidney donors – is based on one’s kinship and friendship networks. Second, a given alter must be agree to be evaluated for donation once they and the candidate have discussed the possibility of donation. Third, the potential donor must be deemed psychologically, medically, and genetically suitable to donate a kidney to the patient. Finally, conditional on a favorable evaluation, the donation must actually occur.
Kinship structure and characteristics influence patients’ LDKT opportunity structure in a number of ways. First and most obviously, larger families include more potential kidney donors. Second, donors are evaluated in part by their degree of genetic match with the transplant candidate, so the proportion of close genetic relatives will influence one’s transplant prospects. Third, donors must be sufficiently healthy to donate.

Race is well known to be related to family structure (Angel and Tienda 1982; Cohen and Casper 2002; Hofferth 1984) and a wide range of health statuses (Blackwell et al. 2002; Elo and Preston 1997; Kelley-Moore and Ferraro 2004; Manton and Gu 2001; Williams 2005), so these represent candidate mechanisms to explain black-white differences in living donor transplantation.

Some additional genetic and immunological mechanisms embedded within kinship structures may help to explain racial inequalities in LDKT as well. In the U.S., black populations have greater overall genetic diversity than whites (Liu et al. 2006; Prugnolle et al. 2005a; Prugnolle et al. 2005b), which could lower the probability of a sufficient genetic match between a patient and kin conditional on their expected genetic relationship. Finally, racial differences in immunological reactivity (Cooper et al. 1995), which influences the chances of immediate rejection, could partially explain black-white differences LDKT.

This paper investigates black-white differences in the opportunity structure for LDKT, estimating group differences in kinship size, genetic relationship structure, kin health statuses, immunological sensitivity, and the probability of a genetic match by race. Based on data on the characteristics of kidney transplant candidates, other population data, and a simulation exercise, the present results suggest that blacks and whites actually
have approximately the same probability (60%) of having one or more suitable living
donors in their kinship networks. While each white kin is more likely to be a suitable
donor, the larger average size of black kinship networks counterbalances this difference.
These results suggest that far fewer ESRD patients are obtaining LDKTs than could do
so, and that factors influencing the commencement and nature of the living donor search
process are likely responsible for black-white differences in rates of LDKT. Finally,
given the relatively sparse number of potential donors typically evaluated for donation
(Weng et al. 2010), the higher probability that a given white alter will be deemed a
suitable donor may help explain white-black living donor differentials.

Background

Racial Inequality in the Transplantation System

Kidney disease is an increasing source of morbidity and mortality in the U.S.,
driven in large part by population increases in the prevalence of hypertension, diabetes,
and overweight (Daumit and Powe 2001; Malek et al. 2011; Norris and Agodoa 2005).
Kidney transplants necessarily involve a donor and a recipient, and there are two major
types of donors – the deceased and the living. Although the number of both types of
transplants has grown since the beginning of widespread transplantation in the mid-
1980s, the number of transplants has not kept pace with the number of transplant
candidates. Under these Malthusian conditions, the kidney transplant waiting list has
grown with the prevalence of ESRD, resulting in quickly lengthening waitlists for
kidneys for transplantation. By the end of 2008, the number of patients awaiting kidney
transplants had grown to 85,440, more than a 500% increase since 1988 and far
outstripping U.S. population growth.
Living donor kidney transplants (LDKTs) are associated with substantially better post-transplant outcomes than are deceased donor kidney transplants (DDKTs; Davis and Delmonico 2005; Kasiske and Bia 1995; Mange et al. 2001; Reese et al. 2009). Whereas deceased donor kidneys are allocated to ESRD patients on a transplant waitlist according to a priority algorithm, LDKTs are obtained more informally, typically from a donor in the candidate’s kinship or friendship networks who is sufficiently healthy and genetically compatible with the intended recipient to donate a kidney. In addition to the advantages of LDKTs (compared to DDKTs) for patient survival and organ rejection, they also typically involve a much shorter waiting period.

The burden of the increasing difficulty of obtaining a kidney transplant has fallen disproportionately on African Americans, who are more likely than whites to need a kidney transplant, and much less likely to obtain one. African Americans are less likely to be evaluated for transplantation if they develop ESRD, are less likely to be placed on the waitlist if they are evaluated, and typically have far longer waits for a deceased donor transplant than do white transplant candidates (Epstein et al. 2000; Hall et al. 2011). Longer periods of dialysis are associated with worse post-transplantation outcomes and higher pre-transplant mortality rates (Eckhoff et al. 2007; Gordon et al. 2010), contributing to African American disadvantages in the kidney transplantation system (Malek et al. 2011). Finally, African Americans are far less likely than whites to obtain a living donor kidney transplant. Compared to white patients, black patients are less likely to have a potential donor evaluated for donation, and are less likely to obtain a living donor transplant if they do have a potential donor (Weng et al. 2010). In fact, a recent
analysis (Daw 2011) suggests that in the last decade racial differences in rates of LDKT are primarily responsible for overall racial inequality in transplantation rates.

**Studies of Living Kidney Donation**

Research on racial disparities in LDKT has focused on the determinants and outcomes of potential kidney donors who were brought into particular transplant centers for evaluation. First, much work investigates who has a potential donor evaluated for donation (Barnieh et al. 2011; Gordon 2001; Rodrigue et al. 2008; Zimmerman et al. 2006). These studies find that many candidates do not believe that they have anyone to discuss donation with (Barnieh et al. 2011). Additionally, while many candidates do have relatives and friends offer to be evaluated for donation, the majority of these offers are refused (Gordon 2001). A major concern among those who refuse such offers is concern for the risks posed to the potential donor. However, another study (Reese et al. 2009) find that younger candidates and those with higher yearly incomes were more likely to have a potential donor evaluated, and that whites were more than twice as likely as blacks to have had a potential donor evaluated. Furthermore, most candidates who do have a donor evaluated have two or fewer potential donors evaluated (Weng et al. 2010), suggesting that the vast majority of transplant candidates do not explore the full range of their kinship networks when seeking an LDKT.

Once a candidate-potential donor pair agrees to be evaluated for transplantation, here are two major obstacles to be overcome in this process: medical and immunological barriers, and procedural barriers. Concerning the latter, a recent study (Clark et al. 2008) found that the potential donors of patients with higher levels of instrumental social support were more likely to complete the full living donor evaluation process, which is a
major site of racial inequality in LDKT. For those who are evaluated, many are excluded for poor health or poor immunological compatibility with the potential recipient, and African American potential donors are more likely to have this result (Lunsford et al. 2007; Reeves-Daniel et al. 2009).

These findings suggest a number of potentially important mechanisms of racial inequality in LDKT rates. First, they strongly indicate that transplant candidates do not have their full networks evaluated for transplantation. Only about half of transplant candidates have any donors evaluated, and the majority of those who do so have only one evaluation. Second, this suggests that racial differences in kin health could play a major role in white and black transplant candidates’ transplantation prospects, as could donor evaluation completion rates, racial differences in the probability of immunological compatibility, and the probability of having any potential donors evaluated.

However, these studies share some major limitations. Most importantly, none contain indications of the full distribution of kinship ties and disqualifying conditions for donation among the social networks of white and black transplant candidates. Although it is unlikely that racial differences in this ‘opportunity structure’ explain the entirety of racial differences in LDKT, this should be the starting point for any analysis of racial inequality in the LDKT system.

**Determinants of the Living Donor Kidney Transplant Opportunity Structure**

Because the vast majority of LDKTs involve kin donors, racial differences in the properties of their kinship networks are a prime candidate to explain racial differences in LDKT. Figure 1 illustrates the factors hypothesized to influence the probability of LDKT. This figure separates the mechanisms influencing the living kidney donation process into
two major categories: factors influencing the LDKT opportunity structure, and factors influencing the probability of LDKT given one’s opportunity structure. Although this research investigates the first set of determinants only, in the following section the relevance of each of these factors for one’s LDKT opportunity structure will be discussed alongside previous findings on racial differences in these factors.

Factors Influencing the LDKT Opportunity Structure

*Kinship network size.* First, the number of living alters in one’s kinship network will be positively related to one’s prospects for an LDKT. All else equal, larger families will be associated with a more favorable LDKT opportunity structure, and racial differences in the distribution of kinship size are a potential explanatory mechanism for racial differences in LDKT. However, the literature on racial differences in kinship network structure is surprisingly sparse. The vast majority of the literature on race and kinship explores differences in household co-residence (e.g., Angel and Tienda 1982; Hofferth 1984), contact (e.g., Raley 1995), and support (e.g., Mazelis and Mykyta 2011; Sarkisian and Gerstel 2004) instead of the entire kinship structure itself. Although all of these variables may be related to kinship size and are important in their own right, in absence of a detailed literature on these factors this connection cannot be assumed. Nonetheless, given that African Americans have larger households on average (Choi 1991; Kamo 2000; Peek et al. 2004), and higher fertility rates (Census 2011), it is likely that African Americans have larger kinship networks on average than whites, which could prove a source of advantage in the LDKT opportunity structure.

*Genetic structure of kinship.* Second, because two forms of genetic similarity (red blood type and human leukocyte antigen compatibility, discussed below in detail) are
associated with better transplantation outcomes and are used by medical staff to
determine donor suitability, the structure of genetic relationships in one’s kinship
networks is also an important determinant of one’s opportunity for LDKT. One has a
higher probability of sharing genes in common with close genetic relatives (e.g., full
siblings) than more biologically distant ones (e.g., cousins). Although the sparseness of
the literature on racial differences in kinship structures limits what is known, the
likelihood of larger average kinship networks among African Americans would also
suggest that they have more close genetic relatives on average than whites.

*Kin Health Status.* Third, before an LDKT can occur, potential living donors are
evaluated on a range of medical and psychological factors to determine their suitability
for donation. The goal of these evaluations is to ensure that the potential donor is capable
of making the donation decision, is doing so without coercion, and can donate a kidney
with minimal risk to the donor and maximum potential benefit to the recipient. Racial
patterns of psychiatric conditions and morbidity are more complicated than is often
recognized. Although African Americans are subject to greater mortality rates (Rogers
1992) and higher morbidity overall (Fiscella et al. 2000; Williams and Collins 1995),
there is considerable variability in racial disparities associated with specific medical
conditions. For instance, although African Americans have higher prevalences of
hypertension (Hajjar and Kotchen 2003), diabetes (Cowie et al. 2006), and obesity (Ford
et al. 2011), whites have higher prevalences of many psychiatric disorders (Kessler et al.
1994), chronic obstructive pulmonary disease (Bang et al. 2009), asthma (McHugh et al.
2009), and breast cancer (Ward et al. 2004). Furthermore, these diseases are not
independent and are rarely studied jointly. However, due to the overall greater burden of
morbidity on African Americans, a black disadvantage in rates of kin contraindications for donation is predicted.

*Genetic compatibility.* Fourth, African Americans are known to have greater genetic diversity than do whites in the United States, a result of historical migration patterns (Liu et al. 2006; Prugnolle et al. 2005a; Prugnolle et al. 2005b) and maintained by ongoing racial homogamy in the U.S., which could result in a disadvantage for blacks in their LDKT prospects. For kidney transplantation, two types of genetic compatibility are especially relevant – red blood cell type (measured by one’s *ABO* genotype), and white blood cell type (measured by one’s *HLA*-A, *HLA*-B, and *HLA*-DR genotypes). These genes play key roles in the human immune system and accordingly structure the probability of organ rejection, in which a transplanted kidney is attacked by the body’s immune system.

These genotypes structure the production of red blood cell (*ABO*) and white blood cell (*HLA*) antigens, which the immune system employs to differentiate host from foreign cells. Cells whose antigens do not contradict the host’s are ‘histocompatible’ and trigger no immune response; cells whose antigens differ from the host’s do trigger a response. Furthermore, certain pairs of *ABO* and *HLA* genotypes are ‘serologically equivalent,’ meaning that the immune system cannot differentiate them. A familiar example is O type blood, which is the ‘universal donor’ blood type because it produces no antigens and therefore triggers no immune response when given to others in blood transfusions.

Racial differences in the distribution of *ABO* and *HLA* genes are thought to explain some portion of racial inequality in the cadaveric transplantation system (Higgins
and Fishman 2006; Malek et al. 2011; Navaneethan and Singh 2006; Vamos et al. 2009). Although progress is being made in overcoming it, the ‘ABO barrier’ is a major obstacle to successful transplantation, sometimes triggering an immediate and devastating immune response when crossed (Nelson et al. 1992). While the effects of HLA mismatch are less severe with modern immunosuppression technology (Murphey and Forsthuber 2008; Su et al. 2004), higher HLA matches are nonetheless associated with improved post-transplantation survival prospects. Accordingly, racial differences in the probability of genetic similarity conditional on overall genetic relationship could partially explain racial differences in LDKT.

Immunological presensitization. Fifth, ESRD patients vary widely in the probability that another person’s cells will trigger a severe immune response, known as hyperacute kidney rejection. In the transplantation literature this probability is defined by Panel Reactive Antibody, or PRA, scores, which are a proxy for the probability of a positive crossmatch. Positive crossmatching occurs when a transplant recipient has antibodies to foreign antigens in the donated organ. As a primary mechanism of disease immunity, antibodies are produced by the body as a defense against cells displaying specific antigens. When cells displaying these antigens are encountered again, antibodies attack them much more rapidly and effectively than the body’s baseline immune responses. As such, until recently transplanting a kidney into an ESRD patient who has already produced antibodies to the donor’s antigens resulted in nearly guaranteed and immediate kidney rejection. Lately therapies designed to avert this outcome have shown some promise (Haririan et al. 2009) but still lag far behind non-crossmatched transplants in patient and graft survival prospects.
African Americans on average have higher PRA scores than whites – in one early study, African Americans ESRD patients had an average score of 15% whereas white patients averaged 6%. Several factors may help to explain this. As with all antibodies, antigen presensitization is associated with prior exposure to foreign antigens. The primary mechanisms through which this occurs are prior transplantations, blood transfusions, and pregnancy (Leffell et al. 1997). Blood transfusion history may represent a major source of immunological presensitization disadvantage for African Americans (Kerman et al. 1992). Similarly, prior transplantation creates a higher likelihood of presensitization (Cooper et al. 1995). Finally, higher fertility, especially with different partners (Census 2011; Harknett and Knab 2007), could create racial differences in PRA as well.

In sum, I expect that African American transplant candidates will on average have larger kinship networks with higher counts of close genetic relatives, providing a source of advantage in the LDKT opportunity structure. However, I also expect that white transplant candidates will have healthier kin on average, a higher probability of HLA and ABO histocompatibility with their kin, and a lower probability of positive crossmatches. How these factors combine to structure racial differences in the LDKT opportunity structure is the subject of this research.

Factors Influencing the Probability of LDKT Given the LDKT Opportunity Structure

A number of processes likely mediate the translation of opportunity structures into LDKT outcomes. While these processes are not directly explored in the present analysis, they will prove helpful in understanding racial differences in LDKT conditional on opportunity structure.
The health care system. First, in order for the opportunity for LDKT to be translated into an LDKT outcome, assistance in navigating the bureaucracies and processes available in the kidney transplantation system will usually be required. For instance, a recent retrospective study found that black patients were less likely to recruit potential donors and, conditional on recruitment, less likely to complete a LDKT (Weng et al. 2010). It could be that differential promotion of and guidance in the LDKT process on the part of the health care providers could explain this difference.

Knowledge of and interest in transplantation. Much medical research on racial differences in transplantation focus on the role of racial differences in knowledge of, and interest in, transplantation (Navaneethan and Singh 2006). Thus patient preferences and beliefs are a central focus of the medical literature on disparities in kidney transplantation and a frequently cited site of potential intervention (Rodrique et al. 2006; Waterman et al. 2006). However, the evidence on racial differences in these factors is mixed (Alexander and Sehgal 2001; Ayanian et al. 1999; Malek et al. 2011). Although perhaps overemphasized in the medical literature on kidney transplantation disparities, beliefs, preferences, and knowledge of transplantation is a theoretically plausible mediator of the relationship between LDKT opportunity and actual LDKT.

Kin relations. Finally, a major and understudied potential mediator of the relationship between LDKT opportunity structures and actual LDKTs is the nature of family relationships. Sociologically, LDKTs are a gift, and an unusually meaningful one. As with all gifts, LDKTs are passed across and potentially shape relations between giver and receiver and are usually subject to norms of reciprocity. Research on social support in black and white families suggests that they differ in the character and degree of
support. For instance, while it is commonly claimed that racial and ethnic minorities have more closely knit kinship networks (Aschenbrenner 1975; Martin and Martin 1985; Stack 1974), other work finds that whites exchange assistance with greater frequency (Cooney and Uhlenberg 1992; Eggebeen 1992; Goldscheider and Goldscheider 1991; Hofferth 1984; Hogan et al. 1993; Hoyert 1990; Lee and Aytac 1998; Roschelle 1997), although the pattern differs for financial and instrumental support (Lee and Aytac 1998; Roschelle 1997; Sarkisian and Gerstel 2004). There is also evidence that black families tend to emphasize same-generation ties more than whites, while white families place greater emphasis on cross-generational ties (Johnson 2000; Johnson and Barer 1990; Johnson and Barer 1995). These relationship patterns by race may structure the probability of seeking or accepting LDKTs from one’s kinship network.

In general, gifts are subject to strong norms of reciprocity, yet rarely can a gift of the magnitude of another’s organ be adequately be repaid, which potentially crates a creditor/debtor relationship between the kidney donor and recipient. Fox and Swazey’s (1978, 1992; see also Healy 2006) seminal work on the subject termed this the “tyranny of the gift” due to the strains such an extraordinary gift places on the relationship between donor and recipient. Transplant candidates’ willingness to accept such a gift may fundamentally depend on their relationships with their kin and their belief in their ability to weather such potential tyrannies. As with all requests and offers for assistance, there are patterned expectations for resource exchanges (Bengtson et al. 1996; Lindblad-Goldberg 1987; Miller-Cribbs and Farber 2008; Neighbors 1997; Nelson 2000; Stack 1974; Tracy 1990), and one’s ability to fulfill reciprocal exchange relations may influence one’s willingness to accept assistance. Furthermore, there is substantial
evidence that these familial exchange norms are of particular importance to African Americans due to traditional norms of mutual family support in impoverished circumstances (Malson 1983; Martin and Martin 1985; McAdoo 1982; Miller-Cribbs and Farber 2008; Testa and Slack 2002). If these patterns are reproduced for social relations of kidney exchange, this suggests a potential mechanism of LDKT inequality. It could be that the lower ability of African Americans to reciprocate such important gifts, combined with stronger norms of reciprocal exchange, could lead African Americans to decline these gifts at higher rates than whites.

**Analytical Strategy, Data, and Measures**

Studying racial differences in the LDKT opportunity structure presents a number of analytical difficulties, the foremost of which is that the requisite information is not available in a single dataset. However, with some assumptions many of these factors may be explored using existing data. The goal of this study is to measure demographically typical kinship networks and health status patterns, accurately assign probabilities of genetic and immunological compatibility, and then calculate the number of suitable available living donors in candidates’ simulated kinship network. To illustrate, figure 2 presents a hypothetical kinship structure (represented as a modified ore graph) where the black dot represents the ESRD patient, each pie graph represents a member of their kinship network, and the blue slice in each pie graph represents the probability that that member of the network is a suitable living kidney donor for the ESRD patient. Once this kinship structure and its attributes is constructed, simulating the patient’s LDKT opportunity structure is relatively simple, as discussed below. To reach this goal the analysis proceeds in a number of steps, drawing separately on information on
demographic patterns of transplantation-relevant genes, biologically-informed kinship structure, and health statuses which would disqualify one as a living kidney donor.

**The Living Donor Kidney Transplant Opportunity Structure Simulation**

To generate a data-driven simulation of white-black differences in the LDKT opportunity structure, 100 simulations (ten each for each imputation of the UNOS dataset, described below) were conducted to measure simulated opportunity structures while allowing for random noise from the simulation process.

**Information on Transplant Candidates**

First, demographic, genetic, and immunological information on transplant candidates were employed to obtain estimates of the race-specific distribution of ABO and HLA genotypes and to calculate the probability of positive crossmatches between donors. Demographic characteristics (race, age, education, and gender) are conserved for use in probabilistically matching transplant candidates to other needed attributes, as discussed below.

**Dataset: United Network for Organ Sharing STAR Files.** Since 1987, the United Network for Organ Sharing (UNOS) has collected detailed information on every organ transplant recipient, donor, and candidate in the U.S., containing information on the demographic, socioeconomic, medical status, laboratory, and medical treatment characteristics of each such person. Importantly, all ESRD patients are required to enroll in the kidney transplant waitlist, even if they have already identified a living donor. Therefore this database contains information on all legal transplant candidates in the U.S. since 1987.
Although this dataset contains information on the social (and sometimes biological) relationship LDKT recipients had with their donors, information on the full social networks of transplant candidates is lacking. Nonetheless, it is useful in analyzing the distribution of demographic, genetic, and immunological characteristics of persons on the kidney transplant waitlist in the U.S. Whites and blacks only were used in the present analysis due to sampling frame limitations of the kinship data used, as discussed below. ABO and HLA typing and antibody screening is performed at the center at which the patient is evaluated.

Ten different imputations were produced from this file using hotdeck imputation methods based on patient age, ethnicity, gender, and education. In hotdeck imputation, discrete groups are assigned to each observation (here, the demographic attributes just described), then non-missing values for the missing variables are drawn at random from other members of that group, proportionate to their representation in that subpopulation. Hotdeck imputation methods are widely used by government agencies such as the Census. Although multiple imputation and direct maximum likelihood methods are more in vogue in secondary data analysis in sociology, the very large size of the datasets involved and the low rates of missingness of key variables made hotdecking, which is a computationally more efficient imputation method, an attractive option for this study. Ten simulations were conducted on each imputed dataset for a total of 100 simulations.\(^1\)

*Calculating genetic compatibility probabilities.* One may have the same alleles at a locus in the genome with another through one of two mechanisms. First, as a result of

\(^1\) Although additional simulations would be preferable, the very large memory requirements of this study and the computational intensiveness of the simulation limited the number of simulations which were feasible for this study. Additionally, as discussed below the distribution of simulated characteristics was very tight in this study, suggesting that additional simulations would not substantively change the primary results of this investigation.
basic processes of genetic descent one is guaranteed to share at least one gene at each
locus in the genome with each of one’s parents at birth because parents’ genes combine
to constitute one’s own genome. By extension, other genetic relatives who may be
reached through parent-child network ties have a defined baseline probability of
matching one’s genes at each locus in the genome. This form of genetic similarity is
known as identity by descent (IBD) and is easily mathematically specifiable. For instance,
one has a 50% chance of sharing a particular copy of a gene IBD with one’s sibling, a
25% of doing so with one’s half sibling, and so on. However, one may also share genes
with related and unrelated alters through a process directly related to the population
distribution of genes at each locus. For instance, if a gene does not vary at all in a
population, one is guaranteed to match on this gene with all others in that population, and
if 75% of all members of that population have the same allele one has an excellent chance
of matching unrelated strangers on that gene, as well. This is known as identity by state
(IBS). Both forms of genetic matching are important when predicting the availability of
suitable living donors in one’s kinship network.

This stage of the analysis requires the assumption that, conditional on race, the
ABO and HLA distributions of kidney transplant candidates are representative of the
general population, and that all families are racially homogenous. Under this assumption,
the probability that a member of one’s kinship network has a compatible blood type with
the transplant candidate may be calculated as follows (Kanter and Hodge 1990):

\[ P(C_{ijk}) = T_{2ijk} + T_{1ijk}q_k + T_{0ijk}q_k^2 \]  \hspace{1cm} (1)

where \( P(C_{ijk}) \) is the probability of blood type compatibility, \( i \) indexes ego, \( j \) indexes alter,
and \( k \) indexes racial/ethnic group. \( T_{xijk} \) is defined as the probability of sharing \( x \) alleles
IBD at the ABO locus for a dyad with the i-j pair’s genetic relationship degree. Parent-child relations necessarily share exactly 1 allele at a locus due to common inheritance, so for these relations $T_1=1$ and $T_2=T_0=0$. For all other relationship types, the $T$ values may be calculated by taking the average genetic relationship, $r$, for that genetic relationship type\(^2\), and calculating $T_2=r^2$, $T_1=r(1-r)$, and $T_0=(1-r)^2$. Finally, $q_k$ is defined as the percentage of the racial/ethnic group that has a compatible blood type with i’s ABO phenotype, as measured in the ABO distribution among transplant candidates in the UNOS dataset. This component of the formula represents the probability of IBS matching. Blood type compatibility (as used in the $q_k$ values) is defined as follows:

$$
\begin{array}{c|cccc}
\text{A} & \text{A} & \text{B} & \text{AB} & \text{O} \\
\hline
\text{A} & 1 & 0 & 0 & 1 \\
\text{B} & 0 & 1 & 0 & 1 \\
\text{AB} & 1 & 1 & 1 & 1 \\
\text{O} & 0 & 0 & 0 & 1 \\
\end{array}
$$

(2)

where recipient blood type is on the rows, donor blood type is on the columns, and blood type compatibility is defined as the matrix equaling 1 for the i,j cell of the compatibility matrix. Thus O is the universal donor, AB is the universal recipient, and otherwise all blood types are compatible with themselves.

A similar procedure is used to calculate HLA compatibility probabilities, but this calculation is necessarily more complicated because of the greater polymorphism at these loci and the fact that there are three such genes under consideration instead of one. To calculate HLA compatibility probabilities, the proportion of HLA haplotypes which are compatible with a given haplotype on one, two, or three loci was calculated and added to the following formulas:

$$
P(M_{ik} = 0) = T_0 q_{1k}^0 q_{2k}^0
$$

\(^2\) $r=.5$ for full siblings, $r=.25$ for half siblings, grandparents, grandchildren, aunts, uncles, nieces, and nephews, $r=.125$ for first cousins and similarly distant relations, and $r=0$ for alters who are not genetically related.
\[ P(M_{ik} = 1) = T_0(q_{1k}^1 q_{2k}^0 + q_{1k}^0 q_{2k}^1) \]
\[ P(M_{ik} = 2) = T_0(q_{1k}^2 q_{2k}^0 + q_{1k}^0 q_{2k}^2 + q_{1k}^1 q_{2k}^1) \]
\[ P(M_{ik} = 3) = T_1 q_{yk}^0 + T_0(q_{1k}^3 q_{2k}^1 + q_{1k}^1 q_{2k}^3 + q_{1k}^2 q_{2k}^2) \]
\[ P(M_{ik} = 4) = T_1 q_{yk}^1 + T_0(q_{1k}^1 q_{2k}^3 + q_{1k}^3 q_{2k}^1 + q_{1k}^2 q_{2k}^2) \]
\[ P(M_{ik} = 5) = T_1 q_{yk}^2 + T_0(q_{1k}^3 q_{2k}^2 + q_{1k}^2 q_{2k}^3) \]
\[ P(M_{ik} = 6) = T_2 + T_1 q_{yk}^3 + T_0 q_{1k}^3 q_{2k}^3 \]

where \( M_{ik} \) is defined as the HLA match degree (out of 6) for person \( i \) in race \( k \), \( q_{1k}^x \) and \( q_{2k}^x \) are defined as the probability of \( x \) matches with an unrelated member of race \( k \) for haplotypes 1 and 2 respectively, and \( q_{yk}^x \) is the probability of \( x \) matches for a randomly chosen haplotype with an unrelated member of race \( k \). As with the simpler ABO formula above, these formulas are designed to combine the ways in which a given match degree can be attained through two different routes – IBD matching (represented by the \( T_x \) components) and IBS matching (represented by the \( q \) components). For these calculations, HLA compatibility was defined using the current list of HLA serological equivalencies.\(^3\)

**Calculating positive antigen crossmatch probabilities.** PRA is measured as the percentage of a representative set of HLA antigens to which the intended recipient’s blood displays an immunological reaction, indicating antibodies for the antigens in question. However, by definition one cannot be crossmatched with antigens serologically equivalent to one’s own, so the probability of a positive crossmatch is inversely proportionate to one’s HLA match degree with the alter in question. Allowing for this, the probability of positive crossmatch is calculated as:

\[ P(XM) = PRA \left( \frac{6 - M_{lk}}{6} \right) \]  

where XM stands for crossmatch, \( M_{lk} \) represents the simulated number of HLA equivalencies, and \( 6 - M_{lk} \) indicates the number of mismatched HLA antigens with that donor pair. In other words, a transplant candidate’s PRA is adjusted to reflect the probability of crossmatch among the mismatched HLA antigens only.

**Information on Kinship Structures**

In order to predict the LDKT opportunity structure for transplant candidates in the U.S., information is employed on the distribution of genetically-defined kinship ties for demographically similar individuals in the U.S. It is important to define these kinship ties genetically rather than socially because genetic compatibility is a crucial determinant of donor suitability.

*Dataset: Panel Study of Income Dynamics Family Information Mapping System.*

It is equally crucial to define candidates’ kinship structure as broadly as possible. The Panel Study of Income Dynamics (PSID) is one of the premier longitudinal studies of families in the U.S. In 1968 the PSID began following a representative sample of about 4,800 households. Subsequently the PSID re-interviewed the original families frequently (every year through 1997; every other year thereafter) and followed descendant families as households split and were formed. As such the PSID includes a strong genealogical component, as much of this household formation consisted of children growing up, moving out of the house, and forming families of their own. Some lineages now include as many as four generations.

Helpfully, the PSID now provides biologically-informed linkage files, known as the Family Identification Mapping System (FIMS), by which parent/child and sibling ties
are defined among all members of the PSID sample. FIMS differentiates between biological and adoptive ties as well as permitting differentiation between full, half, and step-siblings. As such the PSID is now the premier source of population representative, longitudinal information on multigenerational black and white families in the U.S.\(^4\).

For the present analysis all members of the PSID who were alive in 1999 and had at least one measured biological kin tie were included in the analysis. Persons who died before 1999 were included when defining biological kinship networks but excluded thereafter. Each included person was assigned a biologically-informed ego kinship network as described below.

**Characterizing kinship ties.** Parental ties may be defined as \( P_{NiNj} \), where \( P_{ij} = 1 \) if the individual on column \( j \) is the parent of the individual on row \( i \) and \( =0 \) otherwise. This matrix is non-symmetrical because one is not one’s parents’ parent. Similarly, full sibling ties may be defined as \( FS_{NiNj} \), where \( FS_{ij} = 1 \) if the individual on column \( j \) is the sibling of the individual on row \( i \) and \( =0 \) otherwise. Of course, this matrix is symmetrical. Using these matrices only and adapting the formulas in Batagelj and Mrvar (2006; see Goldstein 1999 for a similar approach), biological kin relations may be calculated in matrix terms as follows (where \( X' \) is defined as the transpose of matrix \( X \)):

- **Child:** \( C = P' \)  \hspace{1cm} (5)
- **Half sibling:** \( HS^* = 1 \) if \( PP^* = 1 \) and \( =0 \) otherwise

\(^4\) While Latinos are included in the sampling design as well, over time with high rates of Latin American migration into the U.S. the Latino sample became increasingly unrepresentative of the U.S. Latino population. While the PSID has since supplemented the original sample with additional Latino families, the later date of this sampling procedure means that information is available on fewer generations of these families, and would not permit a valid comparison of the kinship structure of Latinos with whites and African Americans. As such only white and black families are examined in this study. Additionally, the PSID sample design does not permit the identification of kinship linkages among those not directly descended from the originally sampled households through procreation, adoption, marriage, or co-residence. This is a major limitation of this dataset for present purposes because this means that key members of one’s kinship networks are excluded.
- Grandparents: $GP = P^2$
- Grandchildren: $GC = P'P'$
- Aunt/Uncle: $AU = PFS$
- Niece/Nephew: $NN = AU'$, where child ties are set to 0.
- Cousin: $PPP'P'$, where the resultant diagonal is set to 0.

Non-biological kinship ties are defined as the absence of any of these ties within a lineage.

**Information on Health Statuses**

In addition to genetic match degree and positive HLA antigen crossmatches, another reason a member of an ESRD patient’s kinship network may not be a suitable living kidney donor is due to a health condition which would endanger the kidney donor or recipient should an LDKT take place. These conditions are known as contraindications for kidney donation. Although there is no uniform standard for medical evaluations of LDKTs, in 2007 an OPTN committee made a set of recommendations for ‘absolute’ and ‘relative’ contraindications for living kidney donation based on a survey of nephrologists’ evaluation practices. The list of ‘absolute’ contraindications include: age less than 18 years old, hypertension, diabetes, abnormal glucose tolerance test, history of thrombosis or embolism, major psychiatric conditions, extreme obesity (BMI>35), coronary artery disease, symptomatic valvular disease, chronic lung disease, recent malignancies (or cancers with a long time to recurrence), urologic abnormalities of the kidney, low creatinine clearance rates, peripheral vascular disease, proteinuria, HIV infection, Hepatitis C infection, and Hepatitis B infection. Although some transplant centers surely deviate in various manners from this list, for present purposes insofar as possible this list
of statuses and conditions is treated as the full list of contraindications for living kidney donation.

*Dataset: National Health and Nutrition Examination Surveys (NHANES) 1999-2008.* Collected since 1959, the NHANES studies have long served as the nation’s most detailed population representative survey of the health of the U.S. populace. In addition to household, socioeconomic and demographic information, NHANES collects a full medical history, detailed medical examination by a physician, and an impressive collection of laboratory measures assessing the prevalence of major chronic health conditions in the U.S. population. Since 1999, NHANES has been collected in consecutive two-year cycles, with data available for 1999-2000, 2001-2, 2003-4, 2005-6, and 2007-8.

*Medical contraindications.* Hypertension was defined as having an average blood pressure greater than 130/90 on average over four separately measurements. Diabetes was measured as reporting ever being diagnosed with diabetes. Abnormal glucose tolerance was define as a 2-hour glucose tolerance test score greater than 140. Psychiatric conditions were defined using survey-based measures of panic disorder, major depression, and generalized anxiety disorder. Although survey-based measures are not ideal measurements of psychiatric conditions, the measures used were well-validated measures of DSM-defined criteria. Furthermore, this is not an exhaustive list of potentially disqualifying psychiatric conditions; however, these were the only ones available in the NHANES data.

---

5 While this is not the standard cutoff for hypertension, this is the recommended cutoff for evaluating blood pressure as a contraindication for living kidney donation.
Obesity was assessed as a calculated BMI score greater than 35\textsuperscript{6}. Coronary artery disease was based on respondent reports of previous diagnoses of coronary artery disease. Chronic lung disease was assessed by having ever been diagnosed with asthma, emphysema, or having current bronchitis. Cancer history excludes one from kidney donation if one has ever had breast cancer or had any cancer in the last ten years. Creatinine clearance rates (eCCR) were assessed using the Cockcroft-Gault formula for estimated creatinine clearance rates (Cockcroft and Gault 1976), and poor kidney function was defined as eCCR<80.

Peripheral artery disease was defined as having a right or left ankle-brachial index score (Hirsch et al. 2006) of less than 0.9 (Criqui and Denenberg 1998). Proteinuria was measured as having an albumin-creatinine ratio of $\geq 17$ for men and $\geq 25$ for women (Mattix et al. 2002). HIV diagnoses were based on HIV antibodies in the respondent’s blood (McQuillan et al. 2010). Hepatitis B diagnoses were based on the result of a hepatitis B surface antigen test (Ioannou 2011), and hepatitis C diagnoses were based on the results of a hepatitis C antibody test (Armstrong et al. 2006).

Measures were not available in NHANES 1999-2008 for history of thrombosis or embolism, symptomatic valvular disease, or urologic kidney abnormalities. Furthermore, all measures used in this analysis were not available in all years and were not always available for the full sample or persons of all ages. The following steps were taken to address these data limitations. First, if data were not available for all years of NHANES data, the same demographic patterns of that contraindication were assumed for all years. Second, HIV, Hepatitis B, and Hepatitis C, and all psychiatric measures were not

\textsuperscript{6} Similarly, although BMI of 30 is the standard research cutoff for obesity, a BMI of 35 is the cutoff recommended by the OPTN committee.
available for NHANES respondents over 50. This analysis assumes that the prevalences of these diseases for persons aged 51 and older are the same as for persons aged 36-50. Finally, measures of peripheral artery disease were not available for persons younger than 35. This analysis assumes that this prevalence is 0.

**Combining Information on Transplant Candidates, Kinship Structures, and Population Health Distributions**

For this simulation, information on kinship structure and kinship health statuses was assigned in two steps. First, medical contraindications were assigned to members of measured kinship networks proportionate to the probability of having a medical contraindication among demographically similar members of the health status data set. Second, kinship networks and health statuses were assigned to transplant waitlist members using an original weighted matching algorithm designed by the author.

**Assigning medical contraindications to kinship alters**

For the purposes of this study all variables measuring medical contraindications for living kidney donation in NHANES were combined into a single indicator for medical contraindications and probabilistically assigned to members of the PSID based on the weighted proportion of persons with any contraindication in that person’s race, age, education, and gender categories. Race was defined as being either white or black, by self-report. For matching purposes age was coarsened into the following categories: age 0-20, 21-35, 36-50, 51-65, and 66+. Education was recoded into the following categories: less than a high school education, high school education or equivalent, some college courses but no four-year degree, and a four-year college degree or higher. Gender was measured as being either male or female. When members of the PSID dataset were missing information on any of these variables, contraindications were assigned
proportionate to demographic categories on which the respondent had complete
information only. After probabilities of having a contraindication were assigned to all
members of the PSID, their contraindication status was determined by comparing the
value of a uniform random variable to their assigned probability of having any of the
measured medical contraindications.

Assigning Kinship Networks to Transplant Candidates

Individuals’ kinship structures are strongly related to age, and somewhat less so,
race, education, and gender. In the first case, one cannot be a grandparent if one is 10
years old and is unlikely to have a living parent if one is 90 years old. Similarly, due to
fertility and mortality differences by race and education, kinship structure will be related
to these factors as well. Although based on available data one cannot know the kinship
structures of persons on the kidney transplant waiting list, one can probabilistically
reproduce the distribution of measured kinship ties in PSID interactively by age, race,
education, and gender, recoded as described above.

Kinship network assignment was conducted based on a weighted matching
algorithm designed by the author, which functions as follows (and is illustrated in Figure
3). First, members of the kidney transplant waitlist and the PSID were assigned groups
for all combinations of race, education, gender, and age. This assignment was identical
for both datasets. Second, individual sampling weights in the PSID were transformed as
follows:

\[ w'_{lk} = \frac{w_{lk}}{\sum_{i=1}^{N} w_{ik}} \]  

(6)

In other words, individual weights were transformed into the proportion of total
individual weights represented in group k. Thus the transformed weights all summed to
one within each of the demographic groups observed. Third, the $w'_{ik}$ values were transformed so that each individual was assigned a range of the 0-1 probability space equal to their value of $w'_{ik}$. Fourth, individuals on the kidney transplant waitlist were each assigned a uniform random variable ~U(0,1), which was compared to the values of this transformed weight variable so that kidney transplant candidates were assigned kinship networks for persons with identical demographic characteristics proportionate to their weights using a many-to-one matching algorithm.

To aid the reader in understanding this unfamiliar method, Figure 3 illustrates this process in simplified form. In this figure, ten hypothetical members of the kidney transplant waiting list are shown in the spreadsheet to the left, and 20 members of the PSID (two of which have identical demographic characteristics as each of the waitlisted persons) are depicted to the right. In addition to the demographic characteristics, a weight column and range column are assigned to the observations in the hypothetical PSID spreadsheet. The weight column is $w'_{ik}$, and the range column is the transformed version of this variable described above, constructed so that each PSID sample member is assigned a probability space equal to their value of $w'_{ik}$. Because more than one PSID sample member matches the characteristics of each transplant waiting list member, these range values are used to assign kinship networks for demographically identical persons in the PSID proportionally to such persons’ share of the target population of the PSID. The u column in the waiting list spreadsheet is used to determine which kinship network is actually assigned, and rows which are assigned to waiting list members are highlighted in gray in the spreadsheet on the right, with arrows linking the merged observations. So, for instance, observation 1 in the waiting list spreadsheet in this illustration is assigned the
kinship network of observation 1 in the PSID spreadsheet because their value of u was between 0 and 0.4, the range associated with that member of the PSID, and the observations otherwise match on demographic characteristics. If this person’s u value had been .7 instead, the kinship network of observation 2 would have been assigned to them.

The virtue of this approach is to assign kinship networks to members of the kidney transplant waitlist based on one’s kinship-relevant demographic characteristics, and also assigns kinship network directly proportionally to the sampling weights associated with the PSID observation in question. While imperfect, this procedure assigns observed kinship networks in a manner which preserves the association of demographic characteristics with kinship structure and maintains the population representativeness of the kinship distributions conditional on these demographic characteristics.

Calculating the Opportunity Structure Distribution

The procedures just described were used to assign kinship structures to transplant candidates, and probabilities of HLA and ABO histocompatibility, positive crossmatch, and medical contraindications to kinship network alters. These are the full list of proximate determinants of LDKT opportunity structure. As a final step, the joint distribution of these properties was calculated for each transplant candidate to generate a distribution of suitable living kidney donor ties within each assigned network. Each kin that meets the following conditions was counted as a suitable living kidney donor: a) ABO histocompatibility, b) two or more HLA matches, c) no positive crossmatch, d) no medical contraindication, and e) the kin is 18 years old or above.

Using this calculation, each transplant candidate was assigned the number of kin that meet these transplant suitability conditions, and also a dichotomous variable
measuring whether they had any suitable donors in their kinship network. These were the primary dependent variables of the present analysis. Additionally, the distribution of living kidney donor suitability, and reasons for exclusion if not suitable, were preserved for each kin in the patient’s kinship network.

Calculating Counterfactual Effects

The procedures just described are sufficient, contingent on the assumptions of the simulation, to estimate the LDKT opportunity structure for whites and blacks on the kidney transplant waitlist. However, because these characteristics are jointly simulated, the role of each factor in producing differential LDKT opportunity structures will not be clear. To address this shortcoming, a series of counterfactual microsimulations were produced for each simulation run, in which the distributions of each proximate determinant of the LDKT opportunity structure (ABO and HLA match, PRA, kinship structure, and medical contraindications) are redistributed at random across all kidney transplant candidates and then re-simulated.

Blacks and whites in the different datasets employed here enter the simulation with different distributions of the proximate determinants of the LDKT opportunity structure. By re-assigning these characteristics at random from the original distribution, irrespective of the other characteristics of the observed person, LDKT opportunity structures may be estimated in the absence of the baseline differences in these characteristics. The estimated effect of the distributional differences in the proximate determinant is then calculated as:

\[
\beta_X = 100 \left\{ \frac{[(Y_{1X} - Y_{2X}) - (Y_{1B} - Y_{2B})]}{(Y_{1B} - Y_{2B})} \right\}
\]  

(7)
where $\beta_X$ is the estimated percentage of the racial gap in LDKT opportunity structures explained, $Y_{1B}$ and $Y_{2B}$ are the median simulated values of the dependent variable in the non-counterfactual (baseline) simulation for races 1 and 2 respectively (where the group with the higher median value of the dependent variable is substituted into $Y_{1B}$), and $Y_{1X}$ and $Y_{2X}$ are the same median simulated values of the dependent variable when counterfactual simulations for $X$ are conducted.

The resultant value from this calculation may be interpreted as the percentage of the baseline simulation difference in the dependent variable explained by equalizing variable $X$. If $\beta_X = 50$, for instance, this means that the racial gap in the dependent variable is 50% smaller in the counterfactual condition than in the baseline simulation, suggesting that the group with the lower baseline median value of $Y$ is disadvantaged by characteristic $X$. Additionally, $\beta_X$ may take on negative values or values greater than 100. In the former case, this is interpreted to mean that equalizing this factor increases the simulated difference in the dependent variable by race, suggesting that the group with the lower median value of $Y$ derived some advantage from racial differences in $X$. In the latter case, $\beta_X > 100$ suggests that, not only is characteristics $X$ a source of disadvantage for race 2, but that equalizing it would result in the disadvantaged group having an overall advantage in $Y$.

**Results**

**Kidney Transplant Waitlist: Descriptive Statistics**

Table 1 presents descriptive statistics on the demographic composition of the U.S. population based on American Community Survey estimates 2001-2009 (Ruggles et al. 2010), the same figures on the composition of the kidney transplant waitlist from July 1,
2000 through February 26, 2010, and the ratio of their representations. All figures are subsetted to include only white and black persons. The ratio column crudely measures the degree to which members of that demographic group are over- or under-represented on the kidney transplant waitlist during this time relative to their share of the population. Finally, the distribution of PRA for each group is presented.

The results of the ACS-UNOS demographic comparisons reveal the degree to which members of the American populace are overrepresented on the kidney transplant waitlist. Young persons are much less common than older persons to be on the kidney transplant waitlist, and persons aged 36-65 are much more likely to be on the waitlist. Educational patterns are also revealing – although those with less than a high school education are less likely than others to be on the waitlist, this is likely due to the association of this educational attainment with younger ages. For all other educational categories, higher education is associated with lower rates of transplantation waitlisted. Additionally, males are much more likely than females to be on the transplant waitlist. Finally, African Americans are greatly overrepresented on the transplant waitlist – approximately 2.57 times more likely to be on the waitlist than their representation in the population.

Table 1 also reveals appreciable demographic patterns of PRA among those on the kidney transplant waitlist. While the average PRA score is .175, patients aged 51-65, less educated persons, women, and African Americans have substantially higher PRA scores on average than their age, education, gender, and racial counterparts. The remainder of this section is organized around a series of questions the present analyses are designed to answer.
Could Patterns of Medical Contraindications Explain Racial Differences in LDKT?

Table 2 presents the demographic distribution of contraindications for living kidney donation, as estimated using NHANES 1999-2008 data. The ‘All’ column describes the joint distribution of contraindications and the remaining columns describe their individual distribution. Age exclusions are represented in the ‘All’ column only. According to these estimates, 77.5% of whites and 81.6% of blacks are excluded from living kidney donation for medical or demographic reasons. These results suggest health condition disadvantages for African Americans when pursuing an LDKT.

However, the results also show substantial variability in the racial patterns of medical exclusions. African Americans have higher prevalences of hypertension, diabetes, obesity, albuminuria, hepatitis B, hepatitis C, and HIV than whites. However, whites are subject to higher prevalences of abnormal glucose tolerance, psychiatric disorders, coronary artery disease, chronic lung disease, cancer, low creatinine clearance rate, and peripheral artery disease exclusions. On balance the joint distribution of these characteristics produce a moderate disadvantage for African Americans when pursuing an LDKT.

Could Race Differences in Kinship Structure Explain Racial Differences in LDKT?

Table 3 presents the distribution of biologically-informed kinship ties, as assigned to members of the kidney transplant waitlist using the weighted matching algorithm described above. These results suggest that African Americans on the kidney transplant waiting list are likely to have larger kinship networks on average as well as greater variability in their distribution of kinship ties. Furthermore, this difference holds for every measured kinship type. Together, these results indicate that the LDKT opportunity
structures of African Americans are advantaged by the overall size of their networks and the number of close genetic relatives therein.

**Could Race Differences in Histocompatibility Probabilities Explain Racial Differences in LDKT?**

Table 4 presents the distribution of ABO and HLA histocompatibility by race and genetic relationship with alters of the same race, calculated as described above. For members of both races, the probability of ABO histocompatibility is higher for close genetic relatives than for more distant genetic relatives and unrelated alters. However, the probability of ABO histocompatibility is moderately high (>40%) for even unrelated alters of both races. This analysis also shows evidence that the probability of ABO histocompatibility is slightly higher for whites than for blacks for all genetic relationship types, and this race difference grows with decreasing genetic relationships with the alters in question. So, for instance, the probability that a white person is ABO compatible with their full sibling is only 2% higher for whites than for blacks, but this difference is 4.2% for unrelated alters.

Similar patterns are observed for probabilities of HLA histocompatibility degree. For members of both races, parents and full siblings offer the best chance for a strong HLA match degree. Parents are guaranteed to share three or more HLA alleles with their children, but the marginal probability of additional matches beyond three decreases rapidly. In contrast, full siblings have a moderately high chance (approximately 25%) of being a full HLA match with one another. On the other end of the genetic relationship spectrum, unrelated alters have greater than a 50% of having no HLA matches with the transplant candidate, reflecting the high degree of polymorphism in the HLA-A, -B, and –DR loci.
These calculations also reveal that whites are more likely to have a high degree of HLA match with their kinship alters conditional on genetic relationships, and that this difference grows with declining genetic relationships with the alters. Altogether, these results demonstrate that whites have a higher probability of genetic histocompatibility with given members of their kinship network conditional on genetic relationship with that alter.

**How do Kinship Structure, Health Patterns, and Histocompatibility Probabilities Jointly Shape the LDKT Opportunity Structure by Race?**

Table 5 presents the simulated distribution of suitable living donors, as defined above. The results suggest that whites are actually somewhat less likely to have at least one suitable living donor in their kinship networks than are blacks. 58.2% of white ESRD patients are simulated to have a suitable living kidney donor in their kinship network, whereas this is true of 62.5% of black ESRD patients. Furthermore, among those simulated to have a suitable donor in their network, blacks on average are simulated to have more such donors than are whites.

The results also illustrate racial differences in this difference by genetic relationship type. For instance, whites are slightly more likely to have a suitable sibling or child living kidney donor whereas blacks are more likely to have at least one suitable such donor in all other genetic relationship categories. Children are the relationship category in which patients are most likely to have a suitable donor, followed, surprisingly, by non-biological kin. This latter effect is a result of the fact that most kinship networks have a very large number of kin with no defined biological tie to the reference person.
Taken together, these results suggest that African American patients on the kidney transplantation waitlist are more likely to have a suitable living kidney donor in their kinship network, and more likely to have more than one such suitable donor, than are white persons.

**What is the Probability that Each Member of One’s Kinship Network Will Be a Suitable Living Kidney Donor?**

While Table 5 presented the distribution of suitable living donors from the waitlisted patient’s perspective, Table 6 describes the probability that each individual member of one’s kinship network will be a suitable living donor, stratified by race and genetic relationship type. Table 6 also provides the probabilities of living donor exclusions for HLA histocompatibility, ABO histocompatibility, medical or age contraindication, or positive crossmatch reasons. (These outcomes do not add up to 100% because a donor can be excluded for more than one reason.)

These results suggest that a random member of a white person’s kinship network is more likely to be a suitable living donor than a random member of a black person’s kinship network. On average, 6.4% of white kinship alters are simulated to be a suitable living donor, while this is true for only 5.4% of black kinship alters. Black kinship alters are more likely than white kinship alters to be excluded for HLA, ABO, medical contraindication, and positive crossmatch reasons.

These results also suggest considerable variability in the probability that a given kinship alter will be a suitable living donor by genetic relationship. Whites’ full siblings, parents, children, grandchildren, aunts, uncles, first cousins, and unrelated kin are more likely than comparable black alters to be a suitable living donor, whereas black half siblings are more likely than whites’ half siblings to be an appropriate living donor.
Finally, the probability of living donor suitability varies proportionately with the genetic relationship degree – full siblings, children, and parents are the most likely suitable living donors, whereas non-biological kin have a very low probability of being a suitable living donor.

**What are the Contributions of Genetics, Kinship Structure, Health, and PRA to Racial Differences in LDKT Opportunity Structures?**

To answer this question, counterfactual microsimulations were estimated in which each of four factors – genetic distributions, kinship structures, health statuses, and PRA – were re-assigned to the appropriate individuals at random while preserving their overall distributions. The results of this exercise confirm the findings of the previous analyses (Table 7). Genetics are a source of LDKT opportunity structure for whites – equalizing the probability of genetic match degree results in a 14.5% increase in black opportunity structure advantage in the proportion of patients with suitable living donors in their kinship network, as well as a 40.6% increase in their advantage in the average number of suitable living donors. Health distributions are a very small source of white advantage, and equalizing this factor adds only .8% and 1.5% to the black advantage in the proportion with a suitable donor and the number of donors respectively. PRA differences by race are also a source of white advantage in the LDKT opportunity structure. Equalizing this factor results in an 18.7% increase in the black advantage in the proportion with suitable donors and a 20.4% increase in the average number of donors. Finally, all factors were equalized simultaneously to confirm that this equalizes the LDKT opportunity structure. It does – whites and blacks have equivalent LDKT opportunity structures when all four factors are equalized.
In summary, whites transplant candidates are on average expected to be advantaged in the LDKT opportunity structures by their higher probability of genetic histocompatibility, favorable health status, and lower probability of antigen crossmatch. In contrast, the African American advantage in the LDKT opportunity structure stems from their larger average kinship structures. Equalizing this factor gives whites on average a higher proportion of kinship structures including a suitable living donor and a higher average number of such donors than blacks.

**What Proportion of Suitable Living Donors Contribute Kidneys for Transplantation?**

As a crude analysis of the answer to this question, the proportion of white and black transplant candidates who actually obtained an LDKT transplant is compared to the proportion estimated to have a suitable donor in their kinship structure in Table 8. The results suggest that, conditional on having a suitable donor in one’s kinship structure, whites are more likely to obtain an LDKT than are blacks. Although a higher proportion of black patients are estimated to have a suitable donor, they are less than half as likely to actually obtain an LDKT as are whites. Furthermore, the proportion of members of both races actually obtaining an LDKT is substantially lower than the proportion estimated to have a suitable donor in their network.

**Discussion**

This paper investigates racial differences in the opportunity for LDKT in the kinship structures of whites and blacks on the kidney transplant waitlist. By matching data on the distribution of kinship ties, medical contraindications for living kidney donation, the probability of HLA and ABO histocompatibility degrees, and the probability of positive crossmatches between candidates and kin, this research examines
how kinship structures, health conditions, and genetic and immunological factors shape the opportunity for LDKT. If whites and blacks substantially differ in their LDKT opportunity structures, this could partially explain racial differences in LDKT rates.

The results show that this is not the case. If anything, blacks are likely to have suitable living kidney donors in their kinship structures at higher rates than whites, and have more such kin in their network conditional on having any. However, each individual white kin has a higher probability of being a suitable living donor than comparable black kin.

In light of research on the living donor search behaviors of kidney transplant candidates and their kin, however, these results suggest a mechanism by which LDKT opportunity structures could produce racial disparities in LDKT. According to one study, only about half of transplant candidates bring in any potential donors for evaluation, and of those who do so, the large majority bring in two or fewer potential donors (Weng et al. 2010). Furthermore, black potential donors are less likely to complete the evaluation process and are more likely to be excluded for medical, genetic, or immunological reasons, conforming to the patterns observed here. Therefore, because transplant candidates do not have their full social networks evaluated for donation, it may be that the probability that a given alter is a suitable donor is the more important determinant of LDKT outcomes than the number of suitable donors in one’s total network.

Additional factors which are hypothesized to moderate the relationship between LDKT opportunity structures and LDKTs may shed additional light on the causes of racial inequalities in LDKT. First, racial differences in kin relations could structure the probability that kin are evaluated for donation and proceed with donation conditional on a
positive evaluation. Second, racial differences in beliefs concerning the appropriateness and benefits of LDKT could influence these differences. However, racial differences in the rates at which potential donors are evaluated for donation are minor (Weng et al. 2010) and are unlikely to play a major role in the explanation of these differences.

Racial differences in interactions with the health care system may also play a substantial role in the mediation of the effect of the LDKT opportunity structure on LDKT rates. If medical providers differentially promote and support living donor evaluation among whites and blacks, this could potentially explain racial differences in the rates at which donors are brought in for evaluation, complete evaluations, and donate kidneys conditional on positive evaluations. Racial differences in knowledge of transplantation could play a similar role in the mediation of this difference.

Many of the factors which can preclude kidney donation are subject to potential interventions. Progress in techniques to ameliorate the effects of positive crossmatching, HLA mismatching, and the ABO barrier may affect racial differences in the LDKT opportunity structure in the future. Although none of these techniques offer candidate and graft survival rates equivalent to those for more ideal kidney donor-candidate combinations, should progress continue to be made on these fronts it may be that these may undermine racial differences in the LDKT opportunity structure.

Furthermore, many health conditions which preclude living kidney donation are linked to lifestyle and environmental differences which are potentially modifiable. For instance, diabetes, abnormal glucose tolerance, and obesity prevalences have been growing in the general population, particularly for African Americans, and are linked to
quality of diet and exercise. Should these trends be reversed in an equitable fashion this could raise rates of LDKT and ameliorate racial inequalities therein.

The results of this analysis also suggest that white and black patients are underutilizing their LDKT opportunity structures, although blacks do so to a greater degree. In principle, these results suggest that rates of LDKT could be increased by a factor of three for whites and seven for blacks. While an increase on this scale is unlikely, this suggests that there is room for higher LDKT rates among whites and blacks, and that if whites and blacks searched throughout the entirety of their kinship networks, racial inequalities in LDKT could be eliminated or reversed.

Finally, efforts to increase the rate at which white and black transplant candidates search throughout their full kinship networks could serve to improve transplantation prospects for those without suitable living donors in their kinship networks. Because there is a shortage of kidneys compared to the kidney transplant waitlist, each additional LDKT implies a marginally improved transplant prospect for all others on the waitlist. Of course, any efforts to increase the rate at which LDKT opportunity structures are converted into transplants should be conducted in a non-coercive manner.

This research is subject to a number of limitations, the foremost of which is that direct data on the kinship networks of kidney transplant candidates is unavailable except for those alters who actually donate kidneys. Although this study is conducted using high quality data on kidney transplant candidates and on the distribution of biologically-informed kinship ties by race and patterns of relevant health conditions, the simulations discussed here require a number of strong assumptions. The most important of these assumptions are that: a) conditional on race, kidney transplant candidates ABO and HLA
genes are representative of the population; b) conditional on race, age, education, and gender, members of the kidney transplant waitlist are subject to similar distributions of kinship ties to that of the general population; and c) conditional on race, age, education, and gender, the kin of members of the kidney transplant waitlist are subject to identical probabilities of medical contraindications to that of the general population. The degree to which these assumptions are consequentially violated should be a subject of future research.

**Conclusion**

This paper reports on a simulation analysis of the distribution of suitable living kidney donors in the kinship networks of white and black kidney transplant candidates. The goal was to investigate the possibility that racial differences in the availability of kin who would be suitable kidney donors could explain racial differences in LDKT rates. To the contrary, however, the results of the analysis suggest that blacks and whites have approximately the same probability of having a suitable living donor in their kinship network, although the probability that an individual member of the kinship network is a suitable living donor is somewhat higher for whites than for blacks. While white kidney transplant candidates are advantaged by their higher probabilities of genetic similarity with their kin, their lower probabilities of positive crossmatching therewith, and slightly advantaged in the health characteristics of their kin, the larger typical size of black kinship networks ameliorates this advantage.

---

7 Although it would be preferable to attempt to surmise the effects of likely violations of these assumptions, the complex nature of the problem precludes typical reasoning about this problem. For instance, the HLA genes are qualitative variables with numerous categories. Thinking in terms of directional bias in anticipating the biasing effects of the assumption that transplant candidates’ genotypes are representative conditional on race is unhelpful in such a case. However, the conclusions of this paper are bolstered by the conformity of its findings to that implied by much of the medical literature on racial disparities in transplantation. The primary advantage of the present study is that the effects of these factors may be quantitatively estimated rather than merely hypothesized.
Demographers and sociologists have much to contribute to the understanding of racial inequalities in kidney transplantation. Racial differences in kinship structures, kin relations, interactions with medical care providers, beliefs and knowledge concerning transplantation, and genetic and immunological factors may go far in explaining racial disparities in deceased and living donor kidney transplantation. Social scientists know much about these topics. Although the present results do not explain racial differences in LDKT directly, they do suggest a major role for well-studied social processes in the production of these disparities. By engaging with medical researchers in the analysis of this important and growing problem, social science can do much to improve understanding of racial disparities in kidney disease and transplantation.
References


### Table 1: US Population and Kidney Transplant Waitlist Characteristics, 2000-2010

<table>
<thead>
<tr>
<th>Variable</th>
<th>Category</th>
<th>Percentages</th>
<th>PRA</th>
<th>Mean</th>
<th>SD</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>ACS</td>
<td>UNOS</td>
<td>Ratio</td>
<td></td>
</tr>
<tr>
<td>Overall</td>
<td>--</td>
<td>--</td>
<td>--</td>
<td>--</td>
<td>0.175</td>
</tr>
<tr>
<td>Age</td>
<td>0-20</td>
<td>28.09</td>
<td>7.92</td>
<td>0.28</td>
<td>0.129</td>
</tr>
<tr>
<td></td>
<td>21-35</td>
<td>19.64</td>
<td>12.72</td>
<td>0.65</td>
<td>0.175</td>
</tr>
<tr>
<td></td>
<td>36-50</td>
<td>22.69</td>
<td>32.25</td>
<td>1.42</td>
<td>0.167</td>
</tr>
<tr>
<td></td>
<td>51-65</td>
<td>17.20</td>
<td>34.97</td>
<td>2.03</td>
<td>0.198</td>
</tr>
<tr>
<td></td>
<td>66+</td>
<td>12.38</td>
<td>12.13</td>
<td>0.98</td>
<td>0.161</td>
</tr>
<tr>
<td>Education</td>
<td>&lt;HS</td>
<td>33.62</td>
<td>22.13</td>
<td>0.66</td>
<td>0.127</td>
</tr>
<tr>
<td></td>
<td>HS</td>
<td>29.30</td>
<td>37.25</td>
<td>1.27</td>
<td>0.232</td>
</tr>
<tr>
<td></td>
<td>SC</td>
<td>18.33</td>
<td>21.47</td>
<td>1.17</td>
<td>0.168</td>
</tr>
<tr>
<td></td>
<td>BA+</td>
<td>18.75</td>
<td>19.15</td>
<td>1.02</td>
<td>0.129</td>
</tr>
<tr>
<td>Gender</td>
<td>Male</td>
<td>48.95</td>
<td>59.15</td>
<td>1.21</td>
<td>0.127</td>
</tr>
<tr>
<td></td>
<td>Female</td>
<td>51.05</td>
<td>40.85</td>
<td>0.80</td>
<td>0.244</td>
</tr>
<tr>
<td>Race</td>
<td>White</td>
<td>86.00</td>
<td>64.07</td>
<td>0.75</td>
<td>0.152</td>
</tr>
<tr>
<td></td>
<td>Black</td>
<td>14.00</td>
<td>35.93</td>
<td>2.57</td>
<td>0.218</td>
</tr>
</tbody>
</table>

NOTE: PRA means ‘Panel Reactive Antibody,’ a measure of immunological presensitization scaled 0-1, representing the proportion of the US populace for whose HLA antigens one’s immune system has already generated antibodies. ACS figures are weighted percentages in these categories in the 2001-2009 American Community Survey IPUMS 1% samples among blacks and whites only. UNOS figures are percentages in these categories on the UNOS waitlist 7/1/2000 through 2/26/2010 among blacks and whites only. Ratio is the ratio of the UNOS percentage divided by the ACS percentage and is interpreted as a measure of the degree to which this category is over- or under-represented on the kidney transplantation waitlist relative to its share of the population.
Table 2: Percentage Distribution of Contraindications by Demographic Categories and Contraindication Type

<table>
<thead>
<tr>
<th>Variable</th>
<th>Category</th>
<th>All</th>
<th>HYP</th>
<th>DIAB</th>
<th>GLTT</th>
<th>PSYC</th>
<th>OBES</th>
<th>COAR</th>
<th>CHLD</th>
<th>CANC</th>
<th>CRCL</th>
<th>PARD</th>
<th>ALBM</th>
<th>HEP</th>
</tr>
</thead>
<tbody>
<tr>
<td>Race</td>
<td>White</td>
<td>77.50</td>
<td>2.17</td>
<td>6.61</td>
<td>20.46</td>
<td>13.63</td>
<td>7.55</td>
<td>4.00</td>
<td>9.50</td>
<td>6.50</td>
<td>19.65</td>
<td>3.99</td>
<td>0.18</td>
<td>1.99</td>
</tr>
<tr>
<td></td>
<td>Black</td>
<td>81.58</td>
<td>3.23</td>
<td>7.18</td>
<td>10.09</td>
<td>8.97</td>
<td>10.37</td>
<td>1.10</td>
<td>7.88</td>
<td>1.97</td>
<td>10.04</td>
<td>2.90</td>
<td>0.39</td>
<td>2.98</td>
</tr>
<tr>
<td>Gender</td>
<td>Male</td>
<td>78.12</td>
<td>3.17</td>
<td>6.91</td>
<td>16.45</td>
<td>10.46</td>
<td>6.41</td>
<td>3.92</td>
<td>7.50</td>
<td>4.94</td>
<td>13.83</td>
<td>9.50</td>
<td>6.50</td>
<td>4.00</td>
</tr>
<tr>
<td></td>
<td>Female</td>
<td>79.99</td>
<td>1.99</td>
<td>6.75</td>
<td>16.54</td>
<td>13.20</td>
<td>10.80</td>
<td>1.87</td>
<td>10.24</td>
<td>4.59</td>
<td>18.06</td>
<td>3.75</td>
<td>0.20</td>
<td>1.76</td>
</tr>
<tr>
<td>Education</td>
<td>&lt;HS</td>
<td>77.97</td>
<td>5.16</td>
<td>18.59</td>
<td>31.06</td>
<td>15.45</td>
<td>14.80</td>
<td>7.52</td>
<td>13.62</td>
<td>9.61</td>
<td>38.07</td>
<td>9.51</td>
<td>0.89</td>
<td>5.79</td>
</tr>
<tr>
<td></td>
<td>HS</td>
<td>70.21</td>
<td>4.31</td>
<td>12.18</td>
<td>27.64</td>
<td>15.64</td>
<td>13.67</td>
<td>5.14</td>
<td>9.47</td>
<td>8.72</td>
<td>28.98</td>
<td>6.73</td>
<td>0.37</td>
<td>4.33</td>
</tr>
<tr>
<td></td>
<td>SC</td>
<td>65.53</td>
<td>4.71</td>
<td>9.45</td>
<td>23.46</td>
<td>14.14</td>
<td>15.08</td>
<td>4.40</td>
<td>10.56</td>
<td>7.39</td>
<td>22.14</td>
<td>5.05</td>
<td>0.27</td>
<td>3.60</td>
</tr>
<tr>
<td></td>
<td>BA+</td>
<td>60.54</td>
<td>3.85</td>
<td>7.23</td>
<td>22.10</td>
<td>14.10</td>
<td>9.14</td>
<td>3.68</td>
<td>7.92</td>
<td>8.59</td>
<td>22.67</td>
<td>4.35</td>
<td>0.27</td>
<td>2.66</td>
</tr>
<tr>
<td>Age</td>
<td>0-20</td>
<td>91.24</td>
<td>0.10</td>
<td>0.54</td>
<td>4.35</td>
<td>8.05</td>
<td>2.83</td>
<td>0.00</td>
<td>6.99</td>
<td>0.02</td>
<td>1.00</td>
<td>0.00</td>
<td>0.03</td>
<td>0.17</td>
</tr>
<tr>
<td></td>
<td>21-35</td>
<td>37.28</td>
<td>1.88</td>
<td>2.00</td>
<td>7.94</td>
<td>8.78</td>
<td>12.93</td>
<td>0.11</td>
<td>8.24</td>
<td>1.25</td>
<td>2.47</td>
<td>0.00</td>
<td>0.05</td>
<td>1.47</td>
</tr>
<tr>
<td></td>
<td>36-50</td>
<td>60.83</td>
<td>6.77</td>
<td>6.88</td>
<td>16.76</td>
<td>16.44</td>
<td>15.77</td>
<td>1.01</td>
<td>9.62</td>
<td>3.41</td>
<td>7.32</td>
<td>2.95</td>
<td>0.35</td>
<td>5.94</td>
</tr>
<tr>
<td></td>
<td>51-65</td>
<td>77.95</td>
<td>6.58</td>
<td>17.52</td>
<td>26.61</td>
<td>16.08</td>
<td>17.39</td>
<td>6.13</td>
<td>11.80</td>
<td>8.20</td>
<td>21.65</td>
<td>5.95</td>
<td>0.63</td>
<td>5.01</td>
</tr>
<tr>
<td></td>
<td>66+</td>
<td>95.94</td>
<td>3.55</td>
<td>20.22</td>
<td>49.44</td>
<td>17.96</td>
<td>8.71</td>
<td>12.30</td>
<td>11.87</td>
<td>19.39</td>
<td>71.54</td>
<td>15.18</td>
<td>0.74</td>
<td>4.29</td>
</tr>
</tbody>
</table>

NOTE: Figures in cells are shown as percentages. The following abbreviations for kidney donation contraindications are used: HYP: hypertension; DIAB: diabetes; GLTT: glucose tolerance test; PSYC: psychiatric disorders; OBES: obesity; COAR: coronary artery disease; CHLD: chronic lung disease; CANC: cancer; CRCL: creatinine clearance; PARD: peripheral artery disease; ALBM: albuminuria; HEP: Hepatitis B, C, or HIV. See Appendix ___ for the measures and cutoffs used to define each contraindication in this study. The following abbreviations are used for educational categories: <HS: less than high school; HS: high school or GED; SC: some college but no four year degree; BA+: four year college degree or more.
Table 3: Measured Biological Kinship Tie Distribution, by Race

<table>
<thead>
<tr>
<th>Kinship Type</th>
<th>WHITES</th>
<th>BLACKS</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>Standard Deviation</td>
</tr>
<tr>
<td></td>
<td>Median (90% Interval)</td>
<td>Median (90% Interval)</td>
</tr>
<tr>
<td>Full Siblings</td>
<td>0.63 (0.61,0.65)</td>
<td>1.32 (1.30,1.34)</td>
</tr>
<tr>
<td>Half Siblings</td>
<td>0.22 (0.21,0.22)</td>
<td>0.80 (0.78,0.81)</td>
</tr>
<tr>
<td>Parents</td>
<td>0.66 (0.65,0.70)</td>
<td>0.89 (0.88,0.90)</td>
</tr>
<tr>
<td>Children</td>
<td>1.91 (1.84,1.93)</td>
<td>1.43 (1.42,1.45)</td>
</tr>
<tr>
<td>Grandparents</td>
<td>0.14 (0.13,0.20)</td>
<td>0.47 (0.46,0.57)</td>
</tr>
<tr>
<td>Grandchildren</td>
<td>1.59 (1.51,1.66)</td>
<td>2.75 (2.69,2.81)</td>
</tr>
<tr>
<td>Aunts/Uncles</td>
<td>0.12 (0.11,0.19)</td>
<td>0.65 (0.63,0.82)</td>
</tr>
<tr>
<td>Nieces/Nephews</td>
<td>0.95 (0.89,0.97)</td>
<td>2.59 (2.52,2.63)</td>
</tr>
<tr>
<td>1st Cousins</td>
<td>0.28 (0.27,0.45)</td>
<td>1.53 (1.48,1.89)</td>
</tr>
<tr>
<td>Non-Biological Kin</td>
<td>13.58 (13.46,13.69)</td>
<td>13.83 (13.72,13.97)</td>
</tr>
</tbody>
</table>

NOTE: Standard deviation columns indicate the average within-group standard deviation in the number of indicated kinship ties. 90% Interval columns indicate the 5th and 95th percentiles of these values across simulations.
Table 4: Percentage Distribution of ABO Compatibility and HLA Match Degree by Race and Genetic Relationship

<table>
<thead>
<tr>
<th>Genetic Relationship</th>
<th>Race</th>
<th>ABO Compatible</th>
<th>HLA Matches (Out of 6)</th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>(0)</td>
<td>(1)</td>
<td>(2)</td>
<td>(3)</td>
<td>(4)</td>
</tr>
<tr>
<td>Parent-Child</td>
<td>White</td>
<td>64.9</td>
<td>0.0</td>
<td>0.0</td>
<td>0.0</td>
<td>71.6</td>
<td>24.3</td>
</tr>
<tr>
<td></td>
<td>Black</td>
<td>63.0</td>
<td>0.0</td>
<td>0.0</td>
<td>0.0</td>
<td>75.9</td>
<td>21.7</td>
</tr>
<tr>
<td>r=.500</td>
<td>White</td>
<td>68.8</td>
<td>12.8</td>
<td>8.8</td>
<td>2.8</td>
<td>36.4</td>
<td>12.2</td>
</tr>
<tr>
<td></td>
<td>Black</td>
<td>66.8</td>
<td>14.4</td>
<td>8.2</td>
<td>2.0</td>
<td>38.3</td>
<td>10.9</td>
</tr>
<tr>
<td>r=.250</td>
<td>White</td>
<td>55.4</td>
<td>25.5</td>
<td>17.5</td>
<td>5.6</td>
<td>36.9</td>
<td>12.3</td>
</tr>
<tr>
<td></td>
<td>Black</td>
<td>52.4</td>
<td>28.8</td>
<td>16.5</td>
<td>4.1</td>
<td>38.5</td>
<td>10.9</td>
</tr>
<tr>
<td>r=.125</td>
<td>White</td>
<td>50.7</td>
<td>38.3</td>
<td>26.3</td>
<td>8.5</td>
<td>19.6</td>
<td>6.3</td>
</tr>
<tr>
<td></td>
<td>Black</td>
<td>47.1</td>
<td>43.3</td>
<td>24.7</td>
<td>6.1</td>
<td>19.8</td>
<td>5.5</td>
</tr>
<tr>
<td>r=0</td>
<td>White</td>
<td>46.0</td>
<td>51.1</td>
<td>35.0</td>
<td>11.3</td>
<td>2.3</td>
<td>0.3</td>
</tr>
<tr>
<td></td>
<td>Black</td>
<td>41.8</td>
<td>57.7</td>
<td>33.0</td>
<td>8.1</td>
<td>1.1</td>
<td>0.1</td>
</tr>
</tbody>
</table>

NOTE: HLA match percentages are gray scaled such that darker cells indicate higher match probabilities. ABO and HLA matches are defined as having alleles which are either identical or serologically equivalent to one’s alleles. Probabilities were calculated using genetic probability theory and the ABO and HLA-A, -B, and –DR distributions on the kidney transplantation waitlist – see text for details. No parents or children were excluded for HLA reasons because all parents and children share at least three HLA genes in common.
<table>
<thead>
<tr>
<th>Genetic Relationship</th>
<th>Race</th>
<th>(0)</th>
<th>(1)</th>
<th>(2)</th>
<th>(3)</th>
<th>(4)</th>
<th>(5+)</th>
</tr>
</thead>
<tbody>
<tr>
<td>All</td>
<td>White</td>
<td>41.8</td>
<td>30.4</td>
<td>15.4</td>
<td>6.8</td>
<td>3.0</td>
<td>2.7</td>
</tr>
<tr>
<td></td>
<td>Black</td>
<td>37.5</td>
<td>30.4</td>
<td>16.8</td>
<td>8.0</td>
<td>3.7</td>
<td>3.7</td>
</tr>
<tr>
<td>Full Siblings</td>
<td>White</td>
<td>89.6</td>
<td>8.0</td>
<td>1.9</td>
<td>0.4</td>
<td>0.1</td>
<td>0.0</td>
</tr>
<tr>
<td></td>
<td>Black</td>
<td>90.3</td>
<td>6.8</td>
<td>2.0</td>
<td>0.6</td>
<td>0.2</td>
<td>0.1</td>
</tr>
<tr>
<td>Half Siblings</td>
<td>White</td>
<td>98.3</td>
<td>1.5</td>
<td>0.2</td>
<td>0.0</td>
<td>0.0</td>
<td>0.0</td>
</tr>
<tr>
<td></td>
<td>Black</td>
<td>94.4</td>
<td>4.6</td>
<td>0.8</td>
<td>0.1</td>
<td>0.0</td>
<td>0.0</td>
</tr>
<tr>
<td>Parents</td>
<td>White</td>
<td>91.7</td>
<td>7.6</td>
<td>0.7</td>
<td>0.0</td>
<td>0.0</td>
<td>0.0</td>
</tr>
<tr>
<td></td>
<td>Black</td>
<td>91.6</td>
<td>7.9</td>
<td>0.5</td>
<td>0.0</td>
<td>0.0</td>
<td>0.0</td>
</tr>
<tr>
<td>Children</td>
<td>White</td>
<td>67.8</td>
<td>23.2</td>
<td>7.3</td>
<td>1.4</td>
<td>0.2</td>
<td>0.0</td>
</tr>
<tr>
<td></td>
<td>Black</td>
<td>68.0</td>
<td>23.0</td>
<td>6.9</td>
<td>1.6</td>
<td>0.3</td>
<td>0.2</td>
</tr>
<tr>
<td>Grandparents</td>
<td>White</td>
<td>99.1</td>
<td>0.8</td>
<td>0.0</td>
<td>0.0</td>
<td>0.0</td>
<td>0.0</td>
</tr>
<tr>
<td></td>
<td>Black</td>
<td>98.9</td>
<td>1.1</td>
<td>0.0</td>
<td>0.0</td>
<td>0.0</td>
<td>0.0</td>
</tr>
<tr>
<td>Grandchildren</td>
<td>White</td>
<td>93.3</td>
<td>5.6</td>
<td>0.9</td>
<td>0.2</td>
<td>0.0</td>
<td>0.0</td>
</tr>
<tr>
<td></td>
<td>Black</td>
<td>94.4</td>
<td>7.2</td>
<td>1.1</td>
<td>0.2</td>
<td>0.1</td>
<td>0.0</td>
</tr>
<tr>
<td>Aunts/ Uncles</td>
<td>White</td>
<td>98.7</td>
<td>1.1</td>
<td>0.2</td>
<td>0.0</td>
<td>0.0</td>
<td>0.0</td>
</tr>
<tr>
<td></td>
<td>Black</td>
<td>98.7</td>
<td>1.0</td>
<td>0.2</td>
<td>0.1</td>
<td>0.0</td>
<td>0.0</td>
</tr>
<tr>
<td>Nieces/ Nephews</td>
<td>White</td>
<td>94.8</td>
<td>3.8</td>
<td>1.0</td>
<td>0.3</td>
<td>0.1</td>
<td>0.1</td>
</tr>
<tr>
<td></td>
<td>Black</td>
<td>94.1</td>
<td>4.1</td>
<td>1.2</td>
<td>0.4</td>
<td>0.1</td>
<td>0.1</td>
</tr>
<tr>
<td>1st Cousins</td>
<td>White</td>
<td>98.7</td>
<td>1.0</td>
<td>0.2</td>
<td>0.0</td>
<td>0.0</td>
<td>0.0</td>
</tr>
<tr>
<td></td>
<td>Black</td>
<td>97.4</td>
<td>2.0</td>
<td>0.5</td>
<td>0.1</td>
<td>0.0</td>
<td>0.0</td>
</tr>
<tr>
<td>Non-Biological Kin</td>
<td>White</td>
<td>82.6</td>
<td>13.9</td>
<td>2.7</td>
<td>0.6</td>
<td>0.2</td>
<td>0.1</td>
</tr>
<tr>
<td></td>
<td>Black</td>
<td>79.5</td>
<td>16.0</td>
<td>3.4</td>
<td>0.8</td>
<td>0.2</td>
<td>0.1</td>
</tr>
</tbody>
</table>

NOTE: Numbers in cells are expressed as percentages.
Table 6: Percentage Simulated Living Donor Evaluation Outcome, by Race and Genetic Relationship

<table>
<thead>
<tr>
<th>Genetic Relationship</th>
<th>Race</th>
<th>Donor</th>
<th>Exclusions</th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>HLA</td>
<td>ABO</td>
<td>Contraindication</td>
<td>Positive Crossmatch</td>
</tr>
<tr>
<td>All</td>
<td>White</td>
<td>6.43</td>
<td>62.48</td>
<td>48.50</td>
<td>69.05</td>
<td>11.59</td>
</tr>
<tr>
<td></td>
<td>Black</td>
<td>5.44</td>
<td>64.31</td>
<td>52.57</td>
<td>69.87</td>
<td>15.12</td>
</tr>
<tr>
<td>Full Siblings</td>
<td>White</td>
<td>21.00</td>
<td>21.91</td>
<td>31.38</td>
<td>58.72</td>
<td>7.10</td>
</tr>
<tr>
<td></td>
<td>Black</td>
<td>18.18</td>
<td>21.92</td>
<td>33.27</td>
<td>62.77</td>
<td>9.06</td>
</tr>
<tr>
<td>Half Siblings</td>
<td>White</td>
<td>8.72</td>
<td>43.84</td>
<td>44.92</td>
<td>69.49</td>
<td>10.69</td>
</tr>
<tr>
<td></td>
<td>Black</td>
<td>9.17</td>
<td>43.87</td>
<td>47.79</td>
<td>65.41</td>
<td>13.38</td>
</tr>
<tr>
<td>Parents</td>
<td>White</td>
<td>13.45</td>
<td>0.00</td>
<td>35.40</td>
<td>77.63</td>
<td>7.11</td>
</tr>
<tr>
<td></td>
<td>Black</td>
<td>12.88</td>
<td>0.00</td>
<td>37.11</td>
<td>77.47</td>
<td>9.07</td>
</tr>
<tr>
<td>Children</td>
<td>White</td>
<td>22.30</td>
<td>0.00</td>
<td>35.17</td>
<td>63.20</td>
<td>6.85</td>
</tr>
<tr>
<td></td>
<td>Black</td>
<td>18.87</td>
<td>0.00</td>
<td>37.07</td>
<td>66.98</td>
<td>9.14</td>
</tr>
<tr>
<td>Grandparents</td>
<td>White</td>
<td>6.21</td>
<td>43.88</td>
<td>44.90</td>
<td>78.38</td>
<td>10.55</td>
</tr>
<tr>
<td></td>
<td>Black</td>
<td>6.19</td>
<td>43.90</td>
<td>47.90</td>
<td>76.76</td>
<td>12.80</td>
</tr>
<tr>
<td>Grandchildren</td>
<td>White</td>
<td>5.22</td>
<td>43.87</td>
<td>44.58</td>
<td>81.92</td>
<td>9.87</td>
</tr>
<tr>
<td></td>
<td>Black</td>
<td>5.09</td>
<td>43.85</td>
<td>47.85</td>
<td>80.83</td>
<td>13.96</td>
</tr>
<tr>
<td>Aunts/ Uncles</td>
<td>White</td>
<td>11.84</td>
<td>43.77</td>
<td>44.99</td>
<td>58.88</td>
<td>10.32</td>
</tr>
<tr>
<td></td>
<td>Black</td>
<td>9.30</td>
<td>43.86</td>
<td>47.98</td>
<td>65.38</td>
<td>12.45</td>
</tr>
<tr>
<td>Nieces/ Nephews</td>
<td>White</td>
<td>7.44</td>
<td>43.87</td>
<td>44.94</td>
<td>74.06</td>
<td>10.79</td>
</tr>
<tr>
<td></td>
<td>Black</td>
<td>7.63</td>
<td>43.91</td>
<td>47.86</td>
<td>71.07</td>
<td>13.79</td>
</tr>
<tr>
<td>1st Cousins</td>
<td>White</td>
<td>5.37</td>
<td>65.82</td>
<td>49.78</td>
<td>66.08</td>
<td>12.23</td>
</tr>
<tr>
<td></td>
<td>Black</td>
<td>4.87</td>
<td>65.84</td>
<td>53.28</td>
<td>66.13</td>
<td>14.78</td>
</tr>
<tr>
<td>Non-Biological Kin</td>
<td>White</td>
<td>1.67</td>
<td>87.74</td>
<td>54.26</td>
<td>67.02</td>
<td>13.60</td>
</tr>
<tr>
<td></td>
<td>Black</td>
<td>1.40</td>
<td>87.73</td>
<td>58.54</td>
<td>68.63</td>
<td>17.61</td>
</tr>
</tbody>
</table>

NOTE: The ‘Donor’ column is the percentage of kin of that relationship type simulated to be a suitable living donor – i.e., at least two HLA matches, a compatible ABO blood type, no contraindication health conditions, and no positive crossmatch. The remaining columns are the percentage of kin of that type excluded for the indicated reason. These categories are non-exclusive – if a family member was excluded for multiple reasons they are included in the numerator for all such reasons. No parents or children were excluded for HLA reasons because all parents and children share at least three HLA genes in common.
Table 7: Estimated Percentage Race Gap Explained, by Simulation Counterfactual

<table>
<thead>
<tr>
<th>Counterfactual</th>
<th>ANY DONOR</th>
<th>NUMBER OF DONORS</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Whites</td>
<td>Blacks</td>
</tr>
<tr>
<td></td>
<td>Mean</td>
<td>Mean</td>
</tr>
<tr>
<td></td>
<td>90% Interval</td>
<td>90% Interval</td>
</tr>
<tr>
<td>Baseline</td>
<td>0.58</td>
<td>0.63</td>
</tr>
<tr>
<td></td>
<td>(0.57,0.59)</td>
<td>(0.61,0.64)</td>
</tr>
<tr>
<td>Genetic</td>
<td>0.59</td>
<td>0.64</td>
</tr>
<tr>
<td></td>
<td>(0.58,0.60)</td>
<td>(0.63,0.65)</td>
</tr>
<tr>
<td>Kinship</td>
<td>0.60</td>
<td>0.59</td>
</tr>
<tr>
<td></td>
<td>(0.59,0.61)</td>
<td>(0.58,0.60)</td>
</tr>
<tr>
<td>Health</td>
<td>0.58</td>
<td>0.62</td>
</tr>
<tr>
<td></td>
<td>(0.57,0.59)</td>
<td>(0.61,0.64)</td>
</tr>
<tr>
<td>PRA</td>
<td>0.58</td>
<td>0.63</td>
</tr>
<tr>
<td></td>
<td>(0.57,0.59)</td>
<td>(0.62,0.64)</td>
</tr>
<tr>
<td>All</td>
<td>0.61</td>
<td>0.61</td>
</tr>
<tr>
<td></td>
<td>(0.60,0.61)</td>
<td>(0.60,0.61)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Counterfactual</th>
<th>Whites</th>
<th>Blacks</th>
<th>% Race Gap</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>Mean</td>
<td>Explained</td>
</tr>
<tr>
<td></td>
<td>90% Interval</td>
<td>90% Interval</td>
<td></td>
</tr>
<tr>
<td>Baseline</td>
<td>1.10</td>
<td>1.25</td>
<td>--</td>
</tr>
<tr>
<td></td>
<td>(1.08,1.11)</td>
<td>(1.20,1.29)</td>
<td></td>
</tr>
<tr>
<td>Genetic</td>
<td>1.07</td>
<td>1.29</td>
<td>-40.59</td>
</tr>
<tr>
<td></td>
<td>(1.06,1.09)</td>
<td>(1.25,1.33)</td>
<td></td>
</tr>
<tr>
<td>Kinship</td>
<td>1.18</td>
<td>1.10</td>
<td>150.76</td>
</tr>
<tr>
<td></td>
<td>(1.16,1.20)</td>
<td>(1.07,1.13)</td>
<td></td>
</tr>
<tr>
<td>Health</td>
<td>1.09</td>
<td>1.25</td>
<td>-1.53</td>
</tr>
<tr>
<td></td>
<td>(1.07,1.11)</td>
<td>(1.21,1.29)</td>
<td></td>
</tr>
<tr>
<td>PRA</td>
<td>1.08</td>
<td>1.27</td>
<td>-20.39</td>
</tr>
<tr>
<td></td>
<td>(1.06,1.11)</td>
<td>(1.24,1.30)</td>
<td></td>
</tr>
<tr>
<td>All</td>
<td>1.15</td>
<td>1.15</td>
<td>97.23</td>
</tr>
<tr>
<td></td>
<td>(1.12,1.17)</td>
<td>(1.13,1.17)</td>
<td></td>
</tr>
</tbody>
</table>

NOTE: 90% interval columns indicate the range in the indicated figure for the 5th and 95th percentiles of simulations for the indicated counterfactual condition. The ‘% Race Gap Explained’ column is the percentage degree to which the race gap between blacks (higher) and whites (lower) is ameliorated in the counterfactual condition. Negative values in this column indicate an increase in this gap; values between 0 and 100 indicate a partial amelioration, and values greater than 100 indicate a reversal of the inequality.
Table 8: Ratio of Living Donor Transplants to Simulated Available Living Donors, by Race

<table>
<thead>
<tr>
<th>Race</th>
<th>Living Donor Transplant (Observed)</th>
<th>Available Living Donor (Simulated)</th>
<th>% Available Transplanted</th>
</tr>
</thead>
<tbody>
<tr>
<td>White</td>
<td>0.192</td>
<td>0.585</td>
<td>32.8</td>
</tr>
<tr>
<td>Black</td>
<td>0.088</td>
<td>0.618</td>
<td>14.2</td>
</tr>
</tbody>
</table>

NOTE: ‘% Available Transplanted’ is calculated as (Transplant / Available)*100, and interpretable as the estimated percentage of suitable living donors who actually donate a kidney for transplantation. Observed living donor transplant rates by race are subsetted to candidates on the waiting list 2000-2007 only to adjust for censoring differences by race.
Figure 1: Theoretical Framework

Diagram showing relationships between factors such as Kin Health, Kinship Size, Genetic Relationships, Opportunity Structure, Probability of Genetic Crossmatch, and Probability of Genetic Match, leading to outcomes such as Knowledge of Transplantation, Living Donor Transplantation, and Interest in Transplantation.
Figure 2: Illustration of Research Design

NOTE: The black circle indicates the reference transplant candidate; other circles represent kin. The blue sections of each kin circle represent the probability that a person of that relationship type will be a medically and genetically suitable living donor for the reference person. The black X indicates that this person is deceased. This figure is for illustrative purposes only.
Figure 3: Illustration of Kinship Network Matching Algorithm

NOTE: This figure is for illustrative purposes only. The spreadsheet on the left represents the kidney transplant waiting list data. The ‘u’ column in this spreadsheet represents values drawn from a uniform random distribution. The spreadsheet on the right represents persons in the PSID data, from which kinship ego networks will be probabilistically assigned to waiting list candidates. The rows highlighted in dark indicate the PSID observations whose kinship networks were simulated to be assigned to waiting list candidates, and the arrows link the merged observations. Observations are merged based on identical race, education, age, and gender combinations, then PSID observations meeting these requirements are assigned proportionate to their sample weights, using the uniform random draw assigned to the kidney transplant candidates (‘u’) compared to the weight range assigned to the PSID candidate (‘Range’ column). The range column is constructed to have a range equal to the proportion of total weights for demographically identical persons represented by the observation’s weight. Further details are provided in the text of the paper.