Enhancing Kidney Supply Through Geographic Sharing in the United States*

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Abstract

The deceased-donor kidney allocation system suffers from a severe shortage of available organs. We illustrate a mechanism which can increase the supply of cadaveric kidneys in the United States. This supply increase exploits the fact that under the current kidney allocation policy, some kidneys remain unprocured in some procurement areas but would be highly sought in other areas. The current kidney allocation policy procures within a Donor Service Area (DSA) and offers these kidneys first to patients in the DSA; if these offers are not accepted, the kidney will be offered within the region (a cluster of DSAs); if these offers are not accepted, the kidney will be offered nationally. A deceased-donor organ is procured if there is the belief that the offered organ will be transplanted (known as “intent”). We conduct an empirical analysis of the donor and recipient data (at the DSA level) which reveals that the intent increases significantly with organ quality, the median waiting time for a transplant, and higher competition. In particular, it shows that lower quality organs are likely to be procured at a higher rate in DSAs with longer waiting times. Motivated by a new kidney allocation system, we conduct a counterfactual study which shows that geographically broader sharing the bottom 15% quality kidneys leads to stronger intent for the organ. The stronger intent results in an increase in the procurement rates of those organs, thus increasing the supply of procured organs available for transplantation. In particular, the regional sharing of those organs leads to an expected 58 additional procured kidneys per year (3.3% supply increase among the bottom 15% quality kidneys), whereas the national sharing results in an expected 129 additional kidneys per year, increasing the supply of the bottom 15% quality kidneys by 7.3%. We also propose a variation of the new policy that increases the quality threshold to the lowest 20% quality, which results in an even greater increase of the supply than from the current policy. Our analysis shows that this new threshold policy leads to an expected 174 additional kidneys procured per year with national sharing.

Key words: kidney transplantation, discrete choice model, game theory, control function approach

*Formerly titled “What drives the geographical differences in deceased-donor organ procurement in the U.S.?”
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1 Introduction

The gap between the demand for and the supply of cadaveric kidneys has continued to grow steadily. There are currently over 97,000 End-Stage Renal Disease (ESRD) patients waiting for a transplantation in the United States (U.S.); and the growth of the waiting list of patients continues to outstrip the supply of kidneys.\(^1\) The supply shortage in (deceased-donor) kidneys is a first order issue. The allocation policy is effectively transformed into a rationing rule due to this shortage, and a large number of deaths result from this shortage every year.\(^2\) Any increase in the supply of procured organs directly improves the well-being of ESRD patients. Therefore, we seek a mechanism to increase the supply of organs for transplantation as our primary research focus. This differs from most of the existing work in the operations research literature which has focused on the demand side of the deceased-donor allocation problem. We focus on the procurement rate of organs from a given set of donors as the source of supply by conducting an analysis at the Donor Service Area (DSA) level, and indeed ultimately find that by making simple changes to the organ allocation policy which encourages a greater sharing of lower quality organs, more kidneys may be procured and supplied for transplantation.

The procurement and transplantation of organs in the United States operates within the Organ Procurement and Transplantation Network (OPTN) which is governed by the United Network for Organ Sharing (UNOS), authorized by the U.S. Congress. The United States is divided geographically into 11 regions, which are further divided into 58 DSAs. The procurement of deceased-donor organs within each DSA is administered by a local Organ Procurement Organization (OPO).\(^3\) Just like UNOS, each OPO is a non-profit entity regulated by the government, although the OPO is directly responsible for arranging the recovery, testing, tissue typing of organs, and packaging and transporting them to transplantation hospitals. The OPO is also responsible for deciding whether to procure an organ when it becomes available within the DSA. The procurement is done according to the Final Rule\(^4\) issued by the Department of Health and Human Services (DHHS). The procurement occurs unless one of the following occurs: (i) the donor does not meet criteria for eligible donor, (ii) the organ has been ruled out by basic donor information or by laboratory data prior to the donor entering the operating room for excision of organs, (iii) the family does not agree to

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\(^2\) During 2006-2011, more than 4500 patients died each year while waiting for a kidney.

\(^3\) In the remainder of the paper, we will refer to the unique geographic area that the OPO serves as the DSA. Also, note that there is one-to-one relationship between an OPO and its DSA.

donate the organ, (iv) the search for a recipient for that organ has ended unsuccessfully prior to the donor’s entrance into the operating room. If none of these four conditions is true, the DHHS Final Rule states that “intent” is present and the procurement may proceed. We will refer to this “intent” throughout the remainder of the paper as equivalent to procuring the organ.

Unfortunately, not every medically acceptable (deceased-donor) organ is procured and offered for transplantation. This is rather surprising given the severe organ shortage. Understanding the subtleties of the organ allocation system sheds some light on the reasons for this. Upon hearing of the availability of a cadaveric organ, the OPO must assess that intent is present and if so, the organ is procured. However, if the OPO deems that there will not be a willing recipient, typically because the organ may be of lower quality, the organ may not be procured. The intent is established prior to the donor entering the operating room. If the OPO believes there is at least one patient in the DSA who would seriously consider accepting the organ (after deeming the organ is medically acceptable and the family has given consent), then intent is established and procurement will proceed (i.e., “intent” = “procurement”). As discussed in Section 4, our data set includes the information of whether or not an organ is procured. Thus, we observe in the data whether intent was expressed for each medically eligible organ.

Our primary research objective is to investigate the extent of additional kidneys which may be procured if some small changes to the UNOS allocation policy are considered. These kidneys are those which are currently not procured due to their marginal quality and are most likely concentrated in some areas of the country (the high quality kidneys are procured throughout the country; it is the lower quality kidneys which may not be procured in some areas). The change to the UNOS policy we study concerns a greater degree of sharing of these lower quality organs to encourage higher levels of procurement of them. To further understand this, in our empirical study we investigate how the three factors (organ quality, median waiting time in the DSA, transplant center competition in the DSA) affect intent in those different DSAs. The organ quality varies from donor to donor. Moreover, the median waiting times and the degree of transplant center competition vary across different DSAs; so does the intent. The variation in these three factors helps us glean the relationship between the intent and these factors. Building on this analysis, through a counterfactual analysis we then determine the extent of additional kidneys of marginal quality which may be procured under some targeted small changes to the UNOS allocation policy.

UNOS’ geographically-based procedure for seeking a recipient for an organ is the following. First, the OPO is obliged to seek a recipient from the waiting list within the DSA; if no willing
recipient is found in the DSA, the OPO seeks a willing recipient within the region (but outside the DSA); if there are no willing recipients within the region, the organ may then be offered to patients of the waiting list nationally (i.e., outside the region). Given the limited time until the donor’s entrance to the operating room, the intent (procurement) is strongly correlated with the acceptance of the organ (by a recipient in the local DSA). The slight policy modification we are considering is for the OPO to seek recipients for the lower quality kidneys within the region (or the nation) immediately, without seeking them within the DSA first, i.e. the broader sharing of lower quality organs.

We first develop and analyze a game theoretical model of a DSA to study the patients’ accept/reject decisions for deceased-donor organ offers. In essence, patients decide between accepting an organ offer or waiting for a better quality organ. This model helps us understand how the acceptance probability of a deceased-donor organ (and hence, the intent for it) change with the organ quality and the congestion in a DSA (e.g., median waiting time for a transplant) and helps develop testable hypothesis related to organ quality and waiting time until transplantation. Three hypotheses pertaining to how intent is affected by organ quality, waiting time until transplantation, and competition between transplant centers in a DSA (motivated by the literature) are defined prior to testing in our empirical model. The estimation is done using an endogeneity-corrected\(^5\) probit model, described in more detail in Section 5.

We find that the organ quality is the most important factor determining intent. In particular, the intent (and the procurement rate) increases as the quality improves. We also observe that, when considering all the data, procurement rate in a DSA also increases as the median waiting time until transplantation and the competition among transplant centers in that DSA increase. However, the significance of these two variables (i.e., waiting time until transplantation and the competition) is not kept at all times when we partition the data by organ quality or blood type. Importantly, we find that while the waiting time until transplantation is significant when estimating using only the lower quality organs, it is not significant when considering higher quality organs. This suggests that OPOs with substantial median waiting times are more likely to procure the lower quality organs knowing there is a likelihood that someone in the DSA will accept it.

Using the results of the probit estimation, we then undertake a counterfactual study to consider how the supply of organs may be enhanced by an adjustment of the UNOS allocation policy amongst

\(^5\)We extend the control function approach advocated by Petrin and Train (2010) to a multi-variate context to correct for endogeneity. This technique is described in more detail in Section 5.
the lower quality organs. By considering a broader sharing of the lowest quality kidneys (bottom 15%), e.g., regionally or nationally, we observe that more of those organs may be procured under this policy than under the original policy. This policy change is suggested as part of the proposal to substantially revise the kidney allocation policy and the new policy became effective in December 2014.\footnote{https://optn.transplant.hrsa.gov/news/revised-national-kidney-transplant-allocation-system-is-now-in-place, accessed on May 1, 2017.} The analysis in Section 6 shows that 58 additional organs will be procured per year under regional sharing of the bottom 15% of the organs, increasing the supply of the bottom 15% quality kidneys by 3.3%. Similarly, 129 additional organs will be procured per year under national sharing of the bottom 15% of the organs, which reflects the addition of a small- to medium-sized DSA (a supply increase of 7.3% among the bottom 15% quality kidneys). In addition, to inform policy makers further, we extend the counterfactual study and quantify the impact of varying the quality threshold for broader sharing from 15% to 20%. This further fine-tuning of the quality threshold can increase the supply of procured deceased donor kidneys by 174 per year. This increase is significant and corresponds to an addition of a medium-sized DSA, or an increase of 1.2% of the total kidney supply on average each year.

The remainder of the paper is structured as follows. Section 2 provides a literature review. Section 3 develops a game theoretical model of a DSA and describes the transplant center competition by citing the relevant literature, both of which help formulate testable hypotheses. Section 4 describes the data. Section 5 introduces the econometric method and the estimation results. The counterfactual study is undertaken in Section 6. Section 7 concludes. Proofs, additional details of the calculations of variables and figures, the details of the endogeneity correction method, additional estimation results and tables are provided in the Appendix.

2 Background and Literature Review

U.S. Congress passed the National Organ Transplant Act (NOTA) in 1984 to address the deceased-donor organ shortage. Since the passing of this legislation, UNOS has managed the allocation of deceased donor organs in the U.S. The current kidney allocation policy of UNOS is a point system that prioritizes the potential transplant candidates based on medical criteria and the waiting time; see OPTN (2014a) for details. Su and Zenios (2004) note that “The continued shortage of organs and the associated explosion in waiting times has contributed to a convergence of this point system to a system that resembles first-come-first-served (FCFS).” The recent work Schummer (2016)
explores the welfare implications of increasing the acceptance of lower quality organs by (the highly-ranked) patients on the waitlist. The author considers a stylized model where an infinite number of ordered patients are present and make accept/reject decisions. Patients leave the system only upon receiving a transplant. The possibility of death is not modeled directly although the patients’ impatience can be attributed to death and the results in that case may shed light to the case allowing patients to die. Schummer (2016) shows that not interfering with patients’ decisions is Pareto dominant if the patients are risk-neutral or risk-averse and patient. However, when they are impatient the result no longer holds. In particular, there is a tradeoff to be made in that case, e.g., highly ranked patients may suffer whereas those with lower rankings may benefit from increasing the acceptance of lower quality organs by the highly-ranked patients. The author also discusses the implications of the result for the “organ spoilage” problem because the organs may spoil during the sequential offer process due to the limited cold ischemia time.

As mentioned earlier, the point system of the current kidney allocation system is crucially embedded in the geographically-tiered structure: A deceased-donor kidney is first offered to the patients based in the same DSA. If no patient within the DSA accepts the offer, then it is offered to the patients in the same region. Finally, if no patient in the region accepts the offer, then the kidney is offered nationally. These offers must be made before the end of the cold ischemia time during which the procured kidney remains viable for transplantation. Opelz and Döhler (2007) states that cold ischemia time up to 18 hours doesn’t have much influence on the graft survival rates, but cold ischemia time longer than 18 hours can be detrimental. This geographically tiered structure of the policy makes it difficult for organs to be shared across different DSAs. Under the current policy, the vast majority (more than 70%, see Davis (2011)) of deceased donor kidneys are transplanted locally. Therefore, the differences in supply and demand characteristics of different DSAs lead to a significant disparity in waiting times and access to transplantation across different DSAs. Davis (2011) notes that “The overall median waiting time to receive kidney transplantation during 2000-2009 varies from 0.93 years to 4.14 years depending on a patient’s local area of listing.” This discrepancy is even more pronounced for patients with blood types B and O.

The demand side (i.e., the allocation of deceased organs) of the organ transplantation has received substantial attention in the operations research literature. To design optimal allocation policies, researchers seek to match patients and organs to maximize social welfare, see Righter (1989), David (1995), David and Yechiali (1990), and David and Yechiali (1995). Zenios et al. (2000) explores the efficiency-equity trade-off and proposes a dynamic index policy for deceased-

Davis (2011) proposes a probabilistic sharing of available kidneys in neighboring DSAs to address the geographic inequities. Ata et al. (2016b) proposes an operational solution using jets to multiple-list patients to ameliorate the geographic inequity. Their proposal is an incremental solution within the existing system and does not require a policy change. Halldorson et al. (2013) consider the effect of competing transplant centers within a DSA and find that liver patients are more likely to accept donated liver under competition than when no competition exists.

Several researchers consider an individual patient’s problem of accepting/rejecting an organ offer while waiting for a transplant; see for example, David and Yechiali (1990), Ahn and Hornberger (1996), Hornberger and Ahn (1997), Alagoz et al. (2004, 2007a,b), Sandikci et al. (2008), and Sandikci et al. (2013).

Virtually the entire operations research literature takes the supply of organs as given and focuses on the allocation problem. An exception to this is the work on paired kidney exchange, see for example Roth et al. (2005, 2007) and Zenios (2002); also see Ashlagi and Roth (2011). This stream of literature aims at maximizing the use of living donors by resolving various matching difficulties between recipient-donor pairs, which may lead to an increase in the supply of living donors. In contrast, we focus on understanding ways of increasing the supply of procured deceased-donor organs. Another exception is Arora and Subramanian (2017) who analyze the supply side entities’ (i.e., OPO and transplant hospital) decisions on societal outcomes. They show that there exist misalignments between the social planner and supply side players in the cadaver organ donation value chain and propose a pareto-improving contract that achieves socially optimal performance.

Recent strategies to increase the supply of organs in practice include the use of expanded criteria donor (ECD) kidneys and donation after cardiac death (DCD) kidneys; see for example Metzger

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7 These kidneys are from donors older than sixty, or between the ages of 50-59 with at least two of the following comorbidities: hypertension history, serum creatinine > 1.5 mg/dl or cause of death from cerebrovascular accident.
et al. (2003) and O’Connor and Delmonico (2005). Medical research shows that short-term (Stratta et al. (2004)) and the intermediate-term (Stratta et al. (2006)) outcomes of transplants using ECD organs are comparable to those using standard criteria organs. Our work complements these efforts and helps understand what factors affect the procurement rate of organs. Thus, it can help increase the supply of deceased-donor organs further.

Our paper is also related to the growing body of literature on how workload (provider load or patient waiting time) affects clinical decisions and patient outcomes. Kc and Terwiesch (2012) show that at higher levels of intensive care unit (ICU) occupancy, a patient’s early discharge probability increases. However, early discharge is associated with increased likelihood of revisiting the ICU in the future. A similar adverse effect of workload is demonstrated in a study by Kuntz et al. (2014) which shows that when occupancy levels exceed a certain tipping point, a patient’s mortality risk increases significantly. A recent paper by Freeman et al. (2016) shows that increased levels of workload have varying effects depending on the complexity of a patient’s need. In particular, they find that gatekeeper providers display a rationing effect of resource-intensive services for noncomplex cases, whereas they increase the rate of specialist referrals for complex cases. Kc and Terwiesch (2009), Kim et al. (2014), Tan and Netessine (2014), Batt and Terwiesch (2016), and Jaeker and Tucker (2016) are other operations management papers in this stream of research. Different from these papers, our study investigates the impact of transplant center congestion on the deceased donor kidney procurement decisions.

3 Hypotheses Development

3.1 A Game Theoretical Model

This subsection develops an overloaded fluid model of an OPO and considers patients’ accept/reject decisions for deceased-donor organ offers. The model helps glean insights about what factors affect the acceptance probability of an organ, and hence, the OPO’s intent. We formalize the findings of the model as hypotheses and test them in Section 5. As mentioned earlier, when a kidney becomes available, it is procured if the OPO expresses intent. This intent is not a guarantee that the organ will ultimately be transplanted, but if and only if an OPO shows intent, the organ will be procured; i.e., “intent” = “procurement.” Although the OPO’s intent is not captured directly in our model, the intent is the result of the OPO’s belief that at least one patient in the DSA is willing to seriously consider accepting the organ. The model derives the equilibrium quality threshold of
patients accepting organ offers in a DSA which can serve as a proxy for the OPO’s intent.

To be specific, we consider a DSA in isolation and develop a stylized game theoretic model which incorporates: (i) the organs offered by the OPO are of varying quality, and thus, correspond to different post-transplant life years; and (ii) patients can turn down organ offers with no penalty.

Patients may die while waiting for a transplantation. We assume that the hazard rate of time-to-death distribution, denoted by $\gamma(t)$ for $t \geq 0$, is nondecreasing. The expected post-transplant life years associated with an organ takes a value in the range $[L, \bar{L}]$, where $\bar{L} > 1/\gamma(t)$ for all $t \geq 0$, which means that patients prefer the highest quality kidney to staying on dialysis at all times.\(^8\)

The post-transplant life expectancy $L$ associated with an organ can be thought of as the organ’s quality as there is a strong correlation between the two. Let $\lambda$ and $G(y)$ denote the patient arrival rate and the quantity (measure) of organs whose life expectancy is less than or equal to $y$ years, respectively. We assume that $G$ is continuously differentiable on $[L, \bar{L})$ but has a jump at $\bar{L}$, i.e.,

$$\Delta G(\bar{L}) = G(\bar{L}) - G(\bar{L}-) > 0, \tag{1}$$

which corresponds to assuming that arrival rate of the number of highest quality organs is not zero. In our model, these organs are always transplanted, as will be seen below. Morever, the high quality organs are always transplanted in practice. Therefore, $\Delta G(\bar{L})$ can be viewed as the arrival rate of organs with sufficiently high quality so that they are always transplanted.

We assume that $G, \lambda,$ and $\gamma(\cdot)$ are common knowledge among patients. Each patient chooses a threshold life-expectancy for organs acceptable to him as a function of how long he has been waiting. That is, the patient is willing to accept any organ whose life expectancy is above a threshold, but not otherwise. We assume a stationary (overloaded) fluid model of the system, i.e., the model parameters are not time varying; and we are interested in the steady-state equilibrium behavior of the system. Namely, the transplant waiting list will be stationary in steady-state. Let $\tau$ denote the longest waiting time in that stationary system which is determined endogenously. Then the strategy of a patient is denoted by a function $l : [0, \tau] \rightarrow [L, \bar{L}]$, where $l(t)$ denotes the life-years threshold associated with the lowest quality organ a patient, who has waited for $t$ time units, is willing to accept. We restrict attention to (pure strategy) symmetric equilibria, where each patient chooses the same $l(\cdot)$. Also, without loss of generality\(^9\) we restrict attention to nondecreasing $l(\cdot)$

\(^8\)Our analysis only uses the weaker condition that $\bar{L} > 1/\gamma(\tau)$ where $\tau$ is the longest waiting time to get a transplant in equilibrium.

\(^9\)Given a general function $f(\cdot)$ as a patient’s strategy, it can be replaced by the largest nondecreasing function $\hat{f}$ such that $\hat{f} \leq f$ without changing the outcomes because organs are allocated on a FCFS basis and the system is
functions. That is, patients become more selective as they wait longer because they are closer to the top of the queue. Moreover, it is straightforward to argue that\(^{10}\) \(l(\tau) = \bar{L}\).

Let \(\{Q(t) : t \in [0, \tau]\}\) denote the stationary queue length profile. That is, \(Q(t)\) denotes the intensity of patients who waited for \(t\) time units in the system. The following flow-balance equations characterize the stationary queue length profile:

\[
Q(0) = \lambda, \\
Q'(t) = -\gamma(t)Q(t) - G'(l(t))l'(t), \quad 0 < t < \tau, \\
Q(\tau) = \Delta G(\bar{L}),
\]

where the last equation follows since \(l(\tau) = \bar{L}\) and that, the intensity of patients who have waited for \(\tau\) time units, i.e., \(Q(\tau)\), must equal the intensity of organs of \(\bar{L}\) life years, i.e., \(\Delta G(\bar{L})\).

The following proposition characterizes the (pure strategy) symmetric Nash equilibrium for patients’ accept/reject decisions and the resulting queue length profile.

**Proposition 1** The patients’ equilibrium decisions are characterized by the threshold function \(l(\cdot)\) given by

\[
l(t) = \max \left\{ \bar{L}, \exp \left\{ \int_0^t \gamma(s)ds \right\} \left[ \bar{L} \exp \left\{ -\int_0^\tau \gamma(s)ds \right\} + \int_t^\tau \exp \left\{ -\int_0^s \gamma(u)du \right\} ds \right\} \right\},
\]

where \(\tau\) is the unique solution of the following equation:

\[
\int_0^\tau \exp \left\{ \int_0^s \gamma(u)du \right\} dG(l(s)) = \lambda.
\]

overloaded. Recall that patients are assumed homogeneous, and they are differentiated only through their waiting time. Consider the scenario where all patients have a strictly decreasing \(l(t)\). Now consider two patients who have waited \(t_1\) and \(t_2\) where \(t_1 > t_2\). Consequently, \(l(t_1) < l(t_2)\). The implication is that patient 1 would accept all the kidneys patient 2 would accept (that is, those with quality \(l\) such that \(l(t_2) < l < l(t_1)\)) as well as some kidneys patient 2 would not accept (those kidneys with quality \(l\) such that \(l(t_1) < l < l(t_2)\)), since patient 1’s threshold is lower than that of patient 2. Thus, there will never be kidneys of a quality acceptable to patient 2 which will be offered to patient 2 since they will be already accepted by patient 1 beforehand, in this over-loaded queue setting where demand outstrips supply. Consequently, without loss of generality, we can replace the decreasing \(l(t)\) function with a non-decreasing function without affecting the acceptance behavior of patients.

\(^{10}\) If \(l(\tau) < \bar{L}\), then a patient who waited for \(\tau\) time units can deviate and wait for \(\epsilon > 0\) time units more (resulting in a total wait of \(\tau + \epsilon\)) and can receive an organ which offers \(\bar{L}\) life years. This results in a strict improvement in the patient’s utility provided \(\epsilon > 0\) is sufficiently small, but contradicts that \(\tau\) is the longest wait in the system. Therefore, \(l(\tau) = \bar{L}\).
The corresponding stationary queue-length profile is characterized by

\[ Q(t) = \exp \left\{ - \int_0^t \gamma(s) ds \right\} \left[ \lambda - \int_0^t \exp \left\{ \int_0^s \gamma(u) du \right\} dG(l(s)) \right] \quad \text{for } t < \tau, \]  

(7)

and \( Q(\tau) = \Delta G(\bar{L}) \). Moreover, as \( \lambda \) increases, \( \tau \) increases strictly, i.e., patients wait longer and \( l(t) \) decreases strictly (unless it equals \( L \)) for all \( t < \tau \).

The \( l(t) \) curve is the equilibrium solution of all the patients in the DSA, defining their willingness to accept an organ of a specific quality at a particular time since they listed as a transplant patient. The interpretation of \( l(t) \) is that it reflects the OPO’s intent. The OPO knows the population in its DSA and the profile of patients who are listed at transplant centers in the DSA and thus, the patient’s acceptance threshold acts as a surrogate for the OPO’s statement of intent, as discussed earlier. Proposition 1 shows that as the DSA gets more congested (i.e., \( \lambda \) and \( \tau \) increases), patients waiting for a transplant are willing to accept a lower quality organ (i.e., \( l(t) \) decreases for all \( t \)).

The expressions in Proposition 1 simplify as shown in Corollary 1 below if the death rate is constant over time.

**Corollary 1** When the death rate is constant, i.e., \( \gamma(s) = \gamma \), the patients’ equilibrium decisions are characterized by the threshold function \( l(\cdot) \) given by

\[ l(t) = \max \left\{ \frac{L}{\gamma} - \frac{1}{\gamma} + \left( L - \frac{1}{\gamma} \right) e^{-\gamma(\tau-t)} \right\}, \quad t \in [0, \tau], \]  

(8)

where \( \tau \) is the unique solution of the following equation:

\[ e^{\gamma\tau} \int_{l(0)}^{\bar{L}} \left( u - \frac{1}{\gamma} \right) dG(u) = \lambda \left( \bar{L} - \frac{1}{\gamma} \right). \]  

(9)

Moreover, the stationary queue-length profile is characterized by

\[ Q(t) = e^{-\gamma t} \left[ \lambda - e^{\gamma\tau} \int_{l(0)}^{l(t)} \frac{u - 1/\gamma}{\bar{L} - 1/\gamma} dG(u) \right] \quad \text{for } t < \tau. \]  

(10)

Proposition 1 shows that as the DSA gets more congested, i.e., as \( \lambda \) increases, the waiting time increases, and the patients become less selective in the sense that they are willing to accept lower quality organs. This, in turn, increases the acceptance probability of organs, and hence, the intent. We also see from Proposition 1 that organs with life expectancy \( l(0) \) or higher are accepted (and transplanted), whereas those with life expectancy lower than \( l(0) \) are rejected. Therefore, we arrive
at the intuitive conclusion that the intent is stronger for higher quality organs. We formalize these insights into the following two testable hypotheses.

**Hypothesis 1** As the organ quality increases, the OPO’s intent increases.

This hypothesis is motivated by the non-decreasing nature of the threshold function \( l(t) \). As the organ quality improves, more patients within the DSA will be willing to accept the organ.

**Hypothesis 2** As the waiting time until transplantation in a DSA increases, the OPO’s intent increases.

This hypothesis suggests that as the waiting time across all patients in the DSA increases (and hence, congestion increases), the willingness of patients to accept an organ also increases. This hypothesis follows from Proposition 1 and reflects the patients’ increased willingness to accept a potentially lower quality organ as the waiting time in the DSA increases.

### 3.2 Transplant Center Competition

There are 272 transplant programs in the U.S. certified (for each organ type) by the Centers for Medicare and Medicaid Services (CMS) to perform transplants. As mentioned in Section 1, the U.S. is geographically divided into 58 DSAs and each DSA can be composed of one or more transplant centers. There is significant variance in the number of transplant centers across different DSAs; seven DSAs have a single center serving the patients, while some others have ten or more centers competing with each other. Figure 1 displays a histogram of the number of transplant centers in each DSA, showing the heterogeneity of the number of centers.

![Figure 1: Histogram of the Number of Transplant Centers at Each DSA.](image)

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These transplant centers have varying objectives in terms of transplant volume, costs, and outcomes. In order to cover their fixed costs, transplant centers must perform a minimum number of transplants and maintain their market shares in their DSAs. In addition, they aim to receive an incremental profit with each additional organ they procure. As a result, transplant centers in DSAs with multiple centers can behave more aggressively in terms of patient acceptance and exhibit more demand for lower quality organs. On the contrary, transplant centers with no or little competition can create a greater organ wastage. We elaborate more on this center heterogeneity in Section 4 where we highlight certain aspects of and trends in the current kidney allocation system.

The following hypotheses are inspired by Halldorson et al. (2013), Adler et al. (2014), and Cho et al. (2015) all of which analyze the impact of the degree of competition on the transplantation system. In particular, Halldorson et al. (2013) examine the association between competition among transplant centers and post-transplant outcomes using data from cadaveric liver transplant recipients who underwent transplantation between 2003 and 2009. They find that transplant centers facing higher levels of competition are associated with increased patient access for sicker patients and increased utilization of higher risk (i.e., lower quality) organs in comparison to DSAs without competition. In a similar vein, Adler et al. (2014) demonstrate that higher proportion of riskier kidneys are used in the DSAs with higher competition using data from patients who underwent renal transplantation between 2003 and 2012. Using kidney patient listing data and the number of kidneys transplanted in 2011, Cho et al. (2015) indicate that competition increases the patient access (i.e., higher percentage of patients tend to be listed for transplant). However, Cho et al. (2015) report that the percentage of patients receiving transplants is not different at varying levels of competition among transplant centers. Combining the findings of these papers leads to inconclusive results regarding the intent of an OPO; i.e., Halldorson et al. (2013) and Adler et al. (2014) show a positive association between competition and intent, whereas Cho et al. (2015) do not observe a significant impact of competition on the intent. Hence, we test the following two alternative hypotheses regarding competition:

**Hypothesis 3a** As the competition within a DSA increases, the OPO’s intent increases.

**Hypothesis 3b** As the competition within a DSA increases, the OPO’s intent does not change.

We use the Herfindahl-Hirschman Index (HHI) as a measure for competition between transplant centers in the DSA, similar to Halldorson et al. (2013), Adler et al. (2014), and Cho et al. (2015). The HHI is a commonly accepted standard of economic measure of competition among players in
a particular industry or market. There are several papers in the operations management literature that measure market competition using HHI in a healthcare setting (see, Kc and Staats (2012), Andritsos and Tang (2014), and Lu and Lu (2016)). The details of the calculation of the HHI variable is provided in Section 5. Next we describe our data sources, the variables, the models, and test the above hypotheses.

4 Data Description

4.1 Data Sources

The data for this study comes from UNOS’ Standard Transplant Analysis and Research (STAR) Files. Our data set contains information regarding (i) all deceased kidney donors (i.e., donor data) and (ii) waiting list and transplants performed (i.e., recipient data) in the U.S. Our period of study is from January 1, 2000 through June 30, 2010. Overall, we have detailed information of 76,866 deceased donors and 111,579 actual or potential recipients. Table 1 shows some descriptive statistics of the relevant variables we use in our analysis. In Table 1, \( Y_i \) is the indicator variable showing if organ \( i \) is procured (i.e., dependent variable), \( KDRI_i \) is the quality of organ \( i \), \( W_{jt} \) is the median waiting time (in years) until transplantation in DSA \( j \) in quarter \( t \), \( W_{jk} \) is the median waiting time (in years) until transplantation in DSA \( j \) for blood-type \( k \) in quarter \( t \), and \( HHI_{jk} \) is the HHI for blood type \( k \) in DSA \( j \). The term \( HHI_{jk} \) captures the competition between transplant centers in a DSA. This index is calculated by summing the squares of the market share of each transplant center in a DSA. The market share of a transplant center is defined by using the total number of registered patients during our period of study at each DSA for each blood type. The HHI ranges from \( 1/n \) to 1 where \( n \) is the number of transplant centers in a DSA. The closer the HHI gets to zero, the greater the level of competition within a DSA. Our competition variable \( HHI_{jk} \), the kidney quality variable \( KDRI_i \), and the waiting time variable \( W_{jt} \) will be discussed further in Section 5. The waiting time and the competition variables are defined at the DSA level, and the quality variable is defined at the donor level.

The donor data set contains detailed information regarding each deceased kidney donor such as a disposition code, the date of recovery, demographic information of the donor, and several health indicators. The disposition code variable is especially important for our purposes. It helps identify the intent. There are 6 disposition codes for each kidney: (1) organ consent not requested, (2) organ
Variable & Sample Size & Mean & Std. Dev. & Median \\
--- & --- & --- & --- & --- \\
$Y_i$ & 76,866 & 0.90 & 0.30 & 1.00 \\
$KDRI_i$ & 76,399 & 1.33 & 0.51 & 1.21 \\
$W_{jt}$ & 2,516 & 1.79 & 1.00 & 1.63 \\
$HHI_{jk}$ & 244 & 0.41 & 0.28 & 0.33 \\
$W_{jk=A}t$ & 2,510 & 1.40 & 0.79 & 1.26 \\
$W_{jk=O}t$ & 2,508 & 2.03 & 0.95 & 1.91 \\
$W_{jk=AB}t$ & 1,077 & 1.04 & 0.89 & 0.84 \\
$W_{jk=B}t$ & 2,214 & 2.25 & 1.30 & 2.04 \\

$i$: index for donor, $j$: index for DSA, $t$: index for quarter, $k$: index for blood type

| Table 1: Descriptive Statistics of the Dependent and the Independent Variables. |

consent requested but not obtained, (3) organ consented but not recovered,\(^ {11}\) (4) organ recovered for reason other than transplant, (5) organ recovered for transplant but not transplanted, and (6) organ transplanted. When a donor kidney is assigned codes 1, 2, or 3, then it was not recovered; i.e., $Y_i = 0$. The remaining codes 4, 5, and 6 indicate that the kidney was recovered from the donor; i.e., $Y_i = 1$. As indicated in Table 1, the mean value of $Y_i$ equals 0.9 which means that 7,687 observations have $Y_i = 0$ (i.e., 10% of the sample size) and the remaining 69,179 observations have $Y_i = 1$.

The recipient data contains information regarding transplants (living and deceased donor types) and listings on the kidney, pancreas, and kidney/pancreas waitlists prior to September 3, 2010. Detailed demographic and health information of the recipient and the donor (if there is any) is available in this data set. An entry consists of a listing, a transplant, or both (if the listing resulted in a transplantation).

4.2 Data Trends

This subsection presents summary statistics of the data to highlight certain aspects of and trends in the kidney allocation system. As illustrated by Figure 2, while the number of deceased donors has remained relatively flat over the span of time our data covers, there has been a marked increase in the additions to the waiting list. This indicates not only that there is a substantial gap between the supply and demand for kidneys but that the gap is rapidly expanding. There are two deceased donor classifications to specify the quality of a donated kidney: (i) expanded criteria donors (ECD)\(^ {11}\) Only 0.3% of the observations have the disposition code 3.
and (ii) standard criteria donors (SCD). SCD donors often have fewer risks associated with graft failure whereas ECD organs typically relate to higher risks of earlier graft loss (Metzger et al. 2003, Pascual et al. 2008). All candidates are eligible to receive SCD kidneys; however, ECD kidneys are allocated only to candidates who have indicated a willingness to consider them.

Figure 3(a) shows the percentage procured and not procured donors by SCD-ECD breakdown and the proportion of ECD kidneys procured has grown. However, Figure 3(b) shows that there are similar numbers of not procured donors in each category (in absolute terms), so it is not simply that only inferior organs will be added to the supply. Collectively, over the 2000-2009 period, these two non-procured elements (ECD and SCD) appear to be a new growing source of kidneys for transplantation.

![Figure 2: Demand Outstripping Supply.](image)

![Figure 3: Kidneys Procured and Not Procured from 2000 to 2009.](image)

Figure 4 illustrates an example of geographical heterogeneity. In Figure 4(a), we see that there is a large and consistent difference in the median waiting time until transplantation for recipients...
in two geographically different DSAs. The median waiting time until transplantation in NYRT (New York Organ Donor Network) almost always exceeds two years while it is always less than a year in UTOP (Intermountain Donor Services), a DSA based in Utah. Moreover, we see that the quality of procured organs in these areas can differ markedly. In the remainder of the paper, we use the Kidney Donor Risk Index (KDRI) quality metric, which was introduced by UNOS in its most recent kidney allocation policy to fine-tune the binary SCD-ECD donor kidney quality classification system explained above. The KDRI is a reverse-scaled: a high number indicates lower quality than a low number.

Figure 4(b) shows the lowest quality organ procured in NYRT is consistently lower quality than the highest quality organ not procured in UTOP, in every year of our study. *This simple comparison shows that there was no intent in UTOP for organs for which intent would have been easily given in NYRT.* That is, there are organs of a quality not being procured in UTOP which would have been readily procured but not necessarily accepted in NYRT since they exceed the lowest quality organs procured in New York. Similar stories appear among several other OPOs nationally.

Next, we observe in Figure 5(a) that the average quality of transplanted kidneys has gradually worsened over time, perhaps reflecting that demand is progressively outstripping the supply of organs as shown in Figure 2, as well as the increased usage of ECD kidneys in recent years. Figure 5(b) supports two observations. First, it shows the relationship between the waiting time to transplant and the quality of the accepted organs. Within an OPO, the quality of the kidney received does not appear to be correlated with the median waiting time. Second, Figure 5(b) illustrates that the waiting time until transplantation for the CAOP OPO (OneLegacy, an OPO based in Los Angeles) is twice as long as for the ORUO OPO (Pacific Northwest Transplant Bank,
an OPO based in Portland, Oregon), a heterogeneity reflected across all 58 OPOs. There is also a similar heterogeneity across blood types, with longer average wait times for blood type B and O compared with types A and AB.

Finally, Figure 6 shows the average HHI by year and two blood types of the same two OPOs used in Figure 5(b). Traditionally, blood types B and O candidates experience the longest wait time, so we display the time trend of competition for these two blood types. While the competition for organs does not change significantly over time in an OPO, the competition level across different OPOs can be drastically different. We observe in Figure 6 that CAOP OPO kidney transplant market (has four transplant centers) is more competitive than that of ORUO OPO (has three transplant centers). The average HHI at CAOP OPO varies between 0.1 and 0.3; whereas the average HHI at ORUO OPO is always higher (less competition) and varies between 0.3 and 0.8. In addition, there is heterogeneity in competition across blood types within the same OPO. We also observe a similar trend at the aggregate level, the average HHI of all OPOs by year doesn’t vary much when the data is broken down by the blood type (varies between 0.5 and 0.65), however there exist differences across blood types. There seems to be slightly more competition for blood type O compared to blood type B (see, Appendix C, Figure 9).

5 Empirical Model

5.1 Variables Affecting the Acceptance Probability of a Kidney

Waiting Time until Transplantation in a DSA. The waiting time is one of the primary determinants of patients’ priority and decisions in the kidney allocation system. We calculate the
median waiting times of all patients who had transplants at each DSA during each quarter (between the first quarter of 2000 and the second quarter of 2010) by blood type ($W_{jkt}$); see Appendix B for the details of the calculation of these waiting times and all other relevant variables. As mentioned in Section 1, there are multiple sources of significant variation in the waiting times including different DSAs and different blood types. This difference is especially significant when one compares blood types O and B to A and AB and also across different DSAs.

**Kidney Donor Risk Index.** To measure the quality of offered kidneys, we use KDRI following the medical literature. This index and its mathematical model was first developed by Rao et al. (2009); see OPTN (2014b) for further details. This index converts a set of donor characteristics into a single number that captures the risk of graft failure after kidney transplant (i.e., an estimate of the relative risk of a graft failure after transplant of a particular donor compared to the median donor). The calculated score for each donor comes from “mathematical models based on a retrospective analysis of data collected by the Scientific Registry of Transplant Recipients on donor and recipient characteristics over the past several years” (Hippen et al. 2011, p. 1285). The main purpose of KDRI is to help transplant professionals better evaluate the quality and appropriateness of deceased donor kidneys and also to assist potential candidates in making more informed decisions. There are 10 factors considered in calculating the KDRI. These factors are donor age, height, weight, ethnicity, history of hypertension, diabetes status, serum creatinine level, cause of death, Hepatitis C Virus status, and DCD (donation after circulatory death) status. A more detailed explanation is available in Appendix B including the coefficient estimates obtained from the graft survival model of Rao et al. (2009) (Appendix E, Table 8). KDRI has several advantages over the currently used deceased donor classifications (i.e., ECD and SCD). First, KDRI is based on ten different donor factors,
whereas ECD/SCD classification is based on only four factors. Second, it is a continuous number which enables more detailed differentiation of donor kidney quality compared to the dichotomous ECD and SCD classification. Third, the new kidney allocation policy uses KDRI as a measure of kidney quality.

**Competition Among Transplant Centers.** We also explore the effect of competition among transplant centers within a DSA. Different OPOs have varying numbers of transplant centers within their service boundaries (DSAs); see Figure 1. Following the results in Halldorson et al. (2013), Adler et al. (2014), and Cho et al. (2015), we conjecture that the competition may play a role in the procurement decisions and should be controlled for in the regression analyses below. We use the HHI as a measure for competition among transplant centers in the DSA. We first calculate the total number of patients registered at transplant center \( c \) by blood type \( k \) \( \lambda_{ck} \) during the period of our study (January 1, 2000 through June 30, 2010). We also calculate the total number of registered patients at each DSA \( j \) for each blood type \( k \) by adding the total number of registered patients at its transplant centers \( \sum_{c \in \Omega_j} \lambda_{ck} \) where \( \Omega_j \) represents the set of transplant centers in each DSA \( j \). The market share of each transplant center by blood type is then \( s_{ck} = \frac{\lambda_{ck}}{\sum_{c \in \Omega_j} \lambda_{ck}} \) for transplant center \( c \) in DSA \( j \). The HHI is calculated as

\[
HHI_{jk} = \sum_{c \in \Omega_j} s_{ck}^2.
\]

**5.2 Econometric Method**

In this subsection, to model the event of procurement (hence, intent), we use the discrete choice model of binary probit, which specifies the probability that a person (in our case, an OPO) chooses one of two alternatives. The probability is expressed as a function of observed variables. In our model, this is the probability of procuring a donor’s kidney and the choice set is whether or not the donor’s kidney is procured.

Discrete choice models can be derived from utility maximization behavior. We represent the utility that DSA \( j \) obtains from procuring a kidney from donor \( i \) by \( U_{ij}(1) \); the utility from the decision to not procure, denoted by \( U_{ij}(0) \), equals to zero. Letting \( Y_i \) denote the procurement decision (i.e., \( Y_i = 1 \) if the kidney from donor \( i \) is procured, and \( Y_i = 0 \) otherwise), we express the utility function as follows:
\[ U_{ij}(Y_i) = \begin{cases} \beta' x_{ij} + \varepsilon_{ij}, & \text{if } Y_i = 1; \\ 0, & \text{otherwise}, \end{cases} \tag{11} \]

where \( x_{ij} \) denotes the observable variables (e.g., attributes of the kidney from donor \( i \) and attributes of DSA \( j \)), whereas \( \varepsilon_{ij} \) denotes the utility from attributes that the researcher does not observe. The vector \( \beta \) denotes the parameters to be estimated. In our analysis, \( x_{ij} = (KDRI_i, W_{jkt}, HHI_{jk})' \) where \( KDRI_i \) represents the kidney quality index of deceased donor \( i \), \( W_{jkt} \) represents the median waiting time until transplantation of all patients with the same blood type \( k \) \( (k \in \{A, B, AB, O\}) \) who had transplants at DSA \( j \) \( (j \in \{1, \ldots, 58\}) \) during each quarter \( t \) \( (t \in \{1 \ldots 42\}) \), and \( HHI_{jk} \) is the competition variable measured by HHI at DSA \( j \) for each blood type \( k \). We denote the probability that the organ from donor \( i \) in DSA \( j \) is procured by \( P_{ij} \), which is given as follows:

\[ P_{ij} = \Pr(Y_i = 1) = \Pr(U_{ij}(1) > U_{ij}(0)) = \Phi(\beta' x_{ij}) \]

where \( \Phi(\cdot) \) is the cumulative distribution function of the standard normal distribution since the probit model assumes that \( \varepsilon_{ij} \) is standard normally distributed.\(^{12}\)

One potential challenge in estimating the parameters of discrete choice models (e.g., logit or probit models) is the possibility that some component of the utility model that is presumed exogenous is in fact endogenous. In the literature, the term “endogeneity” is used to describe a model in which one (or more) unobservable variable(s) is (are) correlated with observable covariates. Failure to account for this endogeneity in an econometric model results in a violation of the independence assumption which is a necessary condition for obtaining consistent estimates. In our case, there might be unobservable DSA-specific variables which have an impact on the intent of a DSA through some observable covariates. We posit that \( KDRI_i \) cannot be an endogenous variable as the quality level of any donor’s kidney is most likely independent of DSA-specific factors. However, the two other variables, namely \( W_{jkt} \) and \( HHI_{jk} \), may be influenced by unobserved factors that affect the intent of a DSA. These unobserved factors can be financial, managerial, and cultural DSA-specific factors which can have an impact on the procurement decisions at a DSA. For instance, financial burdens can make some transplant centers (and OPOs) be more aggressive in terms of patient selection and perform transplantation for sicker patients. Being more aggressive in patient selection may have direct consequences on the waiting times of patients and the competitive environment in

\(^{12}\)The generalized bivariate Probit model only assumes that \( \varepsilon_{ij} \) follows a normal distribution, however without loss of generality we can assume standard normal distribution in our specification.
a DSA. Hence, it is not unreasonable to suspect a correlation between $W_{jkt}$ and $HHI_{jk}$ and some unobserved factors that are not captured with our data.

To correct for endogeneity, we use a control function approach. The basic idea of this approach is to construct variables (or, control functions) which would account for the nonzero part of the expected value of the error term conditional on the exogenous variables. It is basically a two-step procedure which utilizes a valid instrumental variable (IV) to control for the part of the error term that correlates with an endogenous variable. We extend the existing control function method described in Rivers and Vuong (1988) and Petrin and Train (2010) to a multivariate context in order to test whether or not our model suffers from endogeneity by exploiting the normally distributed errors in the probit estimation. We find evidence of endogeneity in our specification. The competition variable ($HHI_{jk}$) is found to be endogenous, whereas the waiting time until transplantation variable ($W_{jkt}$) turns out to be exogenous. We proceed with the outline of the estimation procedure followed by the results in Section 5.3 provided in Table 2. The details of the implementation of the endogeneity correction method (control function approach) including the results of its two steps (i.e., instrument validity and evidence of endogeneity) and the extension to a multivariate context can be found in Appendix D. We refer the reader to Petrin and Train (2010) for further details of the control function approach for endogeneity in consumer choice models.

**Endogeneity Correction Using the Control Function Approach.** The first step of the control function approach is to regress endogenous variables on exogenous instruments (Step 1). In the second step, the residuals from these regressions enter into the probit model as additional covariates (Step 2). By using two separate IVs (one for $W_{jkt}$ and one for $HHI_{jk}$), we isolate the part of $U_{ij}(Y_i)$ that is not correlated with $\varepsilon_{ij}$.

Let $\widehat{W}_{jkt}$ and $\widehat{HHI}_{jk}$ denote our IVs for the waiting time and competition variables respectively. These IVs are created to reflect the median waiting times and competition in comparable DSAs so that they share similar characteristics of the observed endogeneous variables and at the same time they even off the unobserved DSA-specific factors that might be the reason for endogeneity in the model specification. In this way, these IVs would be correlated with the original (suspected) explanatory variables (i.e., $W_{jkt}$ and $HHI_{jk}$) but not correlated with the error term. To calculate these IVs, we group all 58 DSAs in the U.S. into 8 clusters based on the average number of transplantations per year and the number of transplant centers. Hence, similar DSAs are placed in the same group, which enables us to average out the unobserved factors affecting the intent. Therefore, we define our two IVs as follows:
\( \overline{W}_{jkt} \): The average of the median waiting time at DSAs similar in size to DSA \( j \) during the same quarter \( t \) and for the same blood type \( k \).

\( \overline{HHI}_{jk} \): The average HHI at DSAs similar in size to DSA \( j \) for the same blood type \( k \).

Note that in order not to bias our Hausman-type instrument (Hausman (1996)) calculations, we excluded the DSAs in the same region.

### 5.3 Estimation Results

In this sub-section we report the main results from the endogeneity-corrected model, the details of which are provided in Appendix D.

The results of the Step 1 of the control function approach is provided in Table 6 in Appendix D. Diagnostics in Step 1 show support for the validity of the chosen instruments. Next, we test the evidence of endogeneity in Step 2 of the control function approach. The results provided in Table 7 in Appendix D indicate that the competition variable is endogenous and the waiting time until transplantation variable is exogenous. This implies that an OPO intent model that doesn’t control for the endogeneity of the competition variable leads to bias in the model estimation. Hence, in the remainder of the paper, endogeneity correction refers to the treatment of the endogenous competition variable and we assume that the waiting time until transplantation variable is an exogenous variable. In the endogeneity-corrected model, the utility function \( U_{ij}(Y_i) \) is given as follows:

\[
U_{ij}(Y_i) = \begin{cases} 
\beta' x_{ij} + (\lambda_C) \zeta_{ij} + \tilde{e}_{ij}^C, & \text{if } Y_i = 1; \\
0, & \text{otherwise,} 
\end{cases}
\]

where \( \zeta_{ij} \) is the residual from Step 1 regression (using the IV \( \overline{HHI}_{jk} \)) that is entered as an additional explanatory variable to the uncorrected utility model (11), the vector \( \beta \) and \( \lambda_C \) denote the parameters to be estimated, and \( \tilde{e}_{ij}^C \) is the utility from attributes unobservable to the researcher.

The results of this endogeneity-corrected model are displayed in Table 2. As can be seen, the kidney quality index is significant at 0.1% level, and the median waiting time until transplantation variable together with the competition variable are significant at 5% level for the whole data. The Wald \( \chi^2 \) test statistics yield a p-value less than 0.001 which indicates a significant goodness of fit for the overall model, additionally the concordance of the model is 77.8\%.

\[\text{Concordance (sometimes called the C-statistic or C-index) is a measure of goodness of fit for a binary outcomes model. It is also equal to the area under the receiver operating characteristic (ROC) curve. According to Hosmer and Lemeshow (2000), as a general rule, concordance between 70\% and 80\% is considered acceptable.}\]

Hence, we find evidence that as the organ quality increases, the probability of recovering a kidney
increases because lower KDRI values are associated with increased donor quality. Additionally, the results indicate that as the median waiting time increases, the probability of recovering a kidney from a donor increases as well. Lastly, the results give support to the positive association between competition and the probability of recovering a kidney (i.e., higher HHI means less competition). We also report the average marginal effects in Table 2. For instance, considering the whole data, a one unit increase of KDRI leads to an average decrease of 0.143 in the probability that a kidney is procured; in addition, a one year increase in the median waiting time leads to an average increase of 0.003 in the probability that a kidney is procured. The average marginal effect of the competition variable is insignificant.

Therefore, we find support for the hypotheses 1, 2, and 3a. In other words, the results support the first two hypotheses motivated by the game theoretical model and also the hypothesis inspired by the literature which find that competition leads to procuring higher levels of marginal organs (i.e., Halldorson et al. (2013), Adler et al. (2014)). Even though our intent variable is defined for all kidneys with varying qualities, our results indicate that transplant centers operating in more competitive markets have an incentive to procure lower quality kidneys. Also, note that, as will be seen below, when the data is analyzed at the lower or higher quality levels, the competition variable is no longer significant.

A wide spectrum of quality exists among deceased donor organs. We argue that the decisions for lower quality organs may be different from the decisions when all quality levels are considered. Hence, to test this, we divide the data into two groups: (i) donors whose kidney quality falls in the highest 85% quality level based on KDRI of all donors in that year, i.e., top 85% quality; and (ii) donors whose kidney quality falls in the lowest 15% quality level based on KDRI of all donors in that year, i.e., bottom 15% quality. We calculate the 85th percentile of KDRI values of all the donors for each year in our data set. Any donor whose KDRI value higher (lower) than this threshold value is classified as a bottom (top) 15% (85%) quality donor. Table 9 in Appendix E displays the different KDRI threshold values for each year.

We again estimate the coefficients and average marginal effects using our endogeneity-corrected intent model specification as described above and compare the results of these mutually exclusive

---

14 In addition, we divide the data into different groups by blood type and estimate the coefficients in the endogeneity-corrected model. The results of these estimation models are available in Appendix E.

15 The analysis of the Kidney Transplantation Committee shows that graft survival rate degrades significantly faster after this cut-off, and hence, 85% is a natural choice; see the figure on slide 16 of the Proposal to Substantially Revise the National Kidney Allocation System Document, available at https://www.transplantpro.org/wp-content/uploads/sites/3/Board_06-2013_Kidney_Committee_Actions1.pdf, accessed on May 1, 2017.
### Table 2: Summary of Estimation Models (Intent Model with Whole Data, Bottom 15%, and Top 85% Quality Donors.)

<table>
<thead>
<tr>
<th>Variable (Coefficient)</th>
<th>Whole Data</th>
<th>Bottom 15%</th>
<th>Top 85%</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Parameter Estimate</td>
<td>Average Marginal Effects</td>
<td>Parameter Estimate</td>
</tr>
<tr>
<td>Constant ($\beta_0$)</td>
<td>2.791***</td>
<td>(0.030)</td>
<td>2.761***</td>
</tr>
<tr>
<td>$KDRI_i$ ($\beta_{KDRI}$)</td>
<td>-0.989***</td>
<td>(0.012)</td>
<td>-0.971***</td>
</tr>
<tr>
<td>$W_{jk}t$ ($\beta_W$)</td>
<td>0.016*</td>
<td>(0.008)</td>
<td>0.039**</td>
</tr>
<tr>
<td>$HHI_{jk}$ ($\beta_{HHI}$)</td>
<td>-0.101*</td>
<td>(0.049)</td>
<td>-0.160</td>
</tr>
<tr>
<td>Log-likelihood</td>
<td>-17,917.61</td>
<td></td>
<td>-5,539.20</td>
</tr>
<tr>
<td>Concordance</td>
<td>77.8%</td>
<td></td>
<td>67.5%</td>
</tr>
<tr>
<td>Wald $\chi^2(3)$</td>
<td>7,181.67***</td>
<td></td>
<td>771.96***</td>
</tr>
<tr>
<td>Observations</td>
<td>75,778</td>
<td>75,778</td>
<td>11,363</td>
</tr>
</tbody>
</table>

Note 1: Bootstrap standard errors for parameter estimates are in parenthesis.
Note 2: Delta-method standard errors for average marginal effects are in parenthesis.

*** $p < 0.001$, ** $p < 0.01$, * $p < 0.05$.

The impact of this new kidney allocation policy is studied in the next section (Section 6). It is interesting to note that even though the estimated coefficients indicate the opposite ($|\hat{\beta}_{KDRI}^{top 85\%}| > |\hat{\beta}_{KDRI}^{bottom 15\%}|$), the average marginal effects show that the intent for low quality organs is more sensitive to the changes in the quality level of the organ: a one unit decrease in the KDRI (increase in quality) leads to an average increase of 31% in the probability of recovery for low quality organs, whereas a one unit decrease in the KDRI results in an average increase of 13% in the probability of recovery for high quality organs.
covariate changes by 50%. Based on the estimated coefficients available in Table 2, for a typical lower quality donor (bottom 15% quality) who has the average values of all three variables (i.e., $KDRI_i = 2.27$, $W_{jkt} = 1.83$, and $HHI_{jk} = 0.39$): (i) a 50% increase in the donor quality (50% reduction in the $KDRI_i$) would result in a 15.1% increase in the probability of recovering his/her kidney; and (ii) a 50% increase in the waiting time would result in a 1.2% increase in the probability of recovering his/her kidney. Similarly, for a typical higher quality donor (top 85% quality) who has the average values of all three variables (i.e., $KDRI_i = 1.17$, $W_{jkt} = 1.79$, and $HHI_{jk} = 0.41$): a 50% increase in the donor quality (50% reduction in the $KDRI_i$) would result in a 4.2% increase in the probability of recovering his/her kidney. Both the parameter estimate and the average marginal effect of the waiting time for the top 85% quality sample is insignificant, hence we do not report its marginal effects. Overall, we conclude that the intent for low quality organs is more sensitive to the changes of organ quality than the intent for higher quality organs since the marginal effect of varying the kidney quality by 50% is 15.1% for bottom 15% quality sample and 4.2% for the top 85% quality sample.

6 Counterfactual Analysis

The procurement rates of the deceased-donor kidneys exhibit significant variation across different DSAs. This is illustrated in Figure 7 for the NYRT OPO in New York versus UTOP OPO in Utah. In particular, UTOP OPO procures better quality organs than NYRT OPO. To be more specific, the lowest quality organ procured in Utah has KDRI of 2.4; and the organs of lower quality (i.e. higher KDRI) are not procured in Utah whereas such organs are procured in New York, as shown in Figure 7. Therefore, a natural conclusion is that if such organs became available in New York, they could have been procured, increasing the organ supply. Although Figure 7 shows just one pair of OPOs, such disparities are widespread and hence, the opportunity to better utilize lower quality kidneys is likewise widespread in the deceased-donor kidney allocation system. As a matter of fact, as part of the new kidney allocation system, which became effective in December 2014, lower quality kidneys are shared more broadly. That is, instead of following the current geographically-tiered protocol of sharing (i.e. a kidney is first offered in its DSA, then in its region, and then nationally), the new policy allows those kidneys to be offered directly in their regions followed by the entire country. One can also consider offering the lower quality kidneys nationally without offering them regionally first.
The specific change in the kidney allocation policy is to share the bottom 15% quality of the organs more broadly (i.e., regionally). This section focuses on the impact of this policy change and quantifies its potential benefits. We also study the impact of sharing the low quality organs nationally and increasing the low quality threshold to 20% from 15%.

We have a total of 11,363 observations in the lowest 15% quality in our data set. We run the endogeneity-corrected probit regression described in Section 5 with the same variables over the sample of the bottom 15% quality kidneys, separately for each blood type. Hence, we obtain the estimated coefficients \( \hat{\beta}_{\text{bottom } 15\%}^{0,k}, \hat{\beta}_{\text{bottom } 15\%}^{\text{KDRI},k}, \hat{\beta}_{\text{bottom } 15\%}^{W,k}, \text{ and } \hat{\beta}_{\text{bottom } 15\%}^{\text{HHI},k} \) for each blood type \( k \).

To estimate the additional number of kidneys procured per year, in our regional sharing analysis, we first substitute the largest median waiting time in donor \( i \)'s region \( r \) for blood type \( k \) in quarter \( t \) (\( W_{\text{Max}}^{rkt} \)) as the median waiting time of the donor \( i \). Then, for each donor \( i \) and blood type \( k \), we calculate the estimated probability of procurement using the following probit model:

\[
\hat{Y}_{i,k}^{\text{Reg}} = \Phi(\hat{\beta}_{\text{bottom } 15\%}^{0,k} + \hat{\beta}_{\text{bottom } 15\%}^{\text{KDRI},k} * KDRI_{i} + \hat{\beta}_{\text{bottom } 15\%}^{W,k} * W_{\text{Max}}^{rkt} + \hat{\beta}_{\text{bottom } 15\%}^{\text{HHI},k} * HHI_{jk}).
\]

Therefore, we find the total additional number of kidneys procured per year with regional sharing as follows:

\[
\sum_{k \in \{A, B, O, AB\}} \left( \frac{2 * \sum_{i=1}^{n_k} \hat{Y}_{i,k}^{\text{Reg}} - AP_k}{42} * 4 \right),
\]

\footnote{Whenever a donor has a KDRI value higher than the 85th percentile of KDRI values of all donors in the observed year (displayed in Appendix E Table 9), the organ recovered from this donor belongs to the bottom 15% quality.}
where $AP_k$ is the actual number of blood type $k$ kidneys procured among low quality donors (bottom 15%) during the period of our study, $n_k$ is the number of low quality donors for blood type $k$. Note that we multiply $\sum_{i=1}^{n_k} \hat{Y}_{i,k}^{\text{Reg}}$ by two since our unit of analysis is a kidney and whenever there is a procurement, two kidneys are procured; in addition, we have 42 quarters during the period of our study. The additional number of kidneys procured per year for each blood type under regional sharing is reported in the third column of Table 3.

We also conduct an analogous study under national sharing. As indicated above, national sharing is not considered in the new kidney allocation system. However, this counterfactual analysis calculates the expected number of additional kidneys which may arise due to a national sharing policy and compares its impact to that of the current regional sharing policy. To estimate the additional number of kidneys procured per year, in our national sharing analysis, we first substitute the largest median waiting time (in the whole nation) for blood type $k$ in quarter $t$ ($W_{k,t}^{\text{Max}}$) as the median waiting time of the donor $i$. Then, similar to the regional sharing estimation above, we calculate the estimated probability of procurement $\hat{Y}_{i,k}^{\text{Nat}}$ for each donor $i$ with blood type $k$ using the probit model. Therefore, the additional number of kidneys procured per year with national sharing is

$$\sum_{k \in \{A,B,O,AB\}} \left( \frac{2 \sum_{i=1}^{n_k} \hat{Y}_{i,k}^{\text{Nat}} - AP_k}{42} \right) \times 4.$$  

The additional number of kidneys procured per year for each blood type under national sharing is reported in the last column of Table 3. We also repeat this analysis using more recent 5 years data for which we run the endogeneity-corrected regressions over the period between 2006 and 2010. The last five rows of Table 3 show the results when the last five years is considered in the estimations. Note that in addition to the point estimates, we report the 95% confidence intervals.\footnote{Confidence intervals are displayed in brackets. These intervals are obtained through a bootstrap approach. In order to obtain the standard errors for the mean value of the total estimated number of kidneys procured, we draw a random sample of size equal to the number of observations for each blood type with replacement (i.e., full sample) where each observation $i$ is sampled subjected to an independent Bernoulli trial with parameter $\hat{Y}_i$ (estimated probability of procurement). Then, the confidence intervals are calculated by finding the mean ($\hat{\mu}$) of the samples and standard errors of the means (SE) for 1,000 bootstrap replications for each blood type (separately for regional and national sharing). We obtain repeated samples to calculate the upper ($\hat{\mu} + 1.96 \times \text{SE}$) and lower ($\hat{\mu} - 1.96 \times \text{SE}$) end points of the 95% confidence intervals. The values above the confidence intervals (i.e., point estimates) in Table 3 are calculated directly from the estimated coefficients, so they are not the midpoints of the simulated confidence intervals.}

The additional number of kidneys procured vary by the blood type – highest for blood types O and A. In addition, the gain from this policy change is more conspicuous in more recent years.
Moreover, the national sharing leads to significantly higher gains over regional sharing although both lead to significant increases in the supply of deceased-donor organs.

<table>
<thead>
<tr>
<th></th>
<th>Regional Sharing</th>
<th>National Sharing</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Number of Additional Kidneys Procured Per Year</td>
<td>Number of Additional Kidneys Procured Per Year</td>
</tr>
<tr>
<td></td>
<td>[95% Confidence Interval]</td>
<td>[95% Confidence Interval]</td>
</tr>
<tr>
<td>2000-2010</td>
<td></td>
<td></td>
</tr>
<tr>
<td>A</td>
<td>7.70</td>
<td>19.07</td>
</tr>
<tr>
<td></td>
<td>[7.14; 7.79]</td>
<td>[18.73; 19.36]</td>
</tr>
<tr>
<td>AB</td>
<td>*</td>
<td>*</td>
</tr>
<tr>
<td>B</td>
<td>3.24</td>
<td>6.72</td>
</tr>
<tr>
<td></td>
<td>[3.10; 3.45]</td>
<td>[6.50; 6.85]</td>
</tr>
<tr>
<td>O</td>
<td>28.70</td>
<td>63.07</td>
</tr>
<tr>
<td></td>
<td>[28.63; 29.38]</td>
<td>[62.47; 63.47]</td>
</tr>
<tr>
<td>Total</td>
<td><strong>39.64</strong></td>
<td><strong>88.86</strong></td>
</tr>
<tr>
<td></td>
<td>[39.24; 40.25]</td>
<td>[88.32; 89.32]</td>
</tr>
<tr>
<td>2006-2010</td>
<td></td>
<td></td>
</tr>
<tr>
<td>A</td>
<td>23.16</td>
<td>57.82</td>
</tr>
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<td></td>
<td>[22.56; 23.60]</td>
<td>[56.82; 57.81]</td>
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<tr>
<td>AB</td>
<td>*</td>
<td>*</td>
</tr>
<tr>
<td>B</td>
<td>5.07</td>
<td>11.56</td>
</tr>
<tr>
<td></td>
<td>[5.10; 3.45]</td>
<td>[11.08; 11.65]</td>
</tr>
<tr>
<td>O</td>
<td>29.38</td>
<td>59.51</td>
</tr>
<tr>
<td></td>
<td>[28.84; 30.10]</td>
<td>[58.38; 59.58]</td>
</tr>
<tr>
<td>Total</td>
<td><strong>57.60</strong></td>
<td><strong>128.89</strong></td>
</tr>
<tr>
<td></td>
<td>[56.66; 58.43]</td>
<td>[126.85; 128.47]</td>
</tr>
</tbody>
</table>

*: Insufficient sample size for blood type AB to obtain reasonable estimates.

**Table 3:** Number of Additional Kidneys Procured Per Year for the Bottom 15% Threshold under Regional and National Sharing

<table>
<thead>
<tr>
<th></th>
<th>Regional Sharing</th>
<th>National Sharing</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Number of Additional Kidneys Procured Per Year</td>
<td>Number of Additional Kidneys Procured Per Year</td>
</tr>
<tr>
<td></td>
<td>[95% Confidence Interval]</td>
<td>[95% Confidence Interval]</td>
</tr>
<tr>
<td>2006-2010</td>
<td></td>
<td></td>
</tr>
<tr>
<td>A</td>
<td>42.14</td>
<td>95.86</td>
</tr>
<tr>
<td></td>
<td>[41.00; 41.16]</td>
<td>[95.68; 96.72]</td>
</tr>
<tr>
<td>B</td>
<td>6.09</td>
<td>16.76</td>
</tr>
<tr>
<td></td>
<td>[5.86; 6.51]</td>
<td>[16.56; 17.20]</td>
</tr>
<tr>
<td>O</td>
<td>30.84</td>
<td>61.73</td>
</tr>
<tr>
<td></td>
<td>[29.93; 31.31]</td>
<td>[60.25; 61.86]</td>
</tr>
<tr>
<td>Total</td>
<td><strong>79.07</strong></td>
<td><strong>174.35</strong></td>
</tr>
<tr>
<td></td>
<td>[77.41; 79.35]</td>
<td>[173.08; 174.89]</td>
</tr>
</tbody>
</table>

**Table 4:** Number of Additional Kidneys Procured Per Year for the Bottom 20% Threshold under Regional and National Sharing

The rest of this section quantifies the impact of adjusting the quality threshold from the lowest 15% (see Table 3) to the lowest 20% (see Table 4). We repeat the analysis of the bottom 15% quality kidneys using expanded data including all the donors with the lowest 20% quality kidneys. Increasing the quality threshold increases the additional number of kidneys procured for all blood types: the total number of kidneys increases from 58 to 79 under regional sharing and from 129
to 174 under national sharing (these numbers apply to the most recent five years in the sample, 2006-2010). Similar to the results in Table 3, as seen in Table 4, national sharing results in a greater increase of procured kidneys than regional sharing, although the increase of 95 kidneys per year (79 to 174) under 20% is larger than the increase of 71 kidneys (58 to 129) under 15%. The effect of blood type under the policy change is different, which might suggest a more tailored policy change could enhance the total increase. Blood types A and B enjoy a greater increase of procured kidneys when moving from 15% to 20% than type O. Hence, a possible policy adjustment could be that the lowest 20% quality of blood types A and B kidneys be shared more broadly while retaining 15% for blood type O.

We list the percentage supply increases in Table 5. We include the percentage of all procured kidneys and the percentage of the lowest 15% quality (in the time period 2006-2010). While these percentage increases may appear to be modest, they provide a perspective on how the supply (both total and lower quality sources) are affected by the policy changes. We can see that the broader sharing of the lowest 15% quality kidneys nationally increases the supply of all procured kidneys by 0.9% (1.2% for lowest 20% quality kidneys). This represents an increase of 7.3% of these lowest quality kidneys (9.9% for the lowest 20% quality kidneys).

To put the increase of 129 procured kidneys per year (under national sharing of the lowest 15% quality) in perspective, there are 14 of the 58 DSAs in the U.S. in 2009 with 129 or fewer kidneys available. Therefore, this expected increase of 129 reflects the addition of a small- to medium-sized DSA to the UNOS network.

<table>
<thead>
<tr>
<th></th>
<th>Increase with respect to total number of kidneys procured per year on average between 2006-2010 (N=15,030)</th>
<th>Increase with respect to bottom 15% quality kidneys procured per year on average between 2006-2010 (N=1,764)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bottom 15% regional sharing</td>
<td>58/15,030= 0.4%</td>
<td>58/1,764= 3.3%</td>
</tr>
<tr>
<td>Bottom 15% national sharing</td>
<td>129/15,030= 0.9%</td>
<td>129/1,764= 7.3%</td>
</tr>
<tr>
<td>Bottom 20% regional sharing</td>
<td>79/15,030= 0.5%</td>
<td>79/1,764= 4.5%</td>
</tr>
<tr>
<td>Bottom 20% national sharing</td>
<td>174/15,030= 1.2%</td>
<td>174/1,764= 9.9%</td>
</tr>
</tbody>
</table>

Table 5: Total Number of Additional Kidneys Procured under Regional and National Sharing with Respect to Annual Average Volumes (2006-2010 Data)
7 Concluding Remarks

We study how the deceased-donor kidney supply can be enhanced through geographic sharing in the U.S. In particular, we examine how demand side pressure affects the supply of deceased-donor organs for transplant. We formulate and test hypotheses to glean the impact of organ quality, waiting time, and competition on organ procurement decisions, i.e., the intent. Most importantly, we find that broader sharing of low quality kidneys can lead to a significant increase in the organ supply. To be more specific, both higher organ quality and longer waiting times in a DSA generate greater OPO intent. These characteristics differ markedly across the country. These disparities endow the system with an opportunity to procure more organs if some organs are shared more broadly immediately. Following such a policy nationally (i.e., sharing lowest 15% quality kidneys nationally) is expected to yield 129 additional kidney transplants per year, a number expected to increase as the difference between supply and demand for organs grows. This number reflects an increase of around 1% of all procured kidneys per year on average between 2006 and 2010, and 7.3% of the bottom 15% quality kidneys procured annually on average between 2006 and 2010. Moreover, there are 14 of the 58 DSAs in the United States in 2009 with 129 or fewer kidneys available. Therefore, this expected increase of 129 kidneys reflects the addition of a small- to medium-sized DSA.

References


Appendices for
“Enhancing Kidney Supply Through Geographic Sharing in the United States”

Appendix A: Proofs

Proof of Proposition 1. It follows from (2)-(3) that

\[ Q(t) = \exp \left\{ -\int_0^t \gamma(s) ds \right\} \left[ \lambda - \int_0^t \exp \left\{ \int_0^s \gamma(u) du \right\} G'(l(s)) l'(s) ds \right], \quad t < \tau. \]  

(16)

Then combining (4) and (16), we see that \( \tau \) must satisfy

\[ \exp \left\{ \int_0^\tau \gamma(s) ds \right\} \Delta G(\bar{L}) + \int_0^\tau \exp \left\{ \int_0^s \gamma(u) du \right\} G'(l(s)) l'(s) ds = \lambda. \]  

(17)

At equilibrium a patient who has waited for \( t \) time units must be indifferent between accepting an organ of life years \( l(t) \) and waiting. That is, we must have

\[ l(t) = W_t + L_t \quad \text{for} \quad t < \tau, \quad l(t) > L, \]  

(18)

where \( W_t \) denotes the expected residual waiting time conditional on having waited for \( t \) time units, and \( L_t \) denotes the expected post-transplant life expectancy associated with waiting (not including the waiting time on the transplant list) of a patient who has waited for \( t \) time units.

The following figure shows the various events (and their rates) that can happen to patients who have waited for \( t \) time units:

![Figure 8](image)

**Figure 8:** The portion of the transplant waiting list consisting of patients who have waited for \( t \) time units or more. Viewing this as a system, patients enter at the rate of \( Q(t) \), and can leave the system at time \( s \in [t, \tau) \) with rate \( G'(l(s)) l'(s) \) and at time \( \tau \) with rate \( \Delta G(\bar{L}) \) by receiving a transplant. Patients can also leave the system by dying at rate \( \gamma Q(s) \) (at time \( s \)).

Given the system portrayed in Figure 8, we write by Little’s Law that

\[ W_t = \frac{\int_0^t Q(s) ds}{Q(t)}. \]  

(19)

To compute \( L_t \), consider what happens to the intensity of fluid \( Q(t) \) (of those who have been waiting for \( t \) time units) as shown in Figure 8, and interpret the fraction served at various times as the
probability density (or mass at time $\tau$) of getting transplanted after waiting for $s \geq t$ time units, denoted by $\phi(s)$. Note that

$$\phi(s) = \frac{G'(l(s)) l'(s)}{Q(t)} \quad \text{for } t \leq s < \tau \quad \text{and } \phi(\tau) = \frac{\Delta G(\bar{L})}{Q(t)}.$$  (20)

Then, we write

$$L_t = \int_t^\tau \phi(s)l(s)ds + \phi(\tau)\bar{L}.$$  (21)

Substituting (20) into (21) yields

$$L_t = \int_t^\tau l(s) \frac{G'(l(s)) l'(s)}{Q(t)} ds + \frac{\Delta G(\bar{L})}{Q(t)} \bar{L}.$$  

Equivalently,

$$L_t = \frac{1}{Q(t)} \int_{l(t)}^{\bar{L}} udG(u).$$  (22)

Substituting (19) and (22) into (18) gives

$$\int_t^\tau Q(s)ds + \int_{l(t)}^{\bar{L}} udG(u) = Q(t)l(t) \quad \text{for } t < \tau, \ l(t) > \bar{L}. $$  (23)

In what follows, we will first ignore the restriction $l(t) > \bar{L}$ in (23) and solve for $f(\cdot)$ that solves (23). Then, we will observe that $f(\cdot)$ is strictly increasing. Therefore, truncating $f(\cdot)$ at $\bar{L}$ from below yields $l(\cdot)$. To this end, consider the equation

$$\int_t^\tau Q(s)ds + \int_{f(t)}^{\bar{L}} udG(u) = Q(t)f(t) \quad \text{for } t < \tau.$$

Differentiating both sides with respect to $t$ and substituting for $Q'(t)$ (cf. Equation (3)) gives

$$-1 = -\gamma(t)f(t) + f'(t) \quad \text{for } t < \tau.$$  (24)

Also, using the boundary condition that $f(\tau) = l(\tau) = \bar{L}$ gives

$$f(t) = \exp \left\{ \int_0^t \gamma(s)ds \right\} \left[ \bar{L} \exp \left\{- \int_0^t \gamma(s)ds \right\} + \int_t^\tau \exp \left\{- \int_s^t \gamma(u)du \right\} ds \right], \quad \text{for } 0 < t < \tau.$$  (25)

Then, the patients’ strategy is really the truncated function:

$$l(t) = \max\{l, f(t)\}$$  (26)

which proves (5). Also note that (16) proves (4). To prove (6), we first consider how $f(t)$ and $f'(t)$ change with $\tau$. Note that

$$\frac{\partial f(t)}{\partial \tau} = -(\bar{L}\gamma(\tau) - 1) \exp \left\{- \int_t^\tau \gamma(s)ds \right\} < 0, \ t < \tau,$$  (27)
which incidentally proves the last assertion of the proposition. Also note from (24) that

\[
\frac{\partial f'(t)}{\partial \tau} = \gamma(t) \frac{\partial f(t)}{\partial \tau} < 0. \tag{28}
\]

To establish the uniqueness of \( \tau \) satisfying (6), consider (17) and define \( H(\tau) \) as its left-hand side, i.e.,

\[
H(\tau) = \exp \left\{ \int_0^\tau \gamma(s)ds \right\} \Delta G(\bar{L}) + \int_0^\tau \exp \left\{ \int_0^s \gamma(u)du \right\} G'(l(s))l'(s)ds. \tag{29}
\]

Differentiating this, substituting \( l'(\tau) = \bar{L} \gamma(\tau) - 1 \) from (24), and rearranging terms gives

\[
H'(\tau) = \exp \left\{ \int_0^\tau \gamma(s)ds \right\} \left[ \gamma(\tau) \Delta G(\bar{L}) + G'(\bar{L})(\bar{L} \gamma(\tau) - 1) \right] \\
+ \int_0^\tau \exp \left\{ \int_0^s \gamma(u)du \right\} \frac{\partial}{\partial \tau} \left[ \frac{dl}{ds} G(l(s)) \right] ds. \tag{30}
\]

Changing the order of differentiation for the integrand of the last term on the right-hand side yields

\[
H'(\tau) = \exp \left\{ \int_0^\tau \gamma(s)ds \right\} \left[ \gamma(\tau) \Delta G(\bar{L}) + G'(\bar{L})(\bar{L} \gamma(\tau) - 1) \right] \\
+ \int_0^\tau \exp \left\{ \int_0^s \gamma(u)du \right\} \frac{dl}{ds} \left[ \frac{dl}{ds} G(l(s)) \right] ds. \tag{31}
\]

Note that

\[
\frac{\partial}{\partial \tau} G(l(s)) = G'(l(s)) \frac{dl}{\partial \tau}. \]

Then, note from (27) that

\[
\frac{\partial}{\partial \tau} G(l(s)) = -G'(l(s))(\bar{L} \gamma(\tau) - 1) \exp \left\{ -\int_s^\tau \gamma(u)du \right\}. 
\]

Thus, we conclude that

\[
H'(\tau) = \exp \left\{ \int_0^\tau \gamma(s)ds \right\} \left[ \gamma(\tau) \Delta G(\bar{L}) + G'(\bar{L})(\bar{L} \gamma(\tau) - 1) \right] \\
- \int_0^\tau \exp \left\{ \int_0^s \gamma(u)du \right\} \frac{dl}{ds} \left[ G'(l(s)) \right] ds \tag{32}
\]

Integrating the last term on the right-hand side by parts gives

\[
H'(\tau) = \exp \left\{ \int_0^\tau \gamma(s)ds \right\} \left[ \gamma(\tau) \Delta G(\bar{L}) + G'(\bar{L})(\bar{L} \gamma(\tau) - 1) \right] \\
- \exp \left\{ \int_0^\tau \gamma(u)du \right\} G'(l(s))(\bar{L} \gamma(\tau) - 1) \exp \left\{ -\int_s^\tau \gamma(u)du \right\} \bigg|_0^\tau \\
+ \int_0^\tau \gamma(s) \exp \left\{ \int_0^s \gamma(u)du \right\} G'(l(s))(\bar{L} \gamma(\tau) - 1) \exp \left\{ -\int_s^\tau \gamma(u)du \right\} ds \\
= \exp \left\{ \int_0^\tau \gamma(s)ds \right\} \left[ \gamma(\tau) \Delta G(\bar{L}) + G'(\bar{L})(\bar{L} \gamma(\tau) - 1) \right] \\
- \exp \left\{ \int_0^\tau \gamma(u)du \right\} G'(l(0))(\bar{L} \gamma(\tau) - 1) \\
+ G'(l(0))(\bar{L} \gamma(\tau) - 1) \exp \left\{ -\int_0^\tau \gamma(u)du \right\}
\]
\[ + \int_{s}^{\tau} \gamma(s) \exp \left\{ \int_{0}^{s} \gamma(u) du \right\} G'(l(s))(L\gamma(\tau) - 1) \exp \left\{ - \int_{s}^{\tau} \gamma(u) du \right\} ds \]
\[
> \exp \left\{ \int_{0}^{\tau} \gamma(s) ds \right\} \left[ \gamma(\tau) \Delta G(L) + G'(L)(L\gamma(\tau) - 1) - G'(L)(L\gamma(\tau) - 1) \right] \]
\[ = \exp \left\{ \int_{0}^{\tau} \gamma(s) ds \right\} \gamma(\tau) \Delta G(L) > 0.\]

Therefore, \( H(\cdot) \) is strictly increasing. Also note that \( H(0) = \Delta G(L) \) and \( \lim_{\tau \to \infty} H(\tau) = \infty. \)

Thus, for every \( \lambda > \Delta G(L) \), there exists a unique \( \tau \) such that (6) holds. It is also immediate from the monotonicity of \( H(\cdot) \) that as \( \lambda \) increases, so does \( \tau \), which concludes the proof. \[ \square \]

**Proof of Corollary 1.** (8) follows from (5) by direct substitution of \( \gamma(t) = \gamma \) for all \( t \). Note also that equation (17) in the Proof of Proposition 1, which pins down \( \tau \), becomes

\[ e^{\gamma \tau} \Delta G(L) + \int_{0}^{\tau} e^{\gamma s} G'(l(s)) l'(s) ds = \lambda. \] (33)

Then substituting (8) into (33) and making the change of variable \( u = l(s) \) gives

\[ e^{\gamma \tau} \int_{l(0)}^{L} \left( u - \frac{1}{\gamma} \right) dG(u) = \lambda \left( L - \frac{1}{\gamma} \right). \]

Similarly, substituting \( \gamma(t) = \gamma \) for all \( t \) in (16) gives

\[ Q(t) = e^{\gamma t} \left[ \lambda - \int_{0}^{t} e^{\gamma s} G'(l(s)) l'(s) ds \right], t < \tau. \] (34)

Then substituting (8) into (34) and making the change of variable \( u = l(s) \) gives (10), concluding the proof. \[ \square \]

**Appendix B: Details of the Calculation of Variables**

**Waiting Time to Transplantation in a DSA.** Although the donor and recipient data sets do not directly include this kind of information, we calculate this variable by using three variables in the recipient data: **init_date**, **end_date**, and **trr_id_code**. The first two variables represent the date on which the patient is added to the waiting list and on which the patient is removed from the waiting list, respectively. The third variable is the transplant identifier which is only non-missing if a transplantation has occurred.

The **end_date** variable can represent the transplantation date or the date a patient is removed from the waiting list due to other reasons (e.g., death). By using the **trr_id_code**, we can identify all patients who had a transplantation in our data. Hence, we first group the data by each DSA during each quarter by blood type. Then, we calculate the waiting time of each patient who had a transplantation in the recipient data by finding the time difference between his/her **end_date** and **init_date** if this patient was added to the waiting list prior to the beginning of or within the observed quarter and was removed from the list before the last day of the quarter. Finally, the median value (in terms of years) of this variable is calculated for all observations grouped by DSA, blood type, and quarter.
Kidney Donor Risk Index. This index combines a variety of donor factors into a single continuous scale that captures the risk of graft failure after kidney transplantation. There are 10 factors considered in calculating the KDRI. These factors are donor age, height, weight, ethnicity, history of hypertension, diabetes status, serum creatinine level, cause of death, Hepatitis C Virus status, and DCD (donation after circulatory death) status. Note that the lower the KDRI of a donor, the higher is the donor kidney quality. This index was first developed by Rao et al. (2009) by estimating the association between these 10 donor factors and graft survival by using multivariable Cox proportional hazards regression model. The donor characteristics and their estimated coefficients are provided in Table 8 in Appendix E. There is another index called Kidney Donor Profile Index (KDPI) which is a mapping of the KDRI based on the profiles of all deceased donors in the U.S. from whom a kidney was recovered during the prior calendar year. In this study, instead of using this type of mapping we calculate the KDRI value for each donor in our data set and use this variable in the regressions as a proxy for donor kidney quality.

Herfindahl-Hirschman Index. From our recipient data set, we first calculate the total cumulative number of registered patients during our period of study (January 1, 2000 through June 30, 2010) at each transplant center by using the variable init_date which indicates the date when a patient is added to the waiting list. Next, we calculate the total cumulative number of registered patients at an OPO by adding the total cumulative number of registered patients at all transplant centers that belong to the observed OPO. Note that we use an HHI for each OPO for each blood type separately. Let $\lambda_{ck}$ represent the total number of patients registered at transplant center $c$ by blood type $k$, then the total number of registered patients at each DSA $j$ for each blood type $k$ equals $\sum_{c \in \Omega_j} \lambda_{ck}$ where $\Omega_j$ represents the set of transplant centers in each DSA $j$. The market share of each transplant center $c$ by blood type $k$ then equals to $s_{ck} = \frac{\lambda_{ck}}{\sum_{c \in \Omega_j} \lambda_{ck}}$ for transplant center $c$ in DSA $j$. The Herfindahl-Hirschman Index is calculated as $\sum_{c \in \Omega_j} s_{ck}^2$.

Appendix C: An Additional Figure

Figure 9 indicates that the average HHI of all OPOs by year doesn’t change significantly over time, but there is heterogeneity across different blood types; i.e., the market seems to be more competitive for blood types A and O; and slightly less competitive for blood types B and AB.
Appendix D: Endogeneity Correction Method

Two-stage least squares (2SLS) is the common method for testing and eliminating endogeneity in linear models. However, it cannot be easily extended to non-linear models like ours. The control function approach (Rivers and Vuong (1988), Petrin and Train (2010)) is better suited to nonlinear models with continuous variables. The control function approach can be thought of as a two-step procedure to deal with the issue of endogeneity in econometric models. To illustrate the intuition behind this approach, consider a valid instrumental variable (IV), $z$, and assume only one endogenous variable, $x$ in the model specification. This instrumental variable has to be correlated with the endogenous variable while it should not be correlated with the error term. The control function simply correspond to the estimated residuals of the regression of $x$ on $z$. Then, since the instrumental variable is not correlated with the original error term, the control function captures the part of $x$ which is correlated with the error in the original model and therefore serves as a control for it.

For the first step, we assume the following functional forms:

$$W_{jk} = \omega_i q_{ij} + \eta_{ij}, \quad (35)$$

$$HHI_{jk} = \psi_i q_{ij} + \zeta_{ij}, \quad (36)$$

where $q_{ij} = (KDRI_i, \tilde{W}_{jk}, \tilde{HHI}_{jk})$. The vectors $\omega$ and $\psi$ denote the corresponding parameters to be estimated; and $\eta_{ij}$ and $\zeta_{ij}$ are the error terms.

The control function approach further assumes that $\varepsilon_{ij}$, $\eta_{ij}$, and $\zeta_{ij}$ are one-on-one independent of $KDRI_i$, $\tilde{W}_{jk}$, and $\tilde{HHI}_{jk}$. However, $\varepsilon_{ij}$ and $\eta_{ij}$ (also, $\varepsilon_{ij}$ and $\zeta_{ij}$) are allowed to be correlated. Consider now the distribution of $\varepsilon_{ij}$ conditional on $\eta_{ij}$. We can decompose $\varepsilon_{ij}$ into its mean, conditional on $\eta_{ij}$ as follows:

$$\varepsilon_{ij} = E[\varepsilon_{ij} | \eta_{ij}] + \varepsilon_{ij}^W, \quad (37)$$

where $\varepsilon_{ij}^W$ is the error term and by construction $E[\varepsilon_{ij}^W | \eta_{ij}] = E[\varepsilon_{ij}^W | \eta_{ij}] = 0$. The conditional expectation in (37) is called the control function used for the variable $W_{jk}$ for which we assume a linear functional form. Hence, the control function is a function of $\eta_{ij}$ and is denoted by $\text{CF}_1(\eta_{ij}; \lambda_W) = \lambda_W \eta_{ij}$ where $\lambda_W$ is a coefficient term to be estimated.\(^{18}\)

Consider now the distribution of $\varepsilon_{ij}$ conditional on $\zeta_{ij}$. Similarly, we can decompose $\varepsilon_{ij}$ into its mean, conditional on $\zeta_{ij}$ as follows:

$$\varepsilon_{ij} = E[\varepsilon_{ij} | \zeta_{ij}] + \varepsilon_{ij}^C, \quad (38)$$

where $\varepsilon_{ij}^C$ is the error term and by construction $E[\varepsilon_{ij}^C | \zeta_{ij}] = E[\varepsilon_{ij}^C | \zeta_{ij}] = 0$. The control function (i.e., the conditional expectation in (38)) is a function of $\zeta_{ij}$ and is denoted by $\text{CF}_2(\zeta_{ij}; \lambda_C) = \lambda_C \zeta_{ij}$ where $\lambda_C$ is a coefficient term to be estimated.\(^{19}\) Therefore, since $\varepsilon_{ij} = \text{CF}_1(\eta_{ij}; \lambda_W) + \varepsilon_{ij}^W = \text{CF}_2(\zeta_{ij}; \lambda_C) + \varepsilon_{ij}^C$, 

$$\varepsilon_{ij} = \left(\frac{\lambda_W}{2}\right) \eta_{ij} + \left(\frac{\lambda_C}{2}\right) \zeta_{ij} + \left(\frac{\varepsilon_{ij}^W + \varepsilon_{ij}^C}{2}\right).$$

\(^{18}\)Assuming $\varepsilon_{ij}$ and $\eta_{ij}$ are jointly normal with zero mean, by the properties of the multivariate distribution, $\varepsilon_{ij}^W = \varepsilon_{ij} - \lambda_W \eta_{ij}$ is also normally distributed. Note that $E[\varepsilon_{ij} | \eta_{ij}] = \left(\frac{\text{Cov}(\varepsilon_{ij}, \eta_{ij})}{\text{Var}(\eta_{ij})}\right) \times n$, and hence $\lambda_W$ reflects a covariance term.

\(^{19}\)Assuming $\varepsilon_{ij}$ and $\zeta_{ij}$ are jointly normal with zero mean, by the properties of the multivariate normal distribution, $\varepsilon_{ij}^C = \varepsilon_{ij} - \lambda_C \zeta_{ij}$ is also normally distributed.
Then, the utility function $U_{ij}(Y_i)$ becomes

$$U_{ij}(Y_i) = \begin{cases} 
\beta'x_{ij} + \left( \frac{\lambda W_{ij} + \lambda C_{ij}}{2} \right) + \left( \frac{\hat{e}_{ij}^W + \hat{e}_{ij}^C}{2} \right), & \text{if } Y_i = 1; \\
0, & \text{otherwise.} 
\end{cases} \quad (39)$$

This is an independent probit model with variables $KDRI_i$, $W_{jkt}$, $HHI_{jk}$, $\eta_{ij}$, $\zeta_{ij}$, and the final term i.i.d. with zero mean.\textsuperscript{20} As we assume $e_{ij}$ has a normal distribution, we implement the two-step approach developed by Rivers and Vuong (1988)\textsuperscript{21}: (i) perform two OLS regressions: $W_{jkt}$ on $KDRI_i$, $\hat{W}_{jkt}$, and $HHI_{jk}$; and $HHI_{jk}$ on $KDRI_i$, $\hat{W}_{jkt}$, and $\hat{HHI}_{jk}$ to obtain residuals $\hat{\eta}_{ij}$ and $\hat{\zeta}_{ij}$ respectively; and (ii) perform probit regression of the intent probability on $KDRI_i$, $W_{jkt}$, $HHI_{jk}$, $\hat{\eta}_{ij}$ and $\hat{\zeta}_{ij}$. The results of the Step 1 regressions are in Table 6.

<table>
<thead>
<tr>
<th>Variable (Coefficient)</th>
<th>Coefficient Estimate</th>
<th>Variable (Coefficient)</th>
<th>Coefficient Estimate</th>
</tr>
</thead>
<tbody>
<tr>
<td>Constant ($\omega_0$)</td>
<td>1.266* (0.019)</td>
<td>Constant ($\psi_0$)</td>
<td>0.168* (0.005)</td>
</tr>
<tr>
<td>$KDRI_i$ ($\omega_1$)</td>
<td>0.031* (0.007)</td>
<td>$KDRI_i$ ($\psi_1$)</td>
<td>0.008* (0.002)</td>
</tr>
<tr>
<td>$\hat{W}_{jkt}$ ($\omega_2$)</td>
<td>0.383* (0.007)</td>
<td>$\hat{W}_{jkt}$ ($\psi_2$)</td>
<td>-0.039* (0.002)</td>
</tr>
<tr>
<td>$\hat{HHI}_{jk}$ ($\omega_3$)</td>
<td>-0.520* (0.020)</td>
<td>$\hat{HHI}_{jk}$ ($\psi_3$)</td>
<td>0.737* (0.005)</td>
</tr>
</tbody>
</table>

$F(3,75742) = 1874.74$, $R^2 = 0.07$

$F(3,76170) = 9664.48$, $R^2 = 0.28$

Note: Standard errors are in parenthesis. (*p < 0.0001)

**Table 6: Control Function Approach Step 1 Results**

Step 1 diagnostics such as the F-test and partial $R^2$ provide a sense of how well IVs perform in our model setting. As can be seen in Table 6, both regression models passed the F-test (i.e., Prob $> F = 0.000$) and 1− Partial $R^2$ values (0.98 and 0.99 for the waiting time and the competition IVs respectively) show high explanatory power.\textsuperscript{22} Additionally, there is reasonably high correlation (i.e., 0.25) between the first endogenous variable ($W_{jkt}$) and its IV; and there is high correlation (i.e., 0.52) between the second endogenous variable ($HHI_{jk}$) and its corresponding IV.

We test the evidence of endogeneity in Step 2 of the control function approach in Table 7. As can be seen in Table 7, the coefficient for $\zeta_{ij}$ is significant implying that the competition variable is endogenous. However, the coefficient for $\hat{\eta}_{ij}$ is not significant (i.e., Prob $> t = 0.419$) and hence we conclude that the waiting time until transplantation variable is exogenous. Note that the Wald test of combined exogeneity (Wooldridge (2002), pp. 472-477) for the competition and waiting time variables is rejected, so we have additional evidence of endogeneity in our specification. Note

\textsuperscript{20}Note that $\text{Var}(e_{ij}) > \text{Var}(\frac{\hat{e}_{ij}^W + \hat{e}_{ij}^C}{2})$, hence, the coefficient estimates need to be normalized. In addition, the probit standard errors and test statistics based on the utility function (39) will not be accurate because this regression will include the residuals from regressions based on functional forms (35) and (36). Therefore, we use bootstrapping for estimating the true standard errors.

\textsuperscript{21}Rivers and Vuong (1988) used only one IV in their approach, whereas in our approach we used two IVs and by the properties of the normal distribution we can separate out the part in the error term that correlates with the endogenous variables.

\textsuperscript{22}We calculate the partial $R^2$ (or the coefficient of partial determination) of the variables different form the IVs, which indicates the percentage of variation that is not explained by the IV and is explained by the remaining parameters. Assuming the reduced model includes only the IV and the full model includes all three variables, the partial $R^2 = \frac{\text{SSE(reduced)} - \text{SSE(full)}}{\text{SSE(reduced)}}$. 1− Partial $R^2$ provides a sense of the explanatory power of the IV.
that we also conduct the Wald test of exogeneity only for the competition variable and again find evidence of endogeneity of the competition variable.

\[
\begin{array}{|c|c|c|}
\hline
\text{Variable} & \text{Coefficient Estimate} & p-value \\
\hline
\text{Constant} & 2.851 (0.089) & 0.000 \\
\text{KDRI}_i & -0.988 (0.012) & 0.000 \\
\text{W}_{jkt} & -0.011 (0.038) & 0.773 \\
\text{HHI}_{jk} & -0.130 (0.068) & 0.056 \\
\hat{\eta}_{ij} & 0.031 (0.038) & 0.419 \\
\hat{\zeta}_{ij} & 0.215 (0.075) & 0.004 \\
\hline
\end{array}
\]

Wald test of exogeneity: \(\chi^2(2) = 10.19\) (Prob > \(\chi^2 = 0.006\))

Note: Bootstrap standard errors are in parenthesis.

Table 7: Control Function Approach Step 2 Results

Appendix E: Additional Tables

<table>
<thead>
<tr>
<th>Donor Characteristic</th>
<th>Applies to:</th>
<th>KDRI Coefficient (“Beta”)</th>
<th>KDRI “XBeta” Component</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (integer years)</td>
<td>All Donors</td>
<td>0.0128</td>
<td>0.0128*(age-40)</td>
</tr>
<tr>
<td></td>
<td>Donors with age &lt; 18</td>
<td>-0.0194</td>
<td>-0.0194*(age-18)</td>
</tr>
<tr>
<td></td>
<td>Donors with age &gt; 50</td>
<td>0.0107</td>
<td>0.0107*(age-50)</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>All donors</td>
<td>-0.0464</td>
<td>-0.0464*(hgt-170)/10</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>All donors w/ weight &lt; 80 kg</td>
<td>-0.0199</td>
<td>-0.0199*(wgt-80)/5</td>
</tr>
<tr>
<td>Ethnicity</td>
<td>African American donors</td>
<td>0.1790</td>
<td>0.1790</td>
</tr>
<tr>
<td>History of Hypertension</td>
<td>Hypertensive donors</td>
<td>0.1260</td>
<td>0.1260</td>
</tr>
<tr>
<td>History of Diabetes</td>
<td>Diabetic donors</td>
<td>0.1300</td>
<td>0.1300</td>
</tr>
<tr>
<td>Cause of Death</td>
<td>Donors w/ COD=CVA</td>
<td>0.0881</td>
<td>0.0881</td>
</tr>
<tr>
<td>Serum Creatinine</td>
<td>All donors</td>
<td>0.2200</td>
<td>0.2200*(creat-1)</td>
</tr>
<tr>
<td></td>
<td>Donors with creat &gt; 1.5 mg/dL</td>
<td>-0.2090</td>
<td>-0.2090*(creat-1.5)</td>
</tr>
<tr>
<td>HCV status</td>
<td>HCV positive donors</td>
<td>0.2400</td>
<td>0.2400</td>
</tr>
<tr>
<td>DCD status</td>
<td>DCD donors</td>
<td>0.1330</td>
<td>0.1330</td>
</tr>
</tbody>
</table>

Table 8: KDRI Donor Factors Estimated Coefficients

<table>
<thead>
<tr>
<th>Year</th>
<th>2000</th>
<th>2001</th>
<th>2002</th>
<th>2003</th>
<th>2004</th>
<th>2005</th>
</tr>
</thead>
<tbody>
<tr>
<td>85 Percentile KDRI</td>
<td>1.725</td>
<td>1.735</td>
<td>1.747</td>
<td>1.798</td>
<td>1.828</td>
<td>1.901</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Year</th>
<th>2006</th>
<th>2007</th>
<th>2008</th>
<th>2009</th>
<th>2010</th>
</tr>
</thead>
<tbody>
<tr>
<td>85 Percentile KDRI</td>
<td>1.886</td>
<td>1.903</td>
<td>1.88</td>
<td>1.888</td>
<td>1.851</td>
</tr>
</tbody>
</table>

Table 9: 85th Percentile of KDRI Variable by each Year
<table>
<thead>
<tr>
<th>Variable</th>
<th>Parameter Estimate</th>
<th>Whole Data</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>(Coefficient)</td>
<td>(Blood Type=A)</td>
</tr>
<tr>
<td><strong>Constant</strong></td>
<td>2.824***</td>
<td>2.750***</td>
</tr>
<tr>
<td>(β₀)</td>
<td>(0.067)</td>
<td>(0.056)</td>
</tr>
<tr>
<td><strong>KDRIᵢ</strong></td>
<td>-1.005***</td>
<td>-0.988***</td>
</tr>
<tr>
<td>(βᵦKDRI)</td>
<td>(0.024)</td>
<td>(0.021)</td>
</tr>
<tr>
<td><strong>Wᵢjk</strong></td>
<td>0.016</td>
<td>0.025*</td>
</tr>
<tr>
<td>(βᵦW)</td>
<td>(0.018)</td>
<td>(0.012)</td>
</tr>
<tr>
<td><strong>HHIᵢjk</strong></td>
<td>-0.082</td>
<td>-0.102</td>
</tr>
<tr>
<td>(βᵦHHI)</td>
<td>(0.083)</td>
<td>(0.071)</td>
</tr>
<tr>
<td>Num. of Obs.</td>
<td>28,925</td>
<td>36,417</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Variable</th>
<th>Parameter Estimate</th>
<th>Bottom 15% Quality Donors</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>(Coefficient)</td>
<td>(Blood Type=A)</td>
</tr>
<tr>
<td><strong>Constant</strong></td>
<td>2.634***</td>
<td>2.602***</td>
</tr>
<tr>
<td>(β₀)</td>
<td>(0.142)</td>
<td>(0.117)</td>
</tr>
<tr>
<td><strong>KDRIᵢ</strong></td>
<td>-0.943***</td>
<td>-0.980***</td>
</tr>
<tr>
<td>(βᵦKDRI)</td>
<td>(0.055)</td>
<td>(0.045)</td>
</tr>
<tr>
<td><strong>Wᵢjk</strong></td>
<td>0.043</td>
<td>0.071***</td>
</tr>
<tr>
<td>(βᵦW)</td>
<td>(0.028)</td>
<td>(0.020)</td>
</tr>
<tr>
<td><strong>HHIᵢjk</strong></td>
<td>-0.015</td>
<td>-0.081</td>
</tr>
<tr>
<td>(βᵦHHI)</td>
<td>(0.078)</td>
<td>(0.067)</td>
</tr>
<tr>
<td>Num. of Obs.</td>
<td>4,042</td>
<td>5,577</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Variable</th>
<th>Parameter Estimate</th>
<th>Top 85% Quality Donors</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>(Coefficient)</td>
<td>(Blood Type=A)</td>
</tr>
<tr>
<td><strong>Constant</strong></td>
<td>2.954***</td>
<td>2.957***</td>
</tr>
<tr>
<td>(β₀)</td>
<td>(0.080)</td>
<td>(0.066)</td>
</tr>
<tr>
<td><strong>KDRIᵢ</strong></td>
<td>-1.111***</td>
<td>-1.147***</td>
</tr>
<tr>
<td>(βᵦKDRI)</td>
<td>(0.033)</td>
<td>(0.037)</td>
</tr>
<tr>
<td><strong>Wᵢjk</strong></td>
<td>0.009</td>
<td>0.012</td>
</tr>
<tr>
<td>(βᵦW)</td>
<td>(0.020)</td>
<td>(0.013)</td>
</tr>
<tr>
<td><strong>HHIᵢjk</strong></td>
<td>-0.060</td>
<td>-0.073</td>
</tr>
<tr>
<td>(βᵦHHI)</td>
<td>(0.110)</td>
<td>(0.076)</td>
</tr>
<tr>
<td>Num. of Obs.</td>
<td>25,316</td>
<td>31,319</td>
</tr>
</tbody>
</table>

Note: Bootstrap standard errors are in parenthesis.

*** p < 0.001, ** p < 0.01, * p < 0.05, + p < 0.10.

Table 10: Summary of Estimation Models by Blood Type from Whole Data, Bottom 15%, and Top 85% Quality Donors (Endogeneity-corrected Intent Model)