Choices and Outcomes in Assignment Mechanisms: The Allocation of Deceased Donor Kidneys*†

Nikhil Agarwal, Charles Hodgson, Paulo Somaini‡

Abstract

While the mechanism design paradigm emphasizes notions of efficiency based on agent preferences, policymakers often focus on alternative objectives. School districts emphasize educational achievement, and transplantation communities focus on patient survival. It is unclear whether choice-based mechanisms perform well when assessed based on these outcomes. This paper evaluates the assignment mechanism for allocating deceased donor kidneys on the basis of patient life-years from transplantation (LYFT). We examine the role of choice in increasing LYFT and compare equilibrium assignments to benchmarks that remove choice. Our model combines choices and outcomes in order to study how selection affects LYFT. We show how to identify and estimate the model using instruments derived from the mechanism. The estimates suggest that the design in use selects patients with better post-transplant survival prospects and matches them well, resulting in an average LYFT of 8.78, which is 0.92 years more than a random assignment. However, the maximum aggregate LYFT is 13.84. Realizing the majority of the gains requires transplanting relatively healthy patients, who would have longer life expectancies even without a transplant. Therefore, a policymaker faces a dilemma between transplanting patients who are sicker and those for whom life will be extended the longest.

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‡Agarwal: Department of Economics, MIT, and NBER, email: agarwln@mit.edu. Hodgson: Department of Economics, Yale University, email: charles.hodgson@yale.edu. Somaini: Stanford Graduate School of Business and NBER, email: soma@stanford.edu.
1 Introduction

Assignment mechanisms are commonly used to allocate scarce resources. Examples include public schools, public housing, and organ allocation. While the design of these mechanisms takes choice-theoretic notions of efficiency as a primary objective (Roth and Sotomayor, 1992; Abdulkadiroglu and Sönmez, 2003), this desideratum often differs from the goals of policymakers – school districts emphasize student achievement and organ transplant systems emphasize patient survival.

Because canonical choice-based mechanisms are not designed to optimize these outcomes, they may not perform well on these dimensions. Agents’ choices may not be well-informed and co-ordination failures may undercut this objective. If so, a planner who can dictate assignments based on estimated benefits may be able to do better. However, agents may also have private information about the likely outcomes and using a choice-based mechanism may serve policymakers’ objectives.

This paper evaluates the mechanism used to allocate deceased donor kidneys on the basis of survival outcomes. We compare the performance and distributional consequences of the mechanism to alternative assignments. Our benchmark assignments investigate whether maximizing survival is in conflict with distributional concerns (Atkinson, 1970) or prioritarianism which targets the sickest or neediest (c.f. Persad et al., 2009; Waldinger, 2017). We also assess the role of choice by examining its relationship to survival and considering alternatives that dictate assignments using observables alone.

We make several contributions in service of this objective. We present the first quasi-experimental estimates of the Life-Years from Transplantation (LYFT), defined as the difference between median survival with and without a transplant, as a function of patient/donor-specific observed and unobserved characteristics. The current state-of-the-art in the medical literature relies on observational approaches (Wolfe et al., 2008), in part because conducting randomized control trials is both challenging and creates ethical issues. We use insights from the literature on generalized Roy selection to analyze a joint model of choices and outcomes in

\footnote{Moreover, in the kidney allocation context, surgeons who advise patients may suffer from agency problems that can misalign decisions relative to maximizing survival outcomes.}
an assignment mechanism. In contrast to the standard framework with multiple treatments (e.g. Lee and Salanié, 2018; Heckman and Pinto, 2018), assignment contexts often do not have a small number of treatments, in our case because each donor is unique. We therefore model potential outcomes as a function of patient, donor and match-specific characteristics, some of which are unobserved. Our results show how to identify and estimate the effects of counterfactual assignments by using variation in offers made to patients and choice shifters that are excluded from outcomes.

Deceased donor organs are a scarce and valuable resource. Only a sixth of the approximately 100,000 patients waiting for a kidney are transplanted annually, and thousands die while waiting. Increasing LYFT is an important policy goal: transplantation committees use observational estimates of LYFT to evaluate proposed reforms. When a kidney becomes available, patients on the waitlist are offered the organ in a priority order. Patients, or surgeons acting on their behalf, may choose to reject an offer and instead wait for a future organ. This decision may depend on the perceived benefits of a transplant from the offered organ.

We jointly model acceptance decisions and survival outcomes to incorporate the potential for selection. The first component of our model considers the choices patients make; the second and third components respectively model patient untransplanted survival and post-transplant survival with the offered organ. These models use a rich set of patient and organ attributes as well as time to treatment. Given our focus on evaluating alternative assignments, we also include patient- and patient-donor level unobservables.

Identification of the model is challenging because transplanted patients can be selected on untransplanted survival, post-transplant survival from an average kidney, or patient-kidney match-specific survival. Selection on these margins can be induced both because choices can depend on survival prospects and because patient waiting time is prioritized in the mechanism.

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3Reports to the OPTN Kidney Transplantation Committee generated by the Scientific Registry of Transplant Recipients (SRTR) of alternative designs use average LYFT as a summary measure of performance. The committee’s meeting minutes indicate that this measure is focal. In fact, the U.S. has considered a priority system based on LYFT in the past, and the U.K. uses a “transplant benefit score” to allocate kidneys (Watson et al., 2020).
We identify our model by combining two sources of variation. The first source is randomness in the offers made to a given patient, conditional on the patient’s priority-type in the mechanism. It allows us to compare the survival outcomes of patients whose final assignments differed due to the organs they were offered. Using standard arguments (e.g. Imbens and Angrist, 1994), we show that this instrument identifies a treatment effect for the select group of patients whose assignment is affected by an offer.

An important limitation of this estimand is that it does not allow us to predict survival from counterfactual assignments. It cannot consider changes in the set of patients who are transplanted or changes in the kidneys to which a patient is matched. To fill this gap, we first use novel arguments to identify our choice model. We then show that a continuous shifter of choices that is excluded from outcomes can be used to identify the effects of alternative assignments. Related approaches have been used in other settings by Geweke et al. (2003); Heckman and Navarro (2007); Lewbel (2007); Hull (2018) to correct for selection and to estimate marginal treatment effects (Heckman and Vytlacil, 2005). Our choice shifter is based on organ scarcity controlling for geography and time. We estimate the model using a Gibbs’ sampler similar to Geweke et al. (2003).

Our estimates suggest that choices and assignments are positively correlated with survival outcomes due to both observed and unobserved factors. Patients are more likely to accept kidneys that result in longer survival and those with match-specific benefits. Partly because of this, transplanted patients have a higher LYFT from the average organ as compared to untransplanted patients. Thus, prior approaches that do not account for selection on unobservable factors (e.g. Wolfe et al., 2008) yield biased estimates.

Next, we benchmark the observed assignment from the perspective of a utilitarian planner who’s objective is to maximize LYFT. We focus on survival effects because it is a focal outcome for kidney allocation, and compare the observed assignment to alternatives ranging from a random assignment to one that maximizes LYFT. Because distributional constraints may limit the ability to select which patients get a transplant, we also consider alternatives that re-assigns organs while fixing the set of transplanted patients. Finally, we measure the LYFT increase that can be achieved by a planner who can dictate assignments based only on observed patient and donor characteristics.
The observed assignment produces higher LYFT than random allocation – 8.78 years versus 7.87. Most of this gain comes from allowing patient choice. Assignment to patients based on existing priority rules without allowing for choice only achieves an average LYFT of 8.01. The drop from the observed assignment suggests that choice may not be dispensable if the unobserved types are private information.

But, there is significant room for improvement – the maximum possible LYFT given the available organs is 13.84. The increase comes from selecting patients who benefit more from the transplant and matching these patients to donors who are more suitable for them. A significant portion of these gains can be achieved if a planner can dictate assignments using observables in our dataset.

These potential improvements in LYFT have important distributional consequences that may present real-world challenges. Although a priori unclear because the sickest may also have benefited the most from a transplant, increasing LYFT requires transplanting patients who would have lived longer without a transplant because LYFT and survival without a transplant are strongly correlated. Such re-distribution creates distributional concerns because it increases the dispersion in remaining life-years (Atkinson, 1970). While some medical ethicists may still support maximizing total survival benefits especially in the presence of scarce resources, others consider worst-off prioritarianism for the sickest as important (see Persad et al., 2009, and references therein). Our results indicate that the planner faces a dilemma between these two goals.

Related Literature: We provide an alternative perspective for evaluating assignments to the literature studying assignment mechanisms (Roth and Sotomayor, 1992). For example, the theory of school choice typically bases welfare on student preferences (Abdulkadiroglu and Sönmez, 2003), and the empirical literature uses a willingness to travel measure for welfare comparisons (see Agarwal and Somaini, 2020, for a survey).

Instead of survival outcomes, the economics literature on organ donation focuses either on the number of transplants (e.g. Teltser, 2019; Dickert-Conlin et al., 2019) or on decision-theoretic notions of welfare (Agarwal et al., 2021), with an influential literature focusing on expanding living donor kidney exchange (e.g. Roth et al., 2004; Agarwal et al., 2019). Yet,
the vast majority of kidney transplants come from deceased donor organs.

Our paper also relates to recent approaches that leverage quasi-experimental variation in school choice mechanisms to estimate school quality (e.g. Abdulkadiroglu et al., 2017). This literature estimates a local average treatment effect, which is not sufficient for analyzing outcomes from counterfactual assignments because of changes in the set of compliers. We address this issue using a choice shifter. In contemporaneous work, Kapor et al. (2020) use this message of our paper to study outcomes in a college admissions setting.

The techniques we use build on a large literature studying selection models (Roy, 1951). Our model is related to models that combine outcomes with choice models to correct for selection when estimating treatment effects (Geweke et al., 2003; Heckman and Navarro, 2007; Lewbel, 2007; Hull, 2018), causal survival models (Abbring and Van den Berg, 2003), and models of multi-valued treatments (Lee and Salanié, 2018; Heckman and Pinto, 2018).

The main difference relative to these papers is that patients may have match-specific benefits from an organ, resulting in a large number of unique treatments. This issue is important in assignment contexts whenever there are a large number of heterogeneous objects. We address it by using a model with rich observed heterogeneity across objects and unobserved heterogeneity in outcomes along three dimensions – baseline outcomes, average outcomes given observable characteristics of the transplanted organ, and match-specific effects – with each dimension correlated with unobservables in the choice model.

Overview: Section 2 describes the institutions and the data. The model and the instruments are described in Sections 3 and 4. Section 5 presents the identification results and the empirical model. The estimates, LYFT in the observed mechanism and counterfactuals are in Sections 6, 7 and 8 respectively.

2 Background, Data, and Descriptive Evidence

2.1 Institutional Features

Basics of Kidney Transplantation: Approximately 750,000 patients are afflicted with End-Stage Renal Disease (ESRD) in the United States (USRDS, 2018). Medicare provides near
universal coverage for costs related to ESRD, irrespective of age, costing the taxpayer $35.4 billion in 2016 (7.2% of Medicare claims (USRDS, 2018), approximately 1% of the federal budget).

Transplantation is considered the best treatment for ESRD. Each transplant is estimated to extend a patient’s life by several years (Wolfe et al., 2008) while also saving between $195,000 – $400,000 in dialysis costs (Irwin et al., 2012; Held et al., 2016). These estimates are based on survival models and comparisons of healthcare costs with and without a transplant. We improve on the former set of estimates by using quasi-experimental variation.

There is significant potential for heterogeneity in survival effects, even amongst compatible patient-donor pairs (Danovitch, 2009). First, survival both with and without a transplant can differ across patients. Some patients tolerate dialysis better than others and co-morbidities influence post-transplant survival prospects. Second, donor quality – circumstances of the donor’s death, kidney function, and the donor’s health prior to death – can significantly influence transplant outcomes. Finally, there may be match-specific factors that affect post-transplant survival. Examples include size and weight match as well as tissue-protein similarity between patient and donor.

**The Allocation of Deceased Donor Kidneys:** The allocation of deceased donors organs is organized using a prioritized waiting list. Patients receive offers when an organ becomes available and may choose to accept or reject it. Each donor’s kidneys are allocated to the highest-priority patients on the waitlist who are willing to accept the organs.

During our sample period, priority was based primarily on waiting time and tissue-type similarity between the patient and donor. Each kidney was first offered to patients with a perfect tissue-type match, then to patients from the local area in which the organs were recovered, then regionally, and finally nationally. Within each priority group, a points system that emphasized waiting time was used to order patients (see OPTN, 2014, for details). This allocation system evolved over time with incremental changes to enhance efficiency (Smith et al., 2012).  

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4A revision to the system aimed at improving survival benefits was implemented on December 4, 2014. This system also uses a priority-based waiting list that emphasizes waiting time, geography and patient sensitization. The change gives greater priority to the patients in the top quintile of expected post-transplant survival for the top quintile predicted organ quality.
There are three features of the kidney allocation system that are worth highlighting. First, unlike the assignment systems for some other organs (for example, livers and hearts), the kidney assignment system does not use patient urgency to determine priority. Second, patients who reject an offer remain on the list and may choose to accept the next offer with no penalty in priority for refusing an offer. Third, the design is based on heuristics aided by simulations and compromises in consideration of distributional effects rather than a formal mechanism design approach (see Stegall et al., 2017, for a historical perspective).

2.2 Data and Descriptive Analysis

2.2.1 Data Sources

This study uses data from the Organ Procurement and Transplantation Network (OPTN). The OPTN data system includes data on all donors, wait-listed candidates, and transplant recipients in the US, submitted by the members of the OPTN. The Health Resources and Services Administration (HRSA), U.S. Department of Health and Human Services provides oversight to the activities of the OPTN contractor.

The data include detailed information on patient and donor characteristics, and survival outcomes from the Standard Transplantation Analysis and Research dataset. They also include all offers made by the system and accept/reject decisions from the Potential Transplant Recipient dataset. These data are populated using information gathered during the allocation process, forms submitted by transplant centers from patient follow-ups after a transplant is performed, and patient death dates merged from social security records.

We restrict attention to patients who first joined the kidney waiting list between January 1st, 2000 and December 31st, 2010. From this set, we exclude patients who needed multiple organ transplants and those that received a living donor kidney (see Appendix A for a detailed discussion). Correspondingly, we only use data on donor offers and acceptance decisions for our sample of patients.

The survival records are consistently populated until December 31st, 2015, allowing us to track survival outcomes for up to sixteen years from registration for our sample of patients.\(^5\)

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\(^5\)Our data use agreement allows for periodic updates, which we plan to include in future iterations of the
For patients without death records, we use information from the waitlist for untransplanted patients and from annual post-transplant follow-ups for transplanted patients to construct a censored measure of patient survival.

2.2.2 Descriptive Analysis

Table 1: Patient Characteristics

<table>
<thead>
<tr>
<th>All Patients</th>
<th>Received Deceased Donor Transplant</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Mean</strong></td>
<td><strong>Mean</strong></td>
</tr>
<tr>
<td><strong>S.D.</strong></td>
<td><strong>S.D.</strong></td>
</tr>
<tr>
<td>New Patients per Year</td>
<td>15956</td>
</tr>
<tr>
<td>Died by Year Five (%)</td>
<td>27.4</td>
</tr>
<tr>
<td>Survived Five Years (%)</td>
<td>64.2</td>
</tr>
<tr>
<td>Censored by Year Five (%)</td>
<td>8.4</td>
</tr>
<tr>
<td>Transplanted by Year Five (%)</td>
<td>47.2</td>
</tr>
<tr>
<td>Age at Registration</td>
<td>51.4</td>
</tr>
<tr>
<td>On Dialysis at Registration (%)</td>
<td>77.3</td>
</tr>
<tr>
<td>Diabetic Patient (%)</td>
<td>42.9</td>
</tr>
<tr>
<td>BMI at Registration</td>
<td>28.2</td>
</tr>
</tbody>
</table>

Sample includes 175518 patients who registered between 2000 and 2010. Transplant and survival data are available through 12/31/2015. Patients for whom we do not observe death are censored. The observed survival duration is computed based on the date and status of the patient when we last observe her. See A.4 for detailed computation of observed survival. Durations presented in Panel A are time since registration.

Patients and Donors: Patients face extreme scarcity, with a significant fraction dying while awaiting a transplant. Panel A of Table 1 shows that an average of 15956 patients registered each year on the kidney waiting list, of which 27.4% die within five years of registering and only 47.2% receive a transplant during this time period. The chances of receiving a transplant decline after the first five years, with only 54% of patients ultimately receiving a deceased donor kidney. The remaining patients either still await a kidney or leave the list.

Panel B shows that patients receiving a transplant are younger and appear to have been in better health at the time of registration. Transplanted patients are less likely to be on dialysis at the time of registration, are less likely to be diabetic, and have a lower body mass index. Thus, observed characteristics induce correlation between probability of receiving a transplant and survival without a transplant.
Table 2: Donor Characteristics

<table>
<thead>
<tr>
<th></th>
<th>All Donors</th>
<th>Any Kidney Discarded</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>S.D.</td>
</tr>
<tr>
<td>Number of Donors per Year</td>
<td>6181</td>
<td>51</td>
</tr>
<tr>
<td>Median Number of Offers per Donor</td>
<td>543.5</td>
<td>1927.9</td>
</tr>
<tr>
<td>Average Number of Offers per Donor</td>
<td>39.2</td>
<td>18.4</td>
</tr>
<tr>
<td>Cause of Death -- Head Trauma (%)</td>
<td>39.7</td>
<td>48.9</td>
</tr>
<tr>
<td>Hypertensive Donor (%)</td>
<td>28.6</td>
<td>45.2</td>
</tr>
<tr>
<td>Donor Creatinine</td>
<td>1.2</td>
<td>1.0</td>
</tr>
<tr>
<td>Non-Heart Beating Donor (%)</td>
<td>7.9</td>
<td>26.9</td>
</tr>
<tr>
<td>KDPI</td>
<td>0.5</td>
<td>0.3</td>
</tr>
</tbody>
</table>

Notes: Sample includes deceased donors offered between 2000 and 2010 to patients in the sample.

Patients exercise choice despite scarcity, often rejecting undesirable organs. Table 2 shows that the number of offers per donor is 543.5, but the median is much lower, at 51. This skewed distribution arises because undesirable kidneys are rejected by many, while desirable kidneys are accepted quickly. Indeed, 18.9% of donors have at least one viable kidney discarded. Organs from these donors were refused by 1890.5 patients on average.

Predictors of organ quality are correlated with number of offers and discards in expected ways. Donors whose kidney(s) was/were discarded are older, less likely to have died from head trauma, more likely to be diabetic or hypertensive, have higher creatinine levels (an indicator of lower kidney function), and more likely to have donated after cardiac death (Table 2). An aggregate of these and other characteristics is the Kidney Donor Profile Index (KDPI), which indicates the fraction of donors with a lower estimated risk of graft failure.

Survival: We focus on survival as the primary outcome of interest for several reasons. First, this outcome is arguably the most important one from the perspective of the patient and also the policy-makers. Predicted LYFT from observational models was explicitly used by the OPTN Kidney Transplantation Committee to evaluate proposed designs. Second, moving an ESRD patient from dialysis to transplantation saves on expensive dialysis treatment. While we do not directly evaluate this component, future research can use our estimates to revisit cost-benefit analyses. Third, this outcome can be measured relatively easily. The other most
commonly discussed effect is on quality of life, which is hard to quantify.

Figure 1 shows survival curves for transplanted and untransplanted patients, separated by young and old patients (above/below the median age of 54) and by whether or not the transplanted patient received a kidney from a donor with a discarded kidney. Donors with a discarded kidney are more likely to be undesirable because only one patient accepted the donor’s kidneys. As indicated by the waiting times shown via the vertical dashed lines, the average waiting time for a patient who receives a kidney from a donor without a discard is higher than that for a donor with a discard.

**Figure 1: Patient Survival**

These survival curves show that transplanted patients live significantly longer than patients who do not receive a transplant. Moreover, they are substantially different for young versus old patients and for patients transplanted with a desirable versus undesirable organ. Only about half of the young patients who do not receive a transplant survive more than 7.9
years, but more than half of the young patients who receive a transplant from a donor with desirable organs live past 16 years. These statistics are 5.4 and 11.3 years, respectively, for older patients, indicating that older patients have shorter half-lifes both with and without a transplant.\textsuperscript{6} For both groups of patients, a transplant from an undesirable organ is associated with half-lives that are shorter by about a year or more.

These observations also point to the potential for choices and assignments to be correlated with survival outcomes. Next, we turn to a model that incorporates these features.

3 A Model of Decisions and Outcomes

Our model considers assignment mechanisms in which organs, indexed by \( j \), are assigned to patients, indexed by \( i \). When an organ arrives, offers are made to patients on a waiting list who must decide to accept or reject it. These decisions translate into an assignment, and an outcome is realized.\textsuperscript{7}

3.1 Assignment Mechanism and Observed Outcomes

Organs arrive sequentially, their index \( j \) denotes their arrival order. The mechanism orders patients on the waiting list according to an organ-specific priority score that may depend on the time that a patient has waited. Offers are made in this priority order. Acceptance by \( i \) of an offer for organ \( j \) is denoted with \( D_{i,j} = 1 \). Organs are assigned to the highest priority patients that accept an offer. Finally, patients that have been assigned an organ are removed from the list. Other patients may also leave the list.

Consider the set of organs that are feasible for patient \( i \). Holding fixed the decisions of the other patients, let \( \bar{J}_i \) be an ordered set of organs offered to patient \( i \) if she refuses all offers made to her and she was registered indefinitely. Because patients may die before assignment, she receives a subset of offers denoted by \( \tilde{J}_i \). Thus \( \bar{J}_i = (j \in J_i : Y_{i,0} \geq t_{i,j}) \), where \( t_{i,j} \) is the

\textsuperscript{6}We focus on median survival instead of expected life-years because we can track survival for up to sixteen years. This choice is consistent with prior work measuring the life-year benefits from transplantation (see Wolfe et al., 1999, 2008, for example).

\textsuperscript{7}In our empirical context, patient decisions may be delegated or made jointly with a surgeon. We do not distinguish between the these alternatives.
time between patient $i$’s registration and donor $j$’s arrival, and $Y_{i,0}$ is untransplanted survival. Patient $i$’s assignment depends both on the feasible set of organs and her decisions. Let $T_{i,j} = 1$ denote patient $i$ being assigned organ $j$. Note that

$$T_{i,j} = \prod_{j' < j, j' \in \hat{J}_i} (1 - D_{i,j'}) D_{i,j},$$

where $D_{i,j} = 1$ if patient $i$ accepts organ $j$. Therefore, each patient $i$ is assigned to the first organ that she accepts from the set $\hat{J}_i$. We assume that the analyst observes the offer set $\hat{J}_i$ and the decisions $D_{i,j}$ for the offers made $j \in \hat{J}_i$. Observing the choice set and decisions is typical when administrative data from an assignment mechanism is available.

The observed outcome $Y_i$ depends on whether a patient is assigned and to which organ she is assigned. It is given by

$$Y_i = \sum_{j \in \hat{J}_i} T_{i,j} Y_{i,j} + \left(1 - \sum_{j \in \hat{J}_i} T_{i,j}\right) Y_{i,0},$$

where $Y_{i,j}$ is the survival outcome of patient $i$ from being assigned organ $j$.

This formulation abstracts away from potential truncation of observed survival for simplicity of notation. In our empirical context, we only observe a censored survival outcome for some patients, allowing us to deduce that $Y_i > Y_{i,C}$, where $Y_{i,C}$ is the censoring time. We will account for this censoring, making the standard assumption that the censoring time is independent of the latent duration (see equation 20.22 in Wooldridge, 2010).

### 3.2 Latent Outcomes and Decisions

There are three key sets of primitives in our model:

**Unassigned Outcome:** The outcome for patient $i$ if the patient is not assigned any organ is given by

$$Y_{i,0} = g_0(x_i, \nu_{i,0}),$$

where $x_i \in \mathbb{R}^{d_x}$ are patient-specific observables; $\nu_{i,0} \in \mathbb{R}$ denotes a patient-specific unobservable; and $Y_{i,0} \in \mathbb{R}$. 


**Assignment Outcome:** The outcome of patient $i$ from being assigned organ $j$ is given by

$$Y_{i,j} = g_1(q_j, x_i, \nu_{i,1}, \varepsilon_{i,j,1}),$$  \hspace{1cm} (3.2)

where $x_i \in \mathbb{R}^{d_x}$ is a vector of patient-specific observed characteristics; $q_j \in \mathbb{R}^{d_q}$ denotes the observed characteristics of organ $j$, which we will refer to as organ-types; $\nu_{i,1} \in \mathbb{R}$ denotes a patient-specific unobservable; $\varepsilon_{i,j,1} \in \mathbb{R}$ denotes an unobservable that are patient- and organ-specific; and $Y_{i,j} \in \mathbb{R}$.

Since $Y_{i,j}$ and $Y_{i,0}$ denote survival outcomes in our application, they can be written as arising from survival models with time-varying hazard rates that depend on unobservables. This model allows for rich heterogeneity along observable and unobservable dimensions. It also allows for time to treatment effects since $x_i$ and $q_j$ can include the dates on which patient $i$ and organ $j$ arrive. Moreover, there are multiple levels of unobserved heterogeneity. Outcomes are heterogeneous across $i$ due to $\nu_{i,1}$ and $\nu_{i,0}$, and within treatment types (defined by $q_j$) for a given $i$ because of $\varepsilon_{i,j,1}$.

**Decision Equation:** We model the acceptance decision as

$$D_{i,j} = g_D(q_j, x_i, z_i, \nu_{i,D}, \varepsilon_{i,j,D}) \in \{0, 1\}$$  \hspace{1cm} (3.3)

where $D_{i,j} = 1$ denotes accept; $\nu_{i,D} \in \mathbb{R}$ denotes unobserved selectivity of patient $i$; $\varepsilon_{i,j,D} \in \mathbb{R}$ is a shock that is specific to the patient and the organ; and $z_i \in \mathbb{R}^{d_z}$ are observables that influence the decision of a patient. Without loss of generality, we assume that $g_D$ is non-increasing in $\nu_{i,D}$ and non-decreasing in $\varepsilon_{i,j,D}$.

The choice model nests several primitive models of decisions. It is consistent with both myopic decision rules and a dynamic decision process in which patients do not have foresight over future offers, but base their decisions on their beliefs about the distribution of offers. Although we remain agnostic about the micro-foundations, this formulation and our empirical specification nests the optimal stopping problem in Agarwal et al. (2021). \footnote{In this model, an offer is accepted if the (perceived net present) value from accepting the organ exceeds the option value of waiting. Specifically, let $g_D = 1$ if $U_i(q_j, x_i, \varepsilon_{i,j,D}) > V(x_i, \nu_{i,D})$ where $U(\cdot)$ is the net present}
The main difference between $x_i$ and $z_i$ is that the latter is excluded from the outcome equations. For example, $z_i$ could include variables that influence this decision, say through the distribution of future offers, but is unrelated to the benefits of accepting a given organ. This exclusion restriction, combined with Assumption 1(i) below, introduces instruments in the model that we will use in the empirical strategy. The specific instruments $z_i$ used in our application are discussed in Section 4.

Our data generating process samples a set of patients and a set of organs independently along with their respective characteristics $(x_i, z_i, \nu_i)$ and $q_j$, where $\nu_i = (\nu_{i,0}, \nu_{i,1}, \nu_{i,D})$. It then samples the match-specific unobservables $\varepsilon_{i,j} = (\varepsilon_{i,j,1}, \varepsilon_{i,j,D})$. We make the following restriction on this process:

**Assumption 1.** (i) $\varepsilon_i = \{\varepsilon_{i,j}\}_j$ and $\nu_i$ are mutually independent conditional on $(x_i, z_i)$ and $(q_j)_j$.

(ii) The random vector $\nu_i$ is distributed independent and identically distributed (i.i.d.) across $i$.

(iii) The random vector $\varepsilon_{i,j}$ is distributed i.i.d. across $i$ and $j$.

The assumption allows for dependence between the components of $\nu_i$ and the components of $\varepsilon_{i,j}$, thereby allowing for $Y_{i,j}$ and $Y_{i,0}$ to be correlated with each other and with $D_{i,j}$. The independence assumptions imply that patients' outcomes do not depend on other patients' treatment assignment, which implies the stable unit treatment value assumption.

Our goal is to identify the function $g_D(\cdot)$ and the marginal distributions of $Y_{i,j}$ and $Y_{i,0}$ conditional on the vector $(x_i, q_j, z_i, \varepsilon_{i,j,D}, \nu_{i,D})$. The residual uncertainty in the distribution of $Y_{i,0}$ is only because of patient-specific unobservables $\nu_{i,0}$, whereas it is due to both match-specific effects $\varepsilon_{i,j,1}$ and patient-specific effects $\nu_{i,1}$ for $Y_{i,j}$. Incorporating these sources is necessary for capturing unobserved match-specific drivers of outcomes. Identifying these distributions

---

9For example, the first moments of the marginals we identify are $E[Y_{i,0}|x_i, z_i, \nu_{i,D}] = \int g_0(x_i, \nu) f_{\nu_{i,D}}(\nu) d\nu$ and $E[Y_{i,j}|x_i, q_j, z_i, \varepsilon_{i,j,D}, \nu_{i,D}] = \int \int g_1(q_j, x_i, \nu, \varepsilon) f_{\varepsilon_{i,j,D}}(\varepsilon) f_{\nu_{i,D}}(\nu) d\nu d\varepsilon$, where, the distributions of $\nu_{i,1}$ and $\nu_{i,0}$ may depend on $\nu_{i,D}$, and the distribution of $\varepsilon_{i,j,1}$ may depend on $\varepsilon_{i,j,D}$.  

---

value of accepting an offer for $j$, $V(\cdot)$ is the option value of waiting. Agarwal et al. (2021) estimate this model by first estimating conditional choice probabilities using a probit model where $g_D = 1 \{f(q_j, x_i, \varepsilon_{i,j,D}; \theta) > 0\}$ using a reduced-form function $f$ parametrized in terms of $\theta$. Their empirical specification is more restrictive than ours as it omits $\nu_{i,D}$ and $z_i$, and does not consider survival effects from transplantation.
will also allow us to condition only on a subset of the variables \((x_i, q_j, z_i, \varepsilon_{i,j,D}, \nu_{i,D})\) depending on the quantities on which counterfactual assignments depend.

The model and Assumption 1 together impose three main restrictions. First, unobserved patient selectivity, \(\nu_{i,D}\) is fixed across all organs and time, implying a fixed ordering of patients on selectivity for all organ types. Second, selectivity and survival outcomes can be correlated through \(\nu_i\), but we abstract away from time-varying information about survival that is unobserved to the econometrician and also affects decisions. Relaxing these two restrictions is challenging because patients in our setting can accept at most one offer and we observe a single survival outcome (see also Abbring and Van den Berg, 2003; Unkel et al., 2014). Third, a patient’s decision does not depend directly on the specific decisions of other patients for a given organ since \(\nu_i\) and \(\varepsilon_{i,j}\) are independent of \(\nu_{i'}\) and \(\varepsilon_{i',j'}\).

In addition, we rule out statistical dependence between the subset of organs offered to a patient and her unobservables:

**Assumption 2.** The sequence of offers \(J_i\) is conditionally independent of \((\nu_i, \varepsilon_i)\) given \(x_i\).

Assumption 2 is satisfied if \(x_i\) controls for a sufficiently rich set of patient characteristics such that the remaining variation in potential offers is independent of unobserved determinants of a patient’s outcomes and decisions. The assumption allows for \(J_i\) to depend on the unobservables of other patients \(i'\). But, because \(J_i\) is excluded from \(i'\)’s potential outcomes and affects assignment, it is an instrument for which organ is assigned to \(i\). Section 4.1 argues that the assumption is plausible in our empirical setting.

An implication of this assumption is that, patients cannot alter their decisions or their outcomes in response to specific future offers, ruling out foresight over the organs that will be offered in the future. This restriction parallels the “no anticipation” assumption in Abbring and Van den Berg (2003). Nonetheless, recall that our choice model nests the model in Agarwal et al. (2021) where forward-looking patients strategically refuse offers based on the distribution of future offers.

The sequential nature of choices and treatment assignment in our model resembles that of Heckman and Navarro (2007). There are two main differences. First, outcomes and choices for a patient from different organs of the same type \(q_j\) are heterogeneous in our
framework whereas the standard framework uses a finite set of known types. This allows for the realistic possibility that choices and survival outcomes of a patient can vary across two observationally identical donors. Capturing such match-specific effects can be important in other assignment problems with highly heterogeneous agents. Second, our choice shifter \( z_i \) varies and the individual level, not at the individual-treatment level. As we discuss below, we combine this instrument with variation in offers \( J_i \) to identify treatment effects.

3.3 Sources of Selection

The model allows for selection into transplantation on three dimensions: untransplanted survival \( Y_{i,0} \); average survival across transplants \( \bar{Y}_i = \frac{1}{|J|} \sum_j Y_{i,j} \); and match-specific survival \( Y_{i,j} - \bar{Y}_i \). There are two potential sources of selection: selection due to patient choices and selection due to patient mortality. Selection on these sources creates endogeneity in \( T_{i,j} \) that our framework addresses.

Selection due to choice occurs if choices \( D_{i,j} \) are correlated with survival outcomes \( Y_{i,0} \) or \( Y_{i,j} \). Choice can induce selection on \( Y_{i,0} \) if, for example, patients with higher expected survival without a transplant due to unobserved health conditions are more selective. That is, if \( E[Y_{i,0} | \nu_{i,D}, x_i] \) varies with \( \nu_{i,0} \), where expectations are taken over \( \nu_{i,0} \). Similarly, choice can induce selection on average transplanted survival, \( \bar{Y}_i \), if \( E[Y_{i,j} | \nu_{i,D}, x_i, q_j] \) varies with \( \nu_{i,0} \), where expectations are taken over \( \nu_{i,1} \) and \( \varepsilon_{i,j,1} \). Choice can also induce selection on match-specific survival \( Y_{i,j} - \bar{Y}_i \) if patients are more likely to accept an organs with high \( Y_{i,j} - \bar{Y}_i \).

Selection due to mortality occurs because longer-lived patients (high \( Y_{i,0} \)) are prioritized and have a higher chance of receiving a transplant. Moreover, such selection can also occur due to either time-to-treatment effects or correlation between \( \nu_{i,0} \) and \( \nu_{i,1} \). Our model also features mortality-induced selection because \( \tilde{J}_i \) only includes organs that arrive prior to \( Y_{i,0} \).

4 Instruments

We now describe and probe the two instruments described above. Section 5 will formally prove identification.
4.1 Conditionally Independent Potential Offers

The first instrument exploits randomness in the objects offered to an agent, relying on Assumption 2. We argue that this assumption is plausible in our setting on theoretical and empirical grounds. Our theoretical justification is based on the mechanism used to allocate deceased donor kidneys. Recall that $J_i$ is the sequence of offers to agent $i$ if the agent refuses all offers made to her and participated in the mechanism indefinitely. Thus, $J_i$ depends only on the kidneys that arrive after a patient registers on the waiting list, the decisions of other patients, and determinants of the agent’s priority. It does not depend on the decisions made by agent $i$ or her survival outcome. Our knowledge of the mechanism allows us to include determinants of each patient’s priority in $x_i$ as controls. The remaining variation in $J_i$ is only due to the stochastic arrival of organs and the decisions of agents other than $i$. It is plausible to assume that the arrival of organs is independent of $(\nu_i, \varepsilon_i)$ because it depends primarily on deaths in the local area. And, the decisions of other agents are independent of $(\nu_i, \varepsilon_i)$ in a natural equilibrium model of the waiting list (Agarwal et al., 2021).

We now empirically investigate these assumptions using a specific function of $J_i$. To do this, we construct a set of desirable donors that are achievable for patient $i$ in the two years following the patient’s registration. Specifically, we calculate whether patient $i$ would be placed above the patient in the 10th position on the list for a given donor. A patient is highly likely to receive an offer for an organ from such a donor because only 22.7% of deceased donors are offered to fewer than ten patients. We then calculate the number of donors that would satisfy this criteria for each patient in the two years following the patient’s registration date.

The variation in this variable comes from two sources: variation in the organs that arrived in the two years following patient $i$’s registration and variation in the patients on the waiting list and their decisions when the organ arrived. Our results use fixed effects to control for differences in a patient’s priority, geographical area, and time trends. Therefore, Assumption 2 needs to be satisfied conditional on these controls. The first source of variation is independent of $i$’s decisions because specific patients are not considered in organ donation decisions. Indeed, we cannot detect a correlation between patient characteristics and donor character-
istics conditional on the controls mentioned above (not reported due to space constraints, available on request). The second source of variation is also plausibly exogenous because, given a particular organ, other patients’ decisions should be independent of the selectivity and outcomes of patient \(i\). Consistent with this claim, Appendix Table D.5 shows that this measure varies substantially across patients and is not significantly correlated with the vast majority of patient characteristics.

Given this exclusion restriction, we establish relevance by showing that potential offers strongly influence whether or not a patient receives a transplant and also the type of organ transplanted. Columns (1) to (4) in Table 3 present estimates from linear probability models to examine the relationship between whether the transplanted organ is high quality (as measured by KDPI) and the number of potential top 10 offers from donors from the corresponding group. Columns (1) and (2) show that the number of offers in both donor categories are positively related to the probability of a transplant, whether or not we control for a rich set of patient characteristics. Columns (3) and (4) show that the type of organ transplanted is positively correlated with the number of potential offers from the corresponding type of donor. The F-statistics point to a strong first-stage relationship as they are much higher than the conventional cutoff of 10 used to assess whether an instrument is strong (Stock and Watson, 2012).

### 4.2 A Choice Shifter: Scarcity

Our second set of instruments are measures of scarcity \(z_i\) that alters an agent’s acceptance decisions \(D_{i,j}\) but are excluded from latent outcomes \(Y_{i,j}\). Patients who expect greater transplant opportunities in the future (lower scarcity) should be less willing to accept a given kidney than otherwise identical patients with fewer opportunities (higher scarcity). These instruments must be correlated with decisions but independent of latent outcomes. Formally, Assumption 1(i) requires that, conditional on \(x_i\), \((\nu_i, \varepsilon_i)\) is distributed independently of \(z_i\).

We construct two measures of scarcity. The first is a predictor of offers a patient can expect

\[10\] The only potential effect is if patient \(i\) accepts a kidney that would otherwise have been accepted by another patient who would been pivotal in determining whether \(i\) would be in the top ten positions for a different donor.
Table 3: Top 10 offers: First Stage

<table>
<thead>
<tr>
<th></th>
<th>Transplant Hazard Rate</th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Any Kidney</td>
<td>KDPI &lt;= 50%</td>
<td>KDPI &gt; 50% or Missing</td>
<td></td>
<td></td>
</tr>
<tr>
<td>log(1 + # Top 10 Offers in 2 Years)</td>
<td>Any Kidney</td>
<td>(1)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>KDPI &lt;= 50%</td>
<td>0.0322***</td>
<td>0.0334***</td>
<td>0.0439***</td>
<td>-0.0105***</td>
<td></td>
</tr>
<tr>
<td></td>
<td>(0.00441)</td>
<td>(0.00441)</td>
<td>(0.00306)</td>
<td>(0.00287)</td>
<td></td>
</tr>
<tr>
<td>KDPI &gt; 50% or Missing</td>
<td>0.0303***</td>
<td>0.0297***</td>
<td>-0.0128***</td>
<td>0.0425***</td>
<td></td>
</tr>
<tr>
<td></td>
<td>(0.00475)</td>
<td>(0.00478)</td>
<td>(0.00314)</td>
<td>(0.00294)</td>
<td></td>
</tr>
<tr>
<td>DSA FE, year FE, and blood type FE</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td></td>
</tr>
<tr>
<td>Control for Pediatric at Listing</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td></td>
</tr>
<tr>
<td>CPRA Category Controls</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td></td>
</tr>
<tr>
<td>Patient Characteristics</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td></td>
<td></td>
</tr>
<tr>
<td>F-statistic</td>
<td>93.20</td>
<td>92.23</td>
<td>108.0</td>
<td>130.6</td>
<td></td>
</tr>
<tr>
<td>Number of Observations</td>
<td>132715</td>
<td>131105</td>
<td>131105</td>
<td>131105</td>
<td></td>
</tr>
<tr>
<td>R-Squared</td>
<td>0.210</td>
<td>0.219</td>
<td>0.171</td>
<td>0.065</td>
<td></td>
</tr>
</tbody>
</table>

Notes: * p<0.05, ** p<0.01, *** p<0.001. The sample restricts to patients who registered between 2000 and 2008 because the instrument is calculated using offers in the two years post registration. All regressions control for donor service area (DSA) fixed effect, registration year fixed effect, blood type fixed effect, and priority characteristics (an indicator for pediatric at registration, and indicators for CPRA = 0, 20 <= CPRA < 80, CPRA >= 80, and CPRA missing at registration). Patient characteristics include an indicator for female; indicators for age 18-35, 35-50, and 50-65; indicators and linear controls for dialysis time 1-3, 3-5, 5-10, and >10 years; and an indicator for diabetes. Standard errors, clustered by DSA, registration year, and blood type are in parentheses. F-test tests against the null hypothesis that the coefficients on the instruments are zero.

in the future. Fix an offer for donor $j$ made to patient $i$ in the calendar quarter $t$. Consider the set of offers made in the four quarters before $t$ to other patients in a comparison group consisting of other patients with the same blood type as $i$ that registered in the same DSA as $i$. We count the subset of offers made to this group of patients when they had the same number of waiting time priority points as patient $i$ when she received the offer for donor $j$. The second is a predictor of donor supply, which is constructed analogously to the first but counts the number of unique donors in this set of offers.

Our analysis will include fixed effects for the DSA, blood-type, and the calendar year of the assignment. Therefore, both instruments exploit variation in scarcity in a patient’s DSA while controlling for secular trends. To assess balance, we investigated whether variations in our measures of scarcity significantly correlate with the characteristics of patients that register in a given year. Reassuringly, Table D.6 in the appendix shows that our scarcity
instruments are not significantly correlated with patient characteristics (age, diabetes, female, height, and weight). Our scarcity instruments are also uncorrelated with measures of donor quality (not reported due to space constraints, available on request). The threat to the instrument therefore needs to be a DSA-specific trend in scarcity that correlates with survival outcomes due to factors beyond patient or donor characteristics.

Table 4: Scarcity Instruments: First Stage

<table>
<thead>
<tr>
<th></th>
<th>(1)</th>
<th>(2)</th>
<th>(3)</th>
<th>(4)</th>
<th>(5)</th>
<th>(6)</th>
<th>(7)</th>
<th>(8)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Log(1 + No. Donors)</td>
<td>-0.0490***</td>
<td>-0.0479***</td>
<td>-0.0365***</td>
<td>-0.0360***</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>(0.00341)</td>
<td>(0.00338)</td>
<td>(0.00324)</td>
<td>(0.00323)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Log(1 + No. Offers)</td>
<td>-0.0536***</td>
<td>-0.0528***</td>
<td>-0.0439***</td>
<td>-0.0409***</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>(0.00185)</td>
<td>(0.00183)</td>
<td>(0.00183)</td>
<td>(0.00182)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Offer Year FE</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
</tr>
<tr>
<td>Priority Type FE</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
</tr>
<tr>
<td>DSA FE and blood type FE</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
</tr>
<tr>
<td>Years Waited at Offer FE</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
</tr>
<tr>
<td>Patient Characteristics</td>
<td>x</td>
<td>x</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Donor Characteristics</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
</tr>
<tr>
<td>Match Characteristics</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
</tr>
</tbody>
</table>

F-statistic 205.8 842.1 200.5 829.8 126.7 575.2 124.2 506.3
Number of Observations 912889 912761 912889 912761 900794 900669 900794 900669
R-Squared 0.166 0.172 0.169 0.174 0.263 0.233 0.265 0.268

Notes: * p<0.05, ** p<0.01, *** p<0.001. We use the first 100 offers from each donor between 2000 and 2009, and the dependent variable is acceptance of an offer. All regressions control for DSA fixed effect, blood type fixed effect, and a fixed effect for the number of years waited at the offer, and priority characteristics (an indicator for pediatric at registration, and indicators for CPRA = 0, 20 <= CPRA < 80, CPRA >= 80, and CPRA missing at registration). Patient characteristics include an indicator for female; indicators for age <=18, 18-35, 35-50, and 50-65; indicators and linear controls for dialysis time 1-3, 3-5, 5-10, >10 years; and an indicator for diabetes. Donor characteristics include linear age, indicators and linear controls for donor creatinine > 0.6 and >1.8, and indicators for diabetes, donation after cardiac death, and expanded criteria donor. Match characteristics include the number of Human Leukocyte Antigen (HLA) mismatches via indicators for 0 HLA mismatch, 0 and 1 DR antigen mismatch, identical blood type, local offers, and linear controls for (+) and (-) age difference, interactions between CPRA indicators and # HLA mismatches, donor age over 40 and pediatric patient, donor age over 55 and patient age 18-35, donor age over 60 and patient age 35-50, and donor age below 60 and patient age 50-65. Standard errors clustered by DSA, offer year, number of years waited at offer, and blood types in parentheses.

These instruments are relevant to decisions if they are correlated with beliefs about future offers. This hypothesis is based on the idea that transplant surgeons, who advise patients on decisions, are likely aware of the recent availability of kidneys. Columns (1) to (8) of Table 4 show the results from a linear probability model that regresses a dummy on whether an offer is accepted on the two measures of scarcity and a variety of controls. Both measures of scarcity are negatively correlated with acceptance. Columns (1) and (2) show that the
number of donors or number of offers to patients made in the past to the comparison group is negatively correlated with acceptance rates, controlling for patient priority type and fixed effects for DSA, allocation year, and years waited. These magnitudes are robust to adding an extensive set of controls for patient characteristics (columns 3 and 4), and not very sensitive to additional controls for donor and match-specific characteristics (columns 5 through 8). A residualized binscatter plot suggests that these relationships are monotonic (not reported due to space constraints, available on request).

5 Identification and Estimation

We now show that the instruments introduced in the previous section, $J_i$ and $z_i$, to identify target quantities described in Section 3. Our results condition on the patient type $x_i$ and omit it for simplicity of notation. We assume the analyst observes the organ types $q_j$, the choices $D_{i,j}$ if $i$ is offered $j$, the set of organs offered to each patient $\tilde{J}_i$ and the survival outcome for each patient. Our estimator does not require observing the potential offer sequence $J_i$ as long as Assumption 2 is satisfied.\footnote{Nonetheless, we can simulate $J_i$ in our context using knowledge of the mechanism and data on the offers made for each donor.}

The argument proceeds in three parts. First, we use standard arguments to show that variation in the offers received by a patient can be used to recover distributions of the outcomes conditional on certain sequences of choices. Second, we show that the choice model described in equation (3.3) is identified. Third, we combine continuous variation in scarcity with results from the first part to identify the effect of key unobservables on the distribution of outcomes. All proofs are in Appendix C.

5.1 Identifying Conditional Expected Outcomes

We start by using variation in offers. Given a realization of $J_i$, let $j(i,n)$ denote the $n$-th organ offered to $i$ and $q_i = (q_{j(i,1)}, q_{j(i,2)}, \ldots, q_{j(i,|q_i|)})$ be the sequence of offer-types offered to $i$. Our first result shows that variation in the offer-types can identify a conditional
average treatment effect for patients who accept the $n$–th offer.$^{12}$ Formally, let $N_i$ be one greater than the number of offers that $i$ rejects prior to the first acceptance, that is, $N_i = \min \{ n : D_{i,j(i,n)} = 1 \}$.

**Lemma 1.** Suppose that Assumptions 1 and 2 are satisfied. Fix $z$ and $q_i$. The marginal distributions of $Y_{i,j(i,n)}$ and $Y_{i,0}$ conditional on $N_i = n$, $z_i = z$ and $q_i$ are identified for all $n \leq |q_i|$ such that $P \left( N_i = n \mid q_i, z, Y_{i,0} \geq t_{j(i,n)} \right) > 0$, and $(q_{j(i,1)}, \ldots, q_{j(i,n)})$ and $(q_{j(i,1)}, \ldots, q_{j(i,n-1)})$ belong to the support of the distribution of offer-types induced by the distribution of $J_i$.

This result uses standard arguments (e.g. Imbens and Angrist, 1994) to identify counterfactual outcomes for patients who would have accepted and be assigned to the $n$–th organ offered. Since we directly observe the outcomes $Y_{i,j(i,n)}$ for patients (facing same scarcity level $z_i$ and receiving the same offer-type sequence $q_i$ as $i$) who are assigned to the $n$–th organ offered, the challenge is to estimate the unassigned outcomes for these patients. We do this by focusing on the set of unassigned patients who receive either exactly $n - 1$ or exactly $n$ offers with sequence of types $(q_{j(i,1)}, \ldots, q_{j(i,n-1)})$ and $(q_{j(i,1)}, \ldots, q_{j(i,n)})$. The former group contains patients with $N_i > n - 1$ whereas the latter group only contains patients with $N_i > n$, with weights given by the observed quantity $P \left( N_i = n \mid q_i, z, Y_{i,0} \geq t_{i,j(i,n)} \right)$. Monotonicity of the instrument is implied by our model because a patient cannot be assigned a kidney without receiving an offer.

This result allows us to evaluate the life-years gained in the observed assignment because the alternative is that all patients are unassigned. Identifying the distributions above, however, is not sufficient for evaluating their values under a counterfactual assignment of kidneys to patients because the distributions condition on $N_i = n$, and are therefore selected on $\nu_{i,D}$ and $\varepsilon_{i,j,D}$. We address this selection problem below.

### 5.2 Identifying the Choice Model

The next step uses the variation in offers identify the function $g_D(\cdot)$. To simplify exposition, focus on the case when $t_{i,j} = 0$ where $t_{i,j}$ denotes the time difference between donor arrival

$^{12}$Observe that our model and setting do not allow for always takers since a patient cannot be assigned an organ without receiving an offer for one.
and patient arrival. In this case, \( \nu_i \) is unselected due to survival while waiting on the list. Therefore, we normalize the marginal distributions of \( \nu_{i,D} \) and \( \varepsilon_{i,j,D} \) to be uniform and assume that \( z \) is supported in the unit interval. These normalizations are without further loss of generality because we have not placed restrictions on the functional form of \( g_D(\cdot) \). Because our empirical setting involves dynamic assignments, we prove results for the case when \( t_{i,j} > 0 \) and differs across \( j \) in appendix C.5.

We need to introduce some notation in order to develop our result. For each value of \( z \) and donor type \( q_j \), consider two sets of pairs \( (\nu_D, \varepsilon_D) \) such that one set yields \( g_D(q_j, z, \nu_D, \varepsilon_D) = 0 \) and the other yields \( g_D(q_j, z, \nu_D, \varepsilon_D) = 1 \). These two sets are separated by the function \( v(\varepsilon_D; q_j, z) = \sup\{\nu_D \in [0, 1] : g_D(q_j, z, \nu_D, \varepsilon_D) = 1\} \), where we adopt the convention that the supremum of the empty set is 0. Since \( \varepsilon_D \) and \( \nu_D \) are uniformly distributed, observe that \( v(\varepsilon_D; q_j, z) \) is equal to the fraction of patients that reject an offer of an organ with type \( q_j \) with probability at most \( \varepsilon_D \) when faced with scarcity \( z \). Therefore, identifying the function \( v(\varepsilon_D; q_j, z) \) is equivalent to identifying \( g_D(\cdot) \).

Our next result makes the following assumption on \( v(\cdot; q_j, z) \):

**Assumption 3.** For each \( q_j \) and \( z \), (i) the function \( v(\cdot; q_j, z) \) is differentiable, and (ii) for any \( \nu_D \in (0, 1) \) there exists \( \varepsilon_D \in (0, 1) \) such that \( v(\varepsilon_D; q_j, z) = \nu_D \).

The main restriction is in part (ii). It requires that there are no (interior) values of \( \nu_D \) for which the patient either accepts or rejects all organs of type \( q_j \) when faced with scarcity \( z \). In other words, there are high (low) enough match-specific shocks \( \varepsilon_D \) that would result in acceptance (rejection) of an offer, where the pivotal value of \( \varepsilon_D \) depends on \( \nu_D, q_j \) and \( z \). This condition would violated only if acceptance probabilities were degenerate for some \( q_j, z \) and \( \nu_D \in (0, 1) \). With this assumption, we show that variation in offers can be used to identify the function \( g_D(\cdot) \):

**Lemma 2.** Let \( q_j^n \) be a sequence composed by \( n \) offers of type \( q_j \) with \( t_{i,j} = 0 \), and let \( v_{n-1}(\cdot; q_j, z) \) be the \((n - 1)\)-st order Fourier-Legendre approximation of \( v(\cdot; q_j, z) \). If Assumptions 1 - 3 are satisfied, and \( q_j^n \) is in the support of the distribution of offer-types induced by \( J_i \), then \( v_{n-1}(\cdot; q_j, z) \) is identified for each \( z \in (0, 1) \) and \( q_j \). In particular, if the hypotheses hold for all \( n \), then \( v(\cdot; q_j, z) \) and therefore \( P(D_{i,j} = 1| \nu_{i,D} = \nu_D) \) is identified.
The main challenge is that there are two latent reasons that drive a patient’s decisions, namely \( \nu_{i,D} \) and \( \varepsilon_{i,j,D} \). We observe the probability \( P \left( D_{i,j(i,1)} = \ldots = D_{i,j(i,k)} = 0 \mid q_j^n, z \right) \) for all \( k \leq n \). Because \( v(\varepsilon; q_j, z) \) is the CDF of rejection probability across patients given \( q_j \) and \( z \), we can write

\[
P \left( D_{i,j(i,1)} = \ldots = D_{i,j(i,k)} = 0 \bigg| q_j^n, z \right) = \int_0^1 \varepsilon_k^k d v(\varepsilon; q_j, z).
\]

Therefore, the quantity \( P \left( D_{i,j(i,1)} = \ldots = D_{i,j(i,k)} = 0 \bigg| q_j^n, z \right) \) is the \( k \)-th moment of a random variable with cumulative distribution function \( v(\cdot; q_j, z) \). Learning the function \( v(\cdot; q_j, z) \) is therefore equivalent to the well-known Hausdorff moment problem (Casella and Berger, 2002) because we know the moments if we interpret \( v(\cdot; q_j, z) \) as a CDF. This can be done if an infinite number of moments are known.

In fact, our result is stronger: we show that data with finite \( n \) is informative even without variation in the number of offers because \( v(\cdot) \) can be well-approximated by observing decisions from a given sequence of offer-types \( q_j^n \). This follows because the moments described above determine the \( n \)-th order Fourier-Legendre approximation of \( v(\cdot) \), which converges to the true function \( v(\cdot; q_j, z) \) in the \( L^2 \) norm as \( n \) becomes large.

### 5.3 Identifying Selection on Unobservables

Next, we turn our attention to identifying the components that determine selection on unobservables using an additional regularity assumption:

**Assumption 4.** (i) For each \( z \in (0, 1) \) and \( q_j \), the derivative \( v'(\cdot; q_j, z) = \frac{\partial}{\partial \varepsilon_D} v(\cdot; q_j, z) \) is a continuous, cube-integrable, and strictly positive function of \( \varepsilon_D \in (0, 1) \).

(ii) For each \( \varepsilon_D \) and \( q_j \), the functions \( E[Y_{i,0} \mid \nu_D] \) and \( E[Y_{i,j} \mid \nu_D, \varepsilon_{i,j,D} \geq \varepsilon_D, q_j] \) are continuous in \( \nu_D \), and the first four moments of \( Y_{i,0} \) and \( Y_{i,j} \) exist.

The first part strengthens the differentiability of \( v(\varepsilon; q_j, z) \) imposed in Assumption 3 by requiring a strictly positive in \( L^3([0, 1]) \). Given the interpretation of \( v(\cdot) \) above, observe that \( v'(\cdot; q_j, z) \) is the density function of the distribution of the probability with which a patient rejects an offer of an organ with type \( q_j \). Therefore, we require that this density function is
bounded and is non-zero for all interior values of $\varepsilon_D$ and $z$. The second part imposes weak regularity assumptions on conditional moments of $Y_{i,0}$ and $Y_{i,j}$, where expectation is taken over $\nu_{i,0}$ and $(\nu_{i,1}, \varepsilon_{i,j,1})$ respectively.

Our main result shows identification of the expected values of $Y_{i,0}$ and $Y_{i,j}$ given $\nu_{i,D}$ and $\varepsilon_{i,j,D}$. The result also implies identification of the analogous quantities for any bounded transformation $\psi(\cdot)$ of $Y_{i,0}$ and $Y_{i,j}$, thereby implying identification of their marginal distributions.

**Theorem 1.** Suppose that Assumption 4 and the hypotheses for Lemma 2 hold for all $n$. Then, the quantities $E[Y_{i,0}|\nu_{i,D} = \nu_D]$ and $E[Y_{i,j}|\nu_{i,D} = \nu_D, \varepsilon_{i,j,D} \geq \varepsilon_D]$ are identified for all $\varepsilon_D \in (0, 1)$ and $\nu_D \in (0, 1)$ such that there exists $z$ in the support of its distribution with $\nu_D = \nu(\varepsilon_D; q_j, z)$.

Thus, the expected value of outcomes conditional on values of selectivity and idiosyncratic preferences is identified. We sketch the argument for $E[Y_{i,0}|\nu_{i,D}]$ since the intuition for identifying $E[Y_{i,j}|\nu_{i,D}, \varepsilon_{i,j,D} \geq \varepsilon_D]$ is similar in spirit. The proof begins by using results in Lemma 1 to identify the conditional expectations given scarcity $z$, offer-types and $N_i$. Next, we use the identification results for $v(\cdot)$ and arguments in Lemma 2 to recover the objects of interest. For example, Lemma 1 implies that $E[Y_{i,0} \times 1 \{T_i = 0\}|q_j^k, z_i] = \int_0^1 E[Y_{i,0}|\nu_D = v(\varepsilon_D; z_i, q_j)] \varepsilon_D^k dv(\varepsilon_D; z_i, q_j)$.

If we observe this quantity for all $k \leq n$, then we can recover the $n$–th order Fourier-Legendre approximation of $E[Y_{i,0}|\nu_D = v(\varepsilon_D; q_j, z)] v'(\varepsilon_D; q_j, z)$ when viewed as a function of $\varepsilon_D$, which converges uniformly to the true function in Cesàro mean (Talenti, 1986; Freud, 1971). Finally, since $v'(\varepsilon_D; q_j, z) > 0$ and bounded and the function $v(\varepsilon_D; q_j, z)$ is identified (Lemma 2), we can identify $E[Y_{i,0}|\nu_D]$ for all $\nu_D \in (0, 1)$ if we can find values of $z$ and $\varepsilon_D$ such that $v(\varepsilon_D; q_j, z) = \nu_D$.

This last step resembles strategies in Heckman and Vytlacil (2005); Lewbel (2007); Heckman

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13One qualitative difference is that identifying $E[Y_{i,0}|\nu_D]$ allows us to use variation in either $z$ or $\varepsilon_D$ to trace-out $\nu_D$, whereas the result for $E[Y_{i,j}|\nu_D, \varepsilon_{i,j,D} \geq \varepsilon_D]$ must condition on $\varepsilon_D$. 
and Navarro (2007) whereby a continuous instrument is used to “trace-out” the expected values of potential outcomes conditional on an unobservable. The scarcity instrument $z$ does this by changing the set of $(\nu_D, \varepsilon_D)$ whose treatment status changes in response to the offer instrument. Two differences are worth noting. First, our scarcity instrument is not treatment-specific because the discrete offer instrument generates variation in treatment assignments (c.f. Heckman and Navarro, 2007; Hull, 2018, for example). Our assumption that $\nu_{i,D}$ does not vary across $j$ allows us to use an instrument that varies only across patients $i$ but is fixed across $j$. Second, we do not use “identification at infinity” arguments as values of $z$ need not push choice probabilities to degenerate values that obviate the selection problem. Specifically, $E[Y_{i,0} | \nu_{i,D} = \nu_D]$ and $E[Y_{i,j} | \nu_{i,D} = \nu_D, \varepsilon_{i,j,D} \geq \varepsilon_D]$ are identified as long as we observe values of $z$ such that $\nu_D = v(\varepsilon_D; q_j, z)$. As is common, identification of $E[Y_{i,0}]$ and $E[Y_{i,j}]$ will require full support of $v(\varepsilon_D; q_j, z)$ for fixed $\varepsilon_D$ and $q_j$.

The results in Lemma 2 and Theorem 1 use data from the case when organs arrive at the same time as the patient ($t_{i,j} = 0$). Extending our results to the case when $t_{i,j} > 0$ and differs across $j$ introduces two issues. First is the direct effect of time to treatment, which can be captured by including the patient’s registration date and organ’s arrival date in $x_i$ and $q_j$. The second issue, which is the main challenge, is that the distribution of $\nu_{i,D}$ conditional on waiting until $t_{i,j}$ is no longer unselected.

Our extension in Appendix C.5 addresses these issues and implies identification of the marginal distributions and survival hazard functions of $Y_{i,0}$ and $Y_{i,j}$ (Theorem 2). As in generalized Roy models more broadly, the joint distribution of outcomes is not identified. Thus, we cannot attribute the effect of waiting time $t_{i,j}$ on $Y_{i,j}$ to either time-to-treatment or to correlation between survival outcomes. We ignore this distinction because it is not relevant for evaluating outcomes under counterfactual assignments.

5.4 Estimation

Although our results above show non-parametric identification, directly estimating these quantities is challenging for several reasons. First, we wish to incorporate rich observed and unobserved heterogeneity governing both choices and outcomes. These include patient-
specific, donor-specific, match-specific and time-to-treatment effects. Second, we observe only censored versions of our outcome, complicating a non-parametric analysis. Finally, we would like to incorporate correlations between discrete choices and these censored outcomes. To solve these challenges, we employ a Gibbs’ sampling technique to estimate a parametrized version of equations (3.1) – (3.3):\(^{14}\)

\[ y_{i,0} = B(Y_{i,0}; \rho_0) = x_i \beta_x + \nu_{i,0} \]  

\[ y_{i,j} = B(Y_{i,j}; \rho_1) = \chi(x_i, q_j) \alpha_{x,q} + \alpha_{\eta_j} + \nu_{i,1} + \varepsilon_{i,j,1} \]  

\[ D_{i,j} = 1 \{ \chi(x_i, q_j) \gamma_{x,q} + z_i \gamma_z + \eta_j - \nu_{i,D} + \varepsilon_{i,j,D} > 0 \}, \]  

where \( Y_{i,0} \) is survival since registration without a transplant; \( Y_{i,j} \) is survival since transplantation if patient \( i \) is transplanted organ \( j \); \( B(\cdot; \rho) \) denotes a Box-Cox transformation of the argument with parameter \( \rho \) (Box and Cox, 1964);\(^ {15} \) \( \chi(x_i, q_j) \) is a flexible function of patient observables \( x_i \) and organ observables \( q_j \); \( \eta_j \) is distributed \( \mathcal{N}(0, \sigma^2_\eta) \) with the parameter \( \sigma^2_\eta \) to be estimated; \( \varepsilon_{i,j} = (\varepsilon_{i,j,D}, \varepsilon_{i,j,1})' \) is distributed \( \mathcal{N}(0, \Sigma_\varepsilon) \) where \( \Sigma_{\varepsilon,11} \) is normalized to 1; and \( \nu_i \) is a mean-zero multi-variate normal with a distribution induced by the following factor structure, which is without loss of generality:

\[ \nu_{i,1} = \delta_{1,D} \nu_{i,D} + \nu_{i,f} \]  

\[ \nu_{i,0} = \delta_{0,D} \nu_{i,D} + \delta_{0,f} \nu_{i,f} + \tilde{\nu}_{i,0}, \]  

where \( \nu_{i,D}, \nu_{i,f} \) and \( \tilde{\nu}_{i,0} \) are independently distributed mean-zero normal random variables with variances to be estimated.

This empirical model maps the patient and kidney types into characteristic space, which reduces the number of parameters. It includes \( \eta_j \), which represents unobserved heterogeneity in organ quality due to characteristics observed by patients and surgeons but not included

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\(^{14}\)It is common to use functional form restrictions that are stronger than those necessary for identification when estimating a model that involves selection due to choices and several types of treatments (see Geweke et al., 2003; Hull, 2018, for example).

\(^{15}\)Formally, \( B(Y; \rho) = \frac{Y^{\rho} - 1}{\rho} \). In the special case when \( \rho = 0 \), \( B(Y; \rho) = \log Y \). We set \( \rho \) by comparing an estimated survival curve using the non-parametric Kaplan-Meier estimator to those implied by assuming that \( B(Y; \rho) \) is normally distributed.
in the empirical specifications. We include this term because it may be empirically im-
portant although our our identification results do not, strictly speaking, cover this case.\textsuperscript{16} Table
D.7, column 5, shows that our headline results using a model that excludes this term are
qualitatively similar.

This choice of functional form is motivated by several considerations. First, we wish to allow
for correlations between \( \nu_{i,0} \), \( \nu_{i,1} \), and \( \nu_{i,D} \) and between \( \varepsilon_{i,j,1} \) and \( \varepsilon_{i,j,D} \). For example, the
factor \( \nu_{i,f} \) captures the component of a patient’s unobserved frailty that is not correlated
with decisions. Second, decision are binary, suggesting the use of probit choice models.
These two considerations direct us to use multivariate normals to model the distributions
of \( \nu_{i} \) and \( \varepsilon_{i,j} \). Third, the parametrization allows us to handle censored data and also fit the
shape of the survival curve. Box-Cox transformations yield a tractable likelihood function
while generalizing the functional form (see Spitzer 1982, for example). We hold the Box-Cox
transformation parameters \( \rho_{0} \) and \( \rho_{1} \) fixed and conduct robustness analysis to alternative
choices (see Table D.7).

Directly computing and maximizing the likelihood of this model is difficult because each
patient’s data involves decisions over many donors as well as (potentially censored) survival
outcomes. Computing this likelihood requires integrating a nonlinear function over a high
dimensional space. Instead, we estimate the parameters of the model using a Gibbs’ sampler
(McCulloch and Rossi, 1994; Geweke et al., 2003; Gelman et al., 2014). This method generates
a sequence of draws of the model’s parameters, collected in \( \theta \), and the latent variables
\( \nu_{i} \), \( \varepsilon_{i,j} \), and \( \eta_{j} \) given the parameters from their respective posterior distributions. Our chosen
parametrization is amenable to this approach because the latent variables can be partitioned
so that each group has a posterior distribution given the draws of the other groups that can
be solved in closed form. Details on the method are provided in Appendix B.1. Based on
the Bernstein-von-Mises Theorem (see van der Vaart, 2000, Theorem 10.1), we interpret our
estimator as equivalent to maximum likelihood.

\textsuperscript{16}While formal analysis is left to future research, we conjecture that identification results can be obtained
based on an analogy to non-linear measurement error models (Hu and Schennach, 2008) because each donor
has two kidneys, suggesting that dependence between acceptances of a given donor’s first and second kidney
and the associated survival outcomes can be used for identification.
6 Survival and Choice Estimates

Table 5 present estimates for survival without and with a transplant, and the probability of acceptance in panels A, B and C respectively (detailed estimates are available on request). Our specifications contain a rich set of patient and donor covariates to capture medical history and match quality, including characteristics used in the leading models for predicting pre- and post-transplant survival for patients with kidney failure (see Wolfe et al., 2008, for example) as well as determinants of patient priority. Survival estimates show the marginal half-life effects associated with select characteristics. Effects are shown for a one standard deviation increase in a continuous characteristic or a unit change in an indicator.

We present estimates from three different specifications. The first specification only relies on offer randomness and does not employ the scarcity instruments (columns 1). This specification assumes that $\nu_{i,D}$, $\nu_{i,0}$ and $\nu_{i,1}$, and $\varepsilon_{i,j,D}$ and $\varepsilon_{i,j,1}$ are mutually independent. The second specification, which is our preferred one, includes the number of past donors as the scarcity instrument (columns 2). To assess robustness, we estimate a third specification with our past offers instrument (columns 3). Table D.7 in the appendix shows robustness of our headline findings to numerous variations.

**Survival:** Proxies for baseline patient health predict survival both with and without a transplant. A patient who is older, diabetic, or on dialysis at registration has a significantly shorter half-life both with and without a transplant, with effects that are slightly larger effects for post-transplant survival. For example, a diabetic patient’s half-life with and without transplant is lower than a non-diabetic patient by 2.99 and 1.36 years respectively.

Measures of donor quality, waiting time, and tissue-type similarity also predict post-transplant survival, but donor characteristics have lower estimated effects as compared to tissue-type matching and patient characteristics. For example, a donor with a history of hypertension results in a lower half-life by 0.34 years, which is much smaller than the effects on patient characteristics described above. Receiving a kidney with a perfect tissue-type match has a large effect on half-life, consistent with a lower likelihood of an immune responses.

**Choice:** Measures of donor quality and match-specific benefits are also positively correlated with acceptance. Patients are significantly more likely to accept kidney offers from younger
Table 5: Survival and Choice Estimates

<table>
<thead>
<tr>
<th>Patient Characteristics</th>
<th>Panel A: Survival without Transplant</th>
<th>Panel B: Survival with Transplant</th>
<th>Panel C: Acceptance Model</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>(1)</td>
<td>(2)</td>
<td>(3)</td>
</tr>
<tr>
<td>Diabetic</td>
<td>-1.380</td>
<td>-1.361</td>
<td>-1.361</td>
</tr>
<tr>
<td></td>
<td>(0.030)</td>
<td>(0.030)</td>
<td>(0.030)</td>
</tr>
<tr>
<td>On Dialysis at Registration</td>
<td>-1.019</td>
<td>-1.013</td>
<td>-1.013</td>
</tr>
<tr>
<td></td>
<td>(0.042)</td>
<td>(0.041)</td>
<td>(0.041)</td>
</tr>
<tr>
<td>Age at Registration</td>
<td>-1.070</td>
<td>-1.060</td>
<td>-1.060</td>
</tr>
<tr>
<td></td>
<td>(0.025)</td>
<td>(0.025)</td>
<td>(0.025)</td>
</tr>
<tr>
<td>Donor Characteristics</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age &lt; 18</td>
<td>1.595</td>
<td>1.604</td>
<td>1.647</td>
</tr>
<tr>
<td></td>
<td>(0.906)</td>
<td>(0.916)</td>
<td>(0.916)</td>
</tr>
<tr>
<td>Age 18-35</td>
<td>-0.267</td>
<td>-0.282</td>
<td>-0.249</td>
</tr>
<tr>
<td></td>
<td>(0.973)</td>
<td>(0.981)</td>
<td>(0.980)</td>
</tr>
<tr>
<td>Age 50+</td>
<td>3.383</td>
<td>3.381</td>
<td>3.294</td>
</tr>
<tr>
<td></td>
<td>(2.243)</td>
<td>(2.252)</td>
<td>(2.241)</td>
</tr>
<tr>
<td>Cause of Death - Head Trauma</td>
<td>0.662</td>
<td>0.665</td>
<td>0.691</td>
</tr>
<tr>
<td></td>
<td>(0.313)</td>
<td>(0.316)</td>
<td>(0.314)</td>
</tr>
<tr>
<td>Expanded Criteria Donor (ECD)</td>
<td>-0.622</td>
<td>-0.623</td>
<td>-0.655</td>
</tr>
<tr>
<td></td>
<td>(0.184)</td>
<td>(0.199)</td>
<td>(0.197)</td>
</tr>
<tr>
<td>History of Hypertension</td>
<td>-0.340</td>
<td>-0.342</td>
<td>-0.357</td>
</tr>
<tr>
<td></td>
<td>(0.122)</td>
<td>(0.124)</td>
<td>(0.123)</td>
</tr>
<tr>
<td>Unobservable (ηj)</td>
<td>0.107</td>
<td>0.181</td>
<td>0.000</td>
</tr>
<tr>
<td></td>
<td>(0.183)</td>
<td>(0.177)</td>
<td>(0.008)</td>
</tr>
<tr>
<td>Offer Characteristics</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Perfect Tissue Type Match</td>
<td>2.272</td>
<td>2.269</td>
<td>2.322</td>
</tr>
<tr>
<td></td>
<td>(0.944)</td>
<td>(0.959)</td>
<td>(0.954)</td>
</tr>
<tr>
<td>Log Waiting Time (Years)</td>
<td>-0.487</td>
<td>-0.543</td>
<td>-0.539</td>
</tr>
<tr>
<td></td>
<td>(0.062)</td>
<td>(0.168)</td>
<td>(0.161)</td>
</tr>
<tr>
<td>Scarcity</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Log(#Past Donors)</td>
<td></td>
<td></td>
<td>-0.010</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>(0.001)</td>
</tr>
<tr>
<td>Log(#Past Offers)</td>
<td></td>
<td></td>
<td>-0.020</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>(0.001)</td>
</tr>
</tbody>
</table>

Notes: Select estimates of the marginal effect on the probability of acceptance and half-life. Marginal effects are computed at the median value of observable covariates, integrating over the distribution of all unobservables. All effects are shown for a one standard deviation increase in each continuous covariate and a unit increase in each binary covariate. We generate 250000 draws and burn-in the first 50000 draws. All columns control for DSA fixed effects, blood type fixed effects, and registration year fixed effects. Other patient characteristics include dialysis time at registration, BMI at departure, patient serum albumin, and indicators for female, diabetic, CPRA=0, and prior transplant. Donor characteristics include indicators for other causes of death, expanded criteria donor, donation after cardiac death, male, and bins of creatinine levels. Other offer characteristics include indicators for 2 A, 2 B, 2 DR mismatches, not the same blood type but compatible, regional offer, local offer, and interactions between several patient and donor characteristics. Standard errors are in parentheses.
donors; donors who died of head trauma; donors without a history of hypertension; and donors with whom they have a perfect tissue-type match. Kidneys which have higher unobservable quality, $\eta_j$, are also more likely to be accepted, suggesting that decisions respond to information about the organ that is not perfectly captured by the observable characteristics.

The last two rows record the scarcity instruments’ effects on acceptance. Consistent with the results in Table 4, each instrument has a significant negative effect on the probability of acceptance. Other parameter estimates are similar across the instrumented specifications, suggesting that the choice between these two instruments is unlikely to be an important driver of our results.

A comparison of estimates across the panels indicate that many organ quality measures positively affect both choice and survival. Tissue-type match and donor death by head trauma are both strongly associated with both choice and survival. That said, the association is not perfect: organs from younger donors are more likely to be accepted even though the survival effects are not significant.

### Table 6: Correlation Table

<table>
<thead>
<tr>
<th></th>
<th>Panel A: Selectivity ($\nu_{i,D}$)</th>
<th>Panel B: Match value ($\varepsilon_{ij,D}$)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>(1)</td>
<td>(2)</td>
</tr>
<tr>
<td>Probability of Acceptance</td>
<td>-0.039 (0.001)</td>
<td>0.068 (0.001)</td>
</tr>
<tr>
<td>Post-Transplant Survival</td>
<td>0.008 (0.138)</td>
<td>0.022 (0.258)</td>
</tr>
<tr>
<td>Survival without a Transplant</td>
<td>0.330 (0.060)</td>
<td>0.323 (0.059)</td>
</tr>
</tbody>
</table>

Notes: Estimated effects of a one standard deviation increase in choice unobservables affects acceptance and survival probabilities. Survival durations are calculated using half-lives. Survival effects from changes in $\varepsilon_{ij,1}$ from a one standard deviation rise in $\varepsilon_{ij,D}$ from zero, given the estimated covariance between $\varepsilon_{ij,D}$ and $\varepsilon_{ij,1}$. Likewise, survival effects from changes in $\nu_{i,D}$ are computed using the expected changes in $\nu_{i,1}$ and $\nu_{i,0}$ from a one standard deviation increase in $\nu_{i,D}$ from zero, given the estimated covariances between $\nu_{i,D}$, $\nu_{i,1}$, and $\nu_{i,0}$. All effects are computed at the median value of observable covariates.

### Selection on Unobservables:

Our model measures the correlation between survival and choice induced by unobservable characteristics. Table 6 shows how a one standard deviation increase in $\nu_{i,D}$ (selectivity) and $\varepsilon_{ij,D}$ (match value) affects acceptance and survival. The selectivity...
effects are measured by computing the changes on $\nu_{i,0}$ and $\nu_{i,1}$ induced by their estimated correlation with $\nu_{i,D}$. Likewise, the correlation between $\varepsilon_{i,j,D}$ and $\varepsilon_{i,j,1}$ yields the effects of match value.

Selective patients typically survive longer without a transplant and benefit less from the typical transplant. A one standard deviation rise in selectivity lowers the probability of acceptance by 3.9 percentage points. This effect is of similar order as that of a kidney from a donor with a history of hypertension. Therefore, there is positive selection into treatment on the patient-specific component of survival benefits.

In contrast to selectivity, patient-donor specific factors do not induce significant selection via choices. While we estimate the covariance between $\varepsilon_{ij,D}$ and $\varepsilon_{ij,1}$ to be positive, the effect is not statistically significant.

7  Estimated LYFT

7.1  Calculating Life Years from Transplant (LYFT)

For each patient-donor pair, we compute the difference between the median survival time with a transplant and median survival time without a transplant, measured from the date of transplant. Specifically, for each pair $(i, j)$, we define LYFT conditional on a set of covariates $I_{i,j} = \{x_i, q_j, D_{ij}, \eta_j, \nu_{i,D}, \nu_{i,f}\}$ as follows:

$$LYFT (I_{i,j}) = M (Y_{i,j} | I_{i,j}, Y_{i,0} \geq t_{i,j}) - M (Y_{i,0} | I_{i,j}, Y_{i,j} \geq t_{i,j}), \quad (7.1)$$

where $M (Y | X)$ is the median of random variable $Y$ conditional on $X$ and $t_{i,j}$ is the time between patient $i$’s registration and the arrival of kidney $j$.\textsuperscript{17,18} Therefore, this measure accounts for selection on unobservables induced by the mechanism.

\textsuperscript{17}Some estimates of LYFT place a weight of 0.8 on life years without a functioning kidney to account for the lower quality of life (e.g. Wolfe et al., 2008). This quality-adjustment is arbitrary and is omitted in our specification.

\textsuperscript{18}We use a Gibbs’ sampler to compute the expectation of $LYFT (I_{ij})$ by drawing $\eta_j$, $\nu_{i,D}$, and $\nu_{i,f}$ from their conditional distributions given observables, decisions, and observed survival outcomes. We fix the parameters at the estimate $\hat{\theta}$, generate 200,000 draws, burn-in the first half, and use every 1,000-th draw.
7.2 Life Years from Transplant in the Mechanism

Table 7 presents the average estimated LYFT over all realized transplants. The first row accounts for patient- and kidney-specific unobservables and the decision to accept. The second row conditions only on patient and donor observables, integrating \( LYFT(\mathbf{I}_{i,j}) \) over \( D_{ij}, \eta_j, \nu_{i,D}, \nu_{i,f} \). The average LYFT from our preferred specification is 8.64 years (column 2). Ignoring selection on unobservables yields a lower estimate of 7.94, suggesting positive selection on LYFT into transplantation based on unobservables. The specification that does not use scarcity instruments yields biased estimates, about two-thirds of a year less than our preferred estimate (column 1). This suggests observational methods used in the medical literature may underestimate gains from transplantation.

Table 7: Life-Years from Transplant

<table>
<thead>
<tr>
<th></th>
<th>(1)</th>
<th>(2)</th>
<th>(3)</th>
<th>(4)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Life Years from Transplant</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Accounting for Unobservables</td>
<td>7.93</td>
<td>8.64</td>
<td>8.63</td>
<td>8.63</td>
</tr>
<tr>
<td></td>
<td>(0.28)</td>
<td>(0.39)</td>
<td>(0.33)</td>
<td>(0.33)</td>
</tr>
<tr>
<td>Observables Only</td>
<td>7.90</td>
<td>7.94</td>
<td>7.83</td>
<td>7.71</td>
</tr>
<tr>
<td></td>
<td>(0.28)</td>
<td>(0.49)</td>
<td>(0.47)</td>
<td>(0.50)</td>
</tr>
<tr>
<td>Untransplanted Survival</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>All Patients</td>
<td>7.01</td>
<td>6.95</td>
<td>6.95</td>
<td>6.86</td>
</tr>
<tr>
<td></td>
<td>(0.14)</td>
<td>(0.17)</td>
<td>(0.15)</td>
<td>(0.18)</td>
</tr>
<tr>
<td>Transplanted Patients</td>
<td>7.34</td>
<td>7.21</td>
<td>7.21</td>
<td>7.17</td>
</tr>
<tr>
<td></td>
<td>(0.16)</td>
<td>(0.20)</td>
<td>(0.18)</td>
<td>(0.21)</td>
</tr>
<tr>
<td>Post-Transplant Survival</td>
<td>15.28</td>
<td>15.84</td>
<td>15.84</td>
<td>15.80</td>
</tr>
<tr>
<td></td>
<td>(0.28)</td>
<td>(0.38)</td>
<td>(0.33)</td>
<td>(0.29)</td>
</tr>
</tbody>
</table>

Notes: Life years from transplant and survival durations presented in the table are calculated using half-lives. Future donors (offers) is defined as the number of donors (offers) in the next 4 quarters (see Table 4 for detailed definition). All columns control for patient, donor, and offer characteristics, which are defined analogously as in Table 5 Panel B and Table 5. Standard errors are in parentheses.

The second pair of rows report average survival without a transplant, separately, for all patients and the subset of patients who received a transplant. Across specifications, the untransplanted survival for patients who are transplanted is higher than for patients who are not. Thus, choices and the mechanism result in selection on untransplanted survival into transplantation.
7.3 Selection and LYFT

The selection on LYFT and untransplanted survival reported in Table 7 above can take place along two margins: the patients who are transplanted and the kidneys to which they are matched. We further investigates these sources below.

Patient Selection: There are strong complementarities between baseline health and transplantation. Figure 2(a) presents the joint density of (median) untransplanted survival and the average (median) LYFT from all potential donors for each patient, overlayed with a bin-scatter plot. LYFT and untransplanted survival are strongly positively correlated. Patients who are expected to live longer without a transplant also have the largest life-year gains.

When combined with the observation in Table 7 that transplanted patients have higher baseline survival, this complementarily suggests that patients who are transplanted likely have higher LYFT due to selection on baseline health. In addition, there may be patient selection into transplantation from choice and from the priorities in the mechanism.

The overall selection into transplantation is presented in Figure 2(b), which shows the distribution of predicted LYFT across all potential transplants. This distribution is shifted
(a) Transplanted Survival from Potential and Realized Donors

Figure 3: Patient-Kidney Matching

to the right for transplanted patients, with an average that is 1.2 years higher. Thus, the mechanism selects patients with larger average LYFT and that some of this selection comes from transplanting patients who are relatively healthy at baseline.

Patient-Kidney Matching: The realized allocation also matches patients to kidneys from which they receive greater survival benefits as compared to the average kidney. Figure 3(a) plots the joint distribution of LYFT from the realized donor for a transplanted patient against LYFT from all potential donors. The binscatter is below the 45-degree line, indicating that the realized transplants generate greater than average LYFT for a patient. This finding that matches are selected advantageously complements the finding that the mechanism selects patients with higher than average gains from transplantation.

Part of this advantageous matching comes from the correlation of patients’ acceptance decisions with LYFT. Figure 3(b) presents binscatter plots of kidney-patient acceptance probability against LYFT for all potential transplants, showing two features. First, transplanted patients have a higher predicted probability of acceptance than untransplanted patients. Second, the predicted probability of accepting an offer increases in LYFT. As our estimates
suggest, patients are more likely to accept kidneys with greater life-year benefits (based on both observable and unobservable characteristics). In sum, we find that the allocation matches kidneys to patients based on LYFT and that at least some of this selection is induced by choices in the mechanism.

**Patient Selection vs. Rematching:** Figure 3(a) also provides insight into which of these two assignment margins dominates. The heterogeneity in survival across patients swamps the heterogeneity across donors within a patient. In fact, a decomposition of the total variance in LYFT into patient-specific, donor-specific, and match-specific components (the last being the remainder) shows that the patient-specific component contributes to 6.58 years of the standard deviation in LYFT. The donor-specific and match-specific components are much smaller, accounting for 1.04 years and 0.48 years, respectively.

Thus, the potential for increasing life-years by improving the match between patients and donors without changing which patients are transplanted (rematching) is limited. Distributional constraints may therefore limit the potential gains from improved matching. In particular, maximizing life-year gains may mean reallocating transplants away from the most urgent cases towards patients with longer expected survival without a transplant, pointing to a potential trade-off between efficiency and worst-off prioritarianism for the sickest.

### 8 Potential for Further Increasing LYFT

We now evaluate the performance of the mechanism on LYFT and quantify the importance of patient selection versus rematching. We compare the average LYFT achieved by the realized assignment to benchmarks, ranging from a random assignment to one that maximizes LYFT. Extending patients’ lives is a prima facie objective of the medical profession. But, this objective may raise distributional concerns or conflict with principles of allocation discussed in medical ethics. We highlight these trade-offs by comparing the types of patients who are transplanted under the benchmarks.

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19To verify this point, we regressed the expected value of $LYFT_{ij}$ conditional on $\{x_i, q_j, \eta_j, \nu_i,D, \nu_i,f\}$ on the probability of acceptance given these same covariates, controlling for patient- and donor-specific fixed effects. A one standard deviation increase in the match-specific component of LYFT raises the probability of acceptance by 0.59 percent.
We focus on our preferred specification and, to ease computation, we restrict the sample to the set of patients who registered in 2005. Our results are not sensitive to choice of instrument; varying the Box-Cox shape parameters of our specification; omitting donor unobserved heterogeneity $\eta_j$; or including time between organ extraction and transplantation (see Table D.7).

### 8.1 Comparison with Benchmark Assignments

We start with two extremal benchmarks, random assignment and optimal assignment:

**Random assignment** is simulated by successively assigning patients to kidneys at random from the set of feasible kidneys. Feasibility requires that the patient must be biologically compatible and the kidney should arrive between the patient’s registration date and a simulated death date without a transplant. The latter is drawn from that patient’s predicted survival distribution.

**Optimal assignment** is computed by maximizing the total LYFT from all transplants. This benchmark considers an omniscient planner who knows $x_i$, $q_j$, $\nu_{i,D}$, $\nu_{i,f}$, $\eta_j$, each patient’s arrival and untransplanted death dates, and each kidney’s arrival date. The planner computes LYFT conditional on these characteristics and can dictate assignments. Only feasible transplants are allowed and each patient can receive at most one transplant.\(^{20}\)

The comparison to the random assignment measures the increase in LYFT achieved by the mechanism. Both selecting patients and advantageously matching kidneys to patients drives the difference. To decompose these sources, we evaluate an alternative that allocates kidneys randomly among transplanted patients:

The **random amongst transplanted** assignment is simulated by re-assigning transplanted patients to a kidney at random from the set of feasible kidneys.

The increase in LYFT due to the mechanism results from both the mechanism’s priority rules for kidney offers and the choices made by patients on the waiting list. To separate

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\(^{20}\) Call the $s$-th simulated draw for each patient/donor pair $LYFT_{ij}^s$. Let $a_{ij} = 1$ if $i$ is assigned $j$ and $a_{ij} = 0$ otherwise. Let $c_{ij} = 1$ if $i$ is feasible for $j$ and $c_{ij} = 0$ otherwise. We solve the problem $\max_a \sum_{i,j} a_{ij}LYFT_{ij}^s$ subject to $a_{ij} (1 - c_{ij}) = 0$, $\sum_i a_{ij} \leq k_j$, where $k_j$ is the number of kidneys available from donor $j$, and $\sum_j a_{ij} \leq 1$. 

the gains achieved due to the mechanism’s priority structure from the gains from choice, we evaluate a counterfactual assignment with no patient choice.

The **no choice assignment** is computed by assigning each kidney to the patient with the highest priority among untransplanted patients. Offers cannot be rejected by patients.

Comparing the realized assignment to the optimal assignment bounds the maximum theoretical gain in LYFT that could be achieved by any mechanism. As with the comparison of the realized and random assignments, this gain is driven both by selecting patients and matching patients to kidneys. To decompose these sources, we evaluate an alternative that only reassigns kidneys among transplanted patients:

The **optimal rematching** assignment maximizes the total LYFT using the same information set as in the optimal assignment. In addition to the feasibility constraint, a patient in this assignment can be transplanted only if she was transplanted in the data.

Optimal assignment uses information about factors that induce selection, $\nu_{i,D}$, $\nu_{i,f}$, and $\eta_j$. However, the first two factors may not be observed by the planner and may be hard to elicit in a mechanism. Similarly, $\eta_j$ may be difficult to condition on. These observations motivate a benchmark that uses only observable information:

The **optimal assignment based on observables** is computed by maximizing the total expected LYFT conditional on $x_i$ and $q_j$ by assigning patients to a feasible kidney. $^21$The solution describes the highest possible LYFT that can be achieved by a planner who can dictate assignments based on this information.

Figure 4 presents the results. The average LYFT for the realized assignment amongst patients who registered in 2005 is 8.78 years. This is analogous to the results in Table 7 above.

The realized assignment achieves a 0.92 year increase in average LYFT over random assignment. Both selecting patients and matching patients to kidneys are important: random amongst transplanted yields an increase of only 4.4 months. The remainder of the gain is due to patient-kidney matching.

Patient choice is a key contributor to the mechanism’s gains in LYFT over random assign-

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$^21$For tractibility, we assume the planner has foresight on when patients arrive and depart and when kidneys arrive. Relaxing foresight would require solving a dynamic assignment problem with uncertainty about the future.
Figure 4: LYFT Under Counterfactual Assignments

The no choice assignment results in similar LYFT as the random assignment. Thus, if the priority rules we used to dictate assignments, then only 15.8% of the LYFT increase in the realized assignment would be achieved.\textsuperscript{22}

Although the mechanism does better than a random assignment, there is significant scope for further increasing LYFT. The average LYFT under the theoretical upper bound given by the optimal assignment is 5.1 years higher than the LYFT achieved in the realized assignment. Bias in estimates based on observational studies would miss the potential for these gains.\textsuperscript{23}

A significant fraction, 14.4%, of the increase can be achieved by rematching patients and kidneys while keeping the set of transplanted patients fixed. However, consistent with Figure 3(a), most of the improvements in the optimal allocation come from changing the set of patients who are transplanted.

Finally, a planner who can dictate assignments using the observable characteristics could achieve a significant fraction, but not all, of the potential increase. These observables have been either used to determine priority or considered explicitly in proposed reforms. The average LYFT under the optimal assignment based on observables is 10.48 years. Although less than the theoretical maximum, it is about 1.7 years more than the average LYFT

\textsuperscript{22}We also simulated the no choice assignment using priorities in place after 2014 and found similar results on LYFT.

\textsuperscript{23}A proposed assignment based on maximizing LYFT that uses the specification which omits scarcity instruments yields an average of 11.05 years when evaluated using our preferred specification.
achieved by the mechanism. Therefore, in principle, average LYFT could be substantially raised by targeting transplants using observed characteristics rather than choices.

### 8.2 The Planner’s Dilemma

Achieving the increases in LYFT described above would require changing the set of patients who are transplanted. We now show that this change shifts the demographics and health conditions of transplanted patients, creating a potential barrier due to distributional considerations or the desire to prioritize patient urgency.

The LYFT increases, from random assignment to the mechanism and finally to the optimal solutions, require transplanting relatively healthy patients. Table 8 presents the distribution of patient age, health, and untransplanted survival for patients transplanted under the random assignment, the no choice assignment, the actual assignment, and the optimal assignment. Patients transplanted under the realized assignment are healthier than average – younger, less likely to be diabetic, less likely to be on dialysis, and have longer untransplanted survival. Similarly, transplanted patients are also healthier under the optimal assignment than under the realized assignment. The optimal assignments also reallocates kidneys towards racial/ethnic minority patients who have higher LYFT on average than white patients.
Comparing the realized assignment and the no choice assignment illustrates the role of choice in increasing LYFT. The existing priority rules target transplants between patients and donors with no HLA mismatches. The fraction of zero-mismatch assignments is lower under the realized and optimal assignments as compared to no-choice. Yet, choice also dramatically changes the selection of who is transplanted towards patients with high LYFT by shifting the age distribution towards younger patients and those with longer untransplanted survival. Therefore, while patients benefit from kidneys with a perfect tissue-type match, reassigning kidneys to the right set of patients without perfect tissue-type matches can increase LYFT. These shifts highlight the distributional effects of optimizing LYFT – the realized outcome increases LYFT by selecting younger, healthier patients to transplant. The optimal assignment exacerbates these distributional changes. These results are driven by the strong correlation between survival with and without a transplant, illustrated in Figure 2(b). Thus, in order to maximize LYFT given the scarcity of kidneys available, the planner must transplant healthier patients and let sicker patients go untransplanted.\(^{24}\)

This stark trade-off represents a dilemma. Society may have a moral imperative to prioritize sick patients who may soon die, as done in deceased donor liver allocation. But some medical ethicists discard this principle when faced with scarcity, arguing instead for maximizing total survival or treating people equally (random assignment) (see Persad et al., 2009). Our results suggest that these two principles are in conflict for kidney allocation, with utilitarian principles also raising concerns about discrimination based on patient characteristics such as age and concerns about increased inequality in patient survival.

9 Conclusion

A hitherto overlooked goal in designing assignment mechanisms is to produce matches that improve associated outcomes such as patient survival or student achievement. We take a first step towards an empirical analysis that incorporates these outcomes by studying the LYFT generated using the pool of deceased donor kidneys. To do this, we show how to

\(^{24}\)Indeed, an assignment that transplants the sicker patients first (as measured by \(Y_{i,0}\)) results in an LYFT of 5.67 years.
use variation generated in an assignment mechanism to estimate and identify a model that jointly considers choices and outcomes.

We find that the waitlist mechanism used to allocate deceased donor kidneys does better than a random allocation but leaves much scope for improvement. The mechanism transplants patients for whom life would be extended longer, as compared to the average patient, and matches them to more suitable than average kidneys. However, average LYFT could be boosted by several years. The potential economic value of realizing these gains is enormous. Aldy and Viscusi (2007) place the value of a statistical life year at $300,000. At even half this value and ignoring costs savings on dialysis, the potential benefits from 1 more year of life from the approximately 13,000 deceased donor kidneys transplanted each year accrues to almost $2 billion per year.

Achieving most of these gains will require confronting important distributional considerations because survival without a transplant is a strong predictor of life-year gains. Therefore, the planner faces a dilemma between transplanting the sick and transplanting those for whom life will be extended the longest.

This work opens several avenues for further research. First, our approach avoids micro-founding the choice model at the cost of evaluating benchmark assignments rather than the equilibria of alternative mechanisms. This leaves counterfactual selection in an equilibrium model to future work. Second, we focus on an aggregate measure of LYFT that abstracts away from distributional or non-utilitarian ethical considerations. Formalizing these considerations and incorporating them into the design problem could yield a valuable tool for policymaking. The underlying trade-offs are particularly central to designing mechanisms when outcomes are the target, and deserve further research in other contexts as well.

References


**Stegall, Mark D., Peter G. Stock, Kenneth Andreoni, John J. Friedewald, and Alan B. Leichtman**, “Why do we have the kidney allocation system we have today? A history of the 2014 kidney allocation system,” *Human Immunology*, jan 2017, 78 (1), 4–8.


A Data Appendix

A.1 Obtaining Original Data Files

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

We will retain copies of the data until permitted by our Data Use Agreement with the Organ Procurement and Transplantation Network (OPTN). Further, we plan to send OPTN a copy of our replication archive if and when we are required to destroy our dataset. Researchers interested in using our dataset should directly contact OPTN to obtain permission: https://optn.transplant.hrsa.gov/data/request-data/. We are happy to provide copies of our data to researchers with permission and a data use agreement with the OPTN.

A.2 Data Description

Our data on patients, donors, transplants, and offers are based on information submitted to the Organ Procurement and Transplant Network (OPTN) by its members. The main datasets are the Potential Transplant Recipient (PTR) dataset and the Standard Transplantation Analysis and Research (STAR) dataset.

The PTR dataset contains offers made to patients on the deceased donor kidney waitlist that were not automatically rejected based on pre-specified criteria. Information includes identifiers for the donor, patient, and patient history record that generated the offer; the order in which the offers were made; each patient’s acceptance decision; and if the offer was not accepted, a reason of rejection. Each offer record also contains certain characteristics of
the match, including the number of tissue type mismatches.

The STAR dataset contains separate files on deceased donor characteristics, patient histories, patient characteristics and transplant outcomes, and follow-up data, which are collected at six months and then annually, for kidney transplants. The patient and donor characteristics from these datasets are used to estimate our models of acceptance behavior and patient survival. The patient characteristics and transplant outcomes dataset contains patient death information. For patients who received a transplant through the deceased kidney donor waitlist, the follow-up dataset records whether the patient is still alive at the follow-up point. This information allows us to compute a survival duration for each patient. UNOS also provided supplemental information, including the ordering of distinct match runs conducted for the same deceased donor; the transplant centers of donors and patients in our dataset; and dates of birth for pediatric candidates, who joined the waitlist before turning 18 years of age.

The data contain identifiers that allow us to link the offer and acceptance data to patient and donor characteristics. Each deceased donor has a unique identifier. Similarly, each patient registration generates a unique patient waitlist identifier. Because patients may move to different transplant centers or be registered in multiple centers simultaneously, some individual patients have multiple waitlist identifiers. For this study, we focus on the earliest registration of each patient. The follow-up data contain a unique identifier for each transplant, allowing us to connect the follow-up information to each transplanted patient. The patient history file contains a unique patient record identifier corresponding to a particular state of the patient on the waitlist, including the patient’s CPRA, activity status, and pre-set screening criteria. Each offer in the PTR dataset contains the identifiers for the donor, the patient registration, and the patient history record that were used in the match run. When appropriate, we de-duplicate offers so that each patient can receive at most one offer from each donor.

A.3 Sample Selection

We consider the first waiting period for patients who were actively waiting for a deceased donor kidney between January 1, 2000 and December 31, 2010. This restriction is to avoid
selection arising from patients that remain on the list at the beginning of the sample period. We omit patients who received a living donor transplant as their first transplant or were cross-registered for other organs simultaneously. The outcomes for these patients are likely very different from patients who receive only a kidney from a deceased donor. Most patients that can receive a living donor receive one within the first year of registration and would prefer such a transplant to a deceased donor transplant. The latter restriction is made to focus on a more homogeneous group of patients.

In addition, we made a number of other more minor adjustments to work with a more cohesive sample of patients. The number of patients that survive each step of the sample selection process is described in Table A.1.

A small minority of patients are simultaneously registered in multiple donor service areas, indicating that multiple listings and moves are not common. Our analysis keeps only one waitlist record from each patient. If the patient received a kidney transplant through the deceased donor waitlist before December 31, 2015, we keep the waitlist record with the earliest transplant date; if the patient remained untransplanted as of December 31, 2015, we keep the waitlist record with the earliest registration date.

Next, we exclude a small number of patients who received a prior kidney transplant to focus on survival effects from the first transplant. We also exclude patients removed for administrative reasons. These are patients who were listed on the waitlist by error, who departed because a transplant took place but no transplant was recorded in the STAR dataset, and who could no longer be contacted while waiting on the waitlist. These departure reasons are recorded in the STAR patient and the transplant outcome dataset.

Then, we keep the waitlist records with registration dates between January 1, 2000 and December 31, 2010 because we do not have data on offers prior to 2000. For example, an untransplanted patient active between 2000 and 2010 may not be included in the final sample because said patient’s first waitlist registration is before 2000. This step amounts to be one of the largest cuts.

Finally, we exclude patients who received a transplant through non-standard allocations.

\[\text{We use transplant data through December 31, 2015 to be consistent with the sample period during which we observe patient survival.}\]
rules. This can occur, for example, if the donor is an armed service member; if the donor specified a particular recipient (directed donation); if there is a medical emergency or expedited placement attempt; if the kidney is not offered due to operational issue. We identify these cases by analyzing the PTR data as a large number of offers will be bypassed with a code indicating one of these reasons. In some cases, there is also text specifying specific circumstances justifying a rejection, which we parse to identify invalid offers in cases where the refusal code does not provide a specific reason.

Table A.1: Sample Selection: Patients

<table>
<thead>
<tr>
<th>Step Description</th>
<th>Number of Patients</th>
<th>Number of Wait List Records</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patient’s first waiting period that intersects the period 2000-2010</td>
<td>308,370</td>
<td>372,681</td>
</tr>
<tr>
<td>Exclude patients who received living donor transplants in their first waiting period</td>
<td>241,209</td>
<td>295,075</td>
</tr>
<tr>
<td>Exclude patients were waiting for other organs in their first waiting period</td>
<td>213,685</td>
<td>244,580</td>
</tr>
<tr>
<td>Keep one kidney waitlist record for each patient</td>
<td>213,685</td>
<td>213,685</td>
</tr>
<tr>
<td>Patients with multiple waitlist records</td>
<td>32,191</td>
<td>32,191</td>
</tr>
<tr>
<td>Patients with single waitlist record</td>
<td>181,494</td>
<td>181,494</td>
</tr>
<tr>
<td>Exclude patients who had a previous kidney transplant</td>
<td>212,258</td>
<td>-</td>
</tr>
<tr>
<td>Exclude patients with administrative waitlist removal reason</td>
<td>207,316</td>
<td>-</td>
</tr>
<tr>
<td>Restrict to patients whose remaining waitlist registration is between 2000 and 2010</td>
<td>178,944</td>
<td>-</td>
</tr>
<tr>
<td>Exclude patients who received non-standard kidney allocations</td>
<td>175,518</td>
<td>-</td>
</tr>
</tbody>
</table>

Our sample of deceased kidney donors comes from the intersection of the STAR deceased donor dataset and the PTR dataset. These are deceased donors whose kidneys were allocated between January 1, 2000 and December 31, 2010 to patients on the waitlist. We further exclude donors allocated using non-standard rules and restrict to donors who were offered to patients in the sample.

Table A.2 details the number of donors that survive each filter. The largest cuts come from the last step. This is because the priority for waiting time implies that many offers are only given to patients that registered prior to 2000.

Table A.2: Sample Selection: Donors

<table>
<thead>
<tr>
<th>Step Description</th>
<th>Number of Donors</th>
</tr>
</thead>
<tbody>
<tr>
<td>Deceased donors offered to any kidney waitlist patients between 2000 and 2010</td>
<td>71,738</td>
</tr>
<tr>
<td>Exclude deceased donors offered through non-standard kidney allocations</td>
<td>67,993</td>
</tr>
<tr>
<td>Restrict to deceased donors offered to patients in the sample</td>
<td>61,453</td>
</tr>
</tbody>
</table>
We consider a sample of offers made between January 1, 2000 and December 31, 2010 that could have resulted in transplants between our patient and donor samples. The PTR dataset includes records of all initial patient contacts and patients skipped due to administrative reasons irrespective of whether an offer was made. This happens mainly for three reasons. First, some patients that were contacted have lower priority than the patients that accepted and were transplanted the kidneys from a donor. In this case, we determine the cutoff point for each donor, and exclude all offers made after the cutoff. Second, some match runs were abandoned due to logistical reasons, and were re-run. We only keep the offers from the last match run for a donor. Third, in some cases, the PTR dataset records administrative or logistical reasons for skipping patients in the offer sequence. This can occur, for example, if the kidney has antigens that would result in an immune response; a patient was bypassed due to logistical reasons; or if the kidney does not meet the patient’s minimum criteria. We also exclude non-responsive offers, for example, because either the surgeon or the patient is unavailable or because the patient is temporarily inactive/unsuitable for transplantation. Finally, we restrict to offers made to the patients in the sample. This step cuts the offer sample by 41% because many offers are made to patients that were not in our sample, for example, to patients that registered prior to 2000. Table A.3 describes how we arrive at the final sample of offers.

Table A.3: Sample Selection: Offers

<table>
<thead>
<tr>
<th>Description</th>
<th>Number of Offers</th>
</tr>
</thead>
<tbody>
<tr>
<td>Offers made between 2000 and 2010 from donors in the sample</td>
<td>14,888,539</td>
</tr>
<tr>
<td>Exclude non-responsive offers</td>
<td>14,239,214</td>
</tr>
<tr>
<td>Restrict to offers made to patients in the sample</td>
<td>8,444,106</td>
</tr>
</tbody>
</table>

A.4 Patient Survival

The patient characteristics and transplant outcomes dataset collects patient death dates from the waitlist record and periodically from the social security master file. In a small minority of cases, death dates are inconsistent across multiple waitlist records for a patient, in which case we assume that earlier death dates take precedence over later ones. Transplant dates
and death dates are truncated on December 31, 2015, because death records after this date are inconsistently populated. For patients who received a transplant or died after December 31, 2015, we treat them as untransplanted or alive, respectively, as of December 31, 2015. Among 175518 patients in the sample, we observe death dates before December 31, 2015 for 80168 of them. Of these, 55476 are untransplanted patients and 24692 are transplanted. Patients from whom we do not observe death are censored. The censoring rules differ for transplanted and untransplanted patients. For transplanted patients, we censor on the date of the second transplant if a second transplant took place before December 31, 2015; on the day after transplant if there is no follow-up information for the patient corresponding to the transplant; on the date when the patient is lost to follow-up if the patient is lost to follow-up prior to December 31, 2015; and on December 31, 2015 if the patient is known to be alive as of December 31, 2015. For untransplanted patients, we censor on December 31, 2015 if the patient is known to be alive as of December 31, 2015; and on the date when the patient exits the waitlist if no death date is available and the exit day is prior to December 31, 2015. Table A.4 presents a break down of censor reasons and their corresponding censor dates for the patient sample. Nearly one half of the patient sample is uncensored, and among censored patients, the vast majority (73%) are censored on December 31, 2015. Since December 31, 2015 is an exogenously determined date, patients censored on the date should be similar to uncensored patients in terms of potential outcomes.

Table A.4: Censor Reason

<table>
<thead>
<tr>
<th>Censor Reason</th>
<th>Censor Date</th>
<th># Patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>Transplanted Patients</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Retransplant before Dec 31, 2015</td>
<td>Retransplant date</td>
<td>3,581</td>
</tr>
<tr>
<td>No follow-up information</td>
<td>One day after transplant</td>
<td>979</td>
</tr>
<tr>
<td>Lost to follow-up before Dec 31, 2015</td>
<td>Date lost to follow up</td>
<td>5,856</td>
</tr>
<tr>
<td>Known to be alive as of Dec 31, 2015</td>
<td>December 31, 2015</td>
<td>57,215</td>
</tr>
<tr>
<td>Untransplanted Patients</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Known to be alive as of Dec 31, 2015</td>
<td>December 31, 2015</td>
<td>12,370</td>
</tr>
<tr>
<td>No death date and depart the waitlist before Dec 31, 2015</td>
<td>Date departing waitlist</td>
<td>15,349</td>
</tr>
</tbody>
</table>
B Estimation Appendix

B.1 Gibbs’ Sampler

Recall that our model is given by

\[ y_{i0} = B(Y_{i0}; \rho_0) = x_i \beta_x + \nu_{i,0} \]

\[ y_{ij} = B(Y_{ij}; \rho_1) = \chi(x_i, q_j) \alpha_{x,q} + \alpha_q \eta_j + \nu_{i,1} + \varepsilon_{ij,1} \]

\[ D_{ij} = 1 \{ y_{ij,D} = \chi(x_i, q_j) \gamma_{x,q} + z_i \gamma_z + \eta_j + \nu_{i,D} + \varepsilon_{ij,D} > 0 \} , \]

where we allow for \( \nu_i = (\nu_{i,D}, \nu_{i,1}, \nu_{i,2}) \sim \mathcal{N}(0, \Sigma_\nu) \) and \( \varepsilon_{ij} = (\varepsilon_{ij,1}, \varepsilon_{ij,D}) \sim \mathcal{N}(0, \Sigma_\varepsilon) \).

There are several challenges in estimating this model. First, we often observed censored values of \( y_{i0} \) and \( y_{ij} \). We perform a data augmentation step given the parameters and the censoring point to solve this issue. For \( y_{ij} \), the data augmentation step is necessary only in cases for which \( T_{ij} = 1 \).

Second, \( D_{ij} \) is a binary variable. As is standard in discrete choice models, we perform a data augmentation step to draw \( y_{ij,D} \) given the observed decisions. This step is necessary for the observed values of \( D_{ij} \).

Third, the model incorporates rich correlations between the different observations via \( \eta_j, \nu_i \) and \( \varepsilon_{ij} \). In particular, due to these terms, the covariance matrix between \( \{y_{i0}\}_i \{y_{ij}\}_{ij} \) and \( \{y_{ij,D}\}_{ij} \) conditional on the observables and the parameters does not have a simple block-diagonal structure that would allow us to compute simple posterior distributions. To solve this problem, we re-write these variables using a factor structure such that the posterior distribution of the parameters of each equation is conditionally independent of the others given the factors. Specifically, we rewrite \( \nu_i \) as

\[ \nu_{i,D} = f_{i,1} \]

\[ \nu_{i,f} = f_{i,2} \]

\[ \nu_{i,0} = \beta_{\nu_1} f_{i,1} + \beta_{\nu_2} f_{i,2} + \bar{\varepsilon}_{i0} \]
where \( f_{i,1}, f_{i,2} \) and \( \varepsilon_{i0} \) are each independently distributed mean-zero normal random variables with variances \( \sigma^2_1, \sigma^2_2 \) and \( \sigma^2_{\tilde{\varepsilon},0} \). This structure places no restrictions on the covariance matrix \( \Sigma_{\nu} \). Similarly, we write \( \varepsilon_{ij} \) as

\[
\varepsilon_{ij} = \alpha \varepsilon_{ij,1} + \tilde{\varepsilon}_{ij,1} = f_{ij,3} + \tilde{\varepsilon}_{ij,D}
\]

where \( f_{ij,3}, \tilde{\varepsilon}_{ij,1} \) and \( \tilde{\varepsilon}_{ij,D} \) are independently distributed mean-zero normal random variables with variances \( \sigma^2_3, \sigma^2_{\tilde{\varepsilon},1} \) and \( \sigma^2_{\tilde{\varepsilon},D} \). We normalize the variances \( \sigma^2_{\tilde{\varepsilon},0} \) and \( \sigma^2_{\tilde{\varepsilon},1} \) to 1. Finally, set

\[\eta_j = f_{j,4}\]

with variance \( \sigma^2_4 \). The main difference between \( f \) and \( \tilde{\varepsilon} \) is that it is sufficient to condition on the former in order to render the models above as conditionally independent.

Therefore, the parameters we are interested estimating in are the coefficients in each equation, \( \beta = (\beta_x, \beta_{\nu1}, \beta_{\nu2}), \alpha = (\alpha_{x,q}, \alpha_q, \alpha_{\nu1}, \alpha_\varepsilon), \gamma = (\gamma_x, \gamma_q, \gamma_\varepsilon) \), and the variances \( \sigma^2_{\tilde{\varepsilon},0} = V[\tilde{\varepsilon}_{i0}], \sigma^2_{\tilde{\varepsilon},1} = V[\tilde{\varepsilon}_{ij,1}] \) and \( \sigma^2_l = V[f_l] \) where \( l \in \{1, 2, 4\} \) is the \( l \)-th factor.

For simplicity of notation, we will collect the coefficients in the vector \( \theta \) and the standard deviations in the vector \( \sigma \), with \( \sigma_{\tilde{\varepsilon}} \) and \( \sigma_f \) denoting the sub-vectors for \( \tilde{\varepsilon} \) and \( f \) respectively. And, with some abuse of notation, we collect \( y_{i0}, y_{ij} \) and \( y_{ij,D} \) for all \( i \) and \( j \) in \( y \).

Following standard practice, we assume diffuse conjugate and independent priors for each of these parameters. Specifically, we model the priors \( \alpha, \beta \) and \( \gamma \) using a mean-zero independent normal distribution with variances equal to 1000 and the prior for the variances \( \sigma^2_{\tilde{\varepsilon},0}, \sigma^2_{\tilde{\varepsilon},1} \) and \( \sigma_l^2 \) using independent inverse-Wishart distributions with parameters \((3, 3)\). These priors are diffuse; thus, they have a negligible impact on our estimates.

The Gibbs’ sampler starts with an initial draw \( y^0, \theta^0, \sigma^0 \) and \( f^0 \) and generates a chain of length \( K \) by iterating through the following steps for each \( k \in \{0, \ldots, K - 1\} \):

1. **Data Augmentation**: Sample \( y^k_{i0}, y^k_{ij} \) for censored observations and \( y^k_{ij,D} \) for observed decisions given \( \theta^k, \sigma^k \) and \( f^k \) from truncated normal distributions.
2. **Sample Coefficients**: Sample $\theta^{k+1}$ given $y^{k+1}$, $f^k$, the standard deviations $\sigma^k$ and the prior distribution from a multi-variate normal distribution.

3. **Sample Variances**: Sample $\sigma_{\varepsilon,0}^{2,k+1}$ and $\sigma_{\varepsilon,1}^{2,k+1}$ given $y^{k+1}$, $f^k$, the parameters $\theta^{k+1}$ and the prior distribution from an inverse-Wishart distribution.

4. **Sample Factors**: For each $l \in \{1, 2, 3, 4\}$, sample $f^k_{*,l}$ given $y^{k+1}$, the parameters $\theta^{k+1}$, $\sigma^k_{\varepsilon,0}$, $\sigma^k_{\varepsilon,1}$, $\sigma^k_f$, and the remaining factors $f^k_{*,0}, \ldots, f^k_{*,l-1}$ and $f^k_{*,l+1}, \ldots, f^k_4$.

5. **Sample Factor Variances**: Sample $\sigma_l^{2,k+1}$ for $l \in \{1, 2, 4\}$ given $f^k$ and the prior distribution from an inverse-Wishart distribution.

We draw a chain of length $K = 200,000$ and burn 50,000 draws to allow the chain to convergence. We only keep one every 10 draws to save some computation time and reduce the autocorrelation in the resulting chain. To diagnose the potential for non-convergence, we visually inspect the chains and, as recommended in Gelman et al. (2014), we also ensure that the potential scale reduction factor is below 1.1 for each of the parameters. The distributions in each step can be solved for in closed-form as detailed below:

1. Conditional distributions for $y_{i0}$, $y_{ij}$ and $y_{ij,D}$ given $\theta$, $f$ and $\sigma$:

   (a) For each $i, j$ pair with $D_{ij}$ observed, the distribution of $y_{ij,D}$ conditional on $\gamma$, $f$ and $D_{ij}$ is a truncated normal with mean $E[y_{ij,D}|\gamma, f_{ij}]$ and unit standard deviation. The distribution is truncated below at 0 if $D_{ij} = 1$ and above at 0 otherwise.

   (b) For each $i$ such that $y_{i0}$ is censored, the distribution of $y_{i0}$ conditional on $\beta$ and $f$ is a one-sided truncated normal with mean $E[y_{i0}|\beta, f_{i1}, f_{i2}]$ and standard deviation $\sigma_{\varepsilon,0}$. The distribution of $y_{i0}$ is truncated below at the censoring duration.

   (c) For each observed transplant with $y_{ij}$ censored, the distribution of $y_{ij}$ conditional on $\alpha^k$, $f^k$ is a one-sided truncated normal with mean $E[y_{ij}|\alpha, f]$ and standard deviation $\sigma_{\varepsilon,1}$. The distribution of $y_{ij}$ is truncated below at the censoring duration.

2. Posterior distributions of the co-efficients $\alpha$, $\beta$ and $\gamma$ given $y$, $f$, $\sigma$ and the priors.

Since $y_{i0}$, $y_{ij}$ and $y_{ij,D}$ are mutually independent conditional on $f$, the parameters $\alpha$, $\beta$ and $\gamma$ given $y$, $f$, $\sigma$ and the priors.
$\beta$ and $\gamma$ are each co-efficients in a linear regression model with normally distributed errors. Therefore, the posterior distributions of each of these terms is given by a multivariate normal distribution with closed-form means and variances (Gelman et al., 2014, Chapter 14.2).

3. Posterior distributions of $\sigma^2_{\tilde{\varepsilon}, 0}$ and $\sigma^2_{\tilde{\varepsilon}, 1}$ given $y, f, \sigma$ and the priors. As above, $y_{i0}, y_{ij}$ are mutually independent conditional on $f$. Therefore, the distributions of $\sigma^2_{\tilde{\varepsilon}, 0}$ and $\sigma^2_{\tilde{\varepsilon}, 1}$ are inverse-Wishart with parameters given in Chapter 14.2 of Gelman et al. (2014).

4. Posterior distributions of $f$ given $y, \theta$ and $\sigma$:

   (a) The distribution of $f_{i, 1}$ conditions on the residual

$$f_{i, 1} + \frac{1}{\beta_{\nu 1}} \tilde{\varepsilon}_{i0} = \frac{1}{\beta_{\nu 1}} (y_{i0} - (x_i \beta x + \beta_{\nu 2} f_{i, 2}))$$

and $\sigma_1$ throughout; on the residual

$$f_{i, 1} + \tilde{\varepsilon}_{ij, D} = y_{ij, D} - (\chi (x_i, q_j) \gamma_{x, q} + z_i \gamma_z + \eta_j + f_{ij, 3})$$

for all $j$ such that $D_{ij}$ is observed; and on the residual

$$f_{i, 1} + \frac{1}{\alpha_{\nu 1}} \tilde{\varepsilon}_{ij, 1} = \frac{1}{\alpha_{\nu 1}} (y_{ij} - (\chi (x_i, q_j) \alpha_{x, q} + \alpha_{\eta} \eta_j + \alpha_{f} f_{ij, 3}))$$

if $T_{ij} = 1$. These residuals have prior mean zero and variances $\sigma^2_1 + \frac{\sigma^2_{\tilde{\varepsilon}, 0}}{\beta_{\nu 1}}, \sigma^2_1 + \sigma^2_{\tilde{\varepsilon}, 1}$ and $\sigma^2_1 + \frac{\sigma^2_{\tilde{\varepsilon}, 1}}{\alpha_{\nu 1}}$ respectively. The posterior mean of $f_{i, 1}$ is the precision-weighted average of the residuals corresponding to $i$, and the posterior variance is the inverse of the sum of $\sigma^2_1$ and the precisions of each residual.

(b) The distribution of $f_{i, 2}$ is analogous, where we condition on $\sigma_2$ and the residual

$$\frac{1}{\beta_{\nu 2}} (y_{i0} - (x_i \beta x + \beta_{\nu 1} f_{i, 1}))$$

throughout; and on the residual $y_{ij} - (\chi (x_i, q_j) \alpha_{x, q} + \alpha_{\eta} \eta_j + \alpha_{f} f_{ij, 1})$ if $T_{ij} = 1$. 

(c) The distribution of $f_{ij,3}$ is analogous, where we condition on $\alpha_\epsilon$ throughout; on

$$y_{ij,D} - (\chi(x_i, q_j)\gamma_{x,q} + z_i\gamma_z + \eta_j + f_{i,1})$$

for all $j$ such that $D_{ij}$ is observed; and on

$$\frac{1}{\alpha_\epsilon} \left( y_{ij} - \left( \chi(x_i, q_j)\alpha_{x,q} + \alpha_\eta \eta_j + f_{i,2} \right) \right)$$

if $T_{ij} = 1$. Note that $\sigma_3$ is normalized to 1.

d) The distribution of $f_{j,4}$ is analogous, where we condition on $\sigma_4$ throughout; on

$$y_{ij,D} - (\chi(x_i, q_j)\gamma_{x,q} + z_i\gamma_z + f_{i,1} + f_{ij,3})$$

for all $i$ such that $D_{ij}$ is observed; and on

$$\frac{1}{\alpha_n} \left( y_{ij} - \chi(x_i, q_j)\alpha_{x,q} + f_{i,2} + \alpha_\epsilon f_{ij,3} \right)$$

if $T_{ij} = 1$.

5. The variances $\sigma_l^2$ for $l \in \{1, 2, 4\}$ follow an inverse-Wishart distributions given the prior and respectively, $\{f_{i,1}\}$, $\{f_{i,2}\}$ and $\{f_{j,4}\}$.

C Theoretical Appendix

C.1 Proof of Lemma 1

For simplicity of notation, denote $q_n = (q_{j(i,1)}, \ldots, q_{j(i,n)})$ and $q_{n-1} = (q_{j(i,1)}, \ldots, q_{j(i,n-1)})$, which are truncated from $q_i$ to the first $n$ and $n - 1$ offers respectively. Assumption 2 implies that $P(N_i = n|q_i, z) = P(N_i = n|q_i, z, Y_{i,0} \geq t_{i,j(i,n)})$ because $J_i$ is orthogonal to outcomes. The right hand side is observed since it is the probability that the first offer an agent facing scarcity $z$, with untransplanted survival at least $t_{i,j(i,n)}$ accepts is the $n$-th offer. The conditioning on untreated survival being larger than $t_{i,j(i,n)}$ ensures that we observe the first $n$ offers made to $i$. Similarly, if $P(N_i = n|q_i, z, Y_{i,0} \geq t_{i,j(i,n)}) > 0$, then Assumption 2 implies that for any bounded function $\psi(\cdot)$,

$$E \left[ \psi \left( Y_{i,j(i,n)} \right) \middle| N_i = n, q_i, z \right] = E \left[ \psi \left( Y_{i,j(i,n)} \right) \middle| N_i = n, q_i, z, Y_{i,0} \geq t_{i,j(i,n)} \right]$$

is identified. Because $\psi(\cdot)$ is bounded, the expectations above are finite. Therefore, it remains to show that $E \left[ \psi \left( Y_{i,0} \right) \middle| N_i = n, q_i, z \right]$ is identified. First, use Assumption 2 to rewrite $E \left[ \psi \left( Y_{i,0} \right) \middle| N_i = n, q_i, z \right] P(N_i = n|q_i, z) = E \left[ \psi \left( Y_{i,0} \right) \middle| N_i = n, q_n, z \right] P(N_i = n|q_n, z)$, where the right hand side conditions on the subset of agents who receive exactly $n$ offers with the sequence of types given by $q_n$. By assumption, this sequences of offer types is in
the support of the sequence of offer types induced by $J_i$. Now, re-write

$$E [\psi (Y_{i,0}) | N_i = n, q_i, z] P (N_i = n | q_i, z)$$

$$= E [\psi (Y_{i,0}) | N_i = n, q_n, z, Y_{i,0} \geq t_{i,j(i,n)}] P (N_i = n | q_n, z, Y_{i,0} \geq t_{i,j(i,n)})$$

$$= E [\psi (Y_{i,0}) | N_i > n - 1, q_n, z, Y_{i,0} \geq t_{i,j(i,n)}] P (N_i > n - 1 | q_n, z, Y_{i,0} \geq t_{i,j(i,n)})$$

$$- E [\psi (Y_{i,0}) | N_i > n, q_n, z, Y_{i,0} \geq t_{i,j(i,n)}] P (N_i > n | q_n, z, Y_{i,0} \geq t_{i,j(i,n)})$$

$$= E [\psi (Y_{i,0}) | N_i > n - 1, q_{n-1}, z, Y_{i,0} \geq t_{i,j(i,n)}] P (N_i > n - 1 | q_{n-1}, z, Y_{i,0} \geq t_{i,j(i,n)})$$

$$- E [\psi (Y_{i,0}) | N_i > n, q_n, z, Y_{i,0} \geq t_{i,j(i,n)}] P (N_i > n | q_n, z, Y_{i,0} \geq t_{i,j(i,n)}) .$$

The first equality follows from the same arguments as above showing that $P (N_i = n | q_i, z) = P (N_i = n | q_i, z, Y_{i,0} \geq t_{i,j(i,n)})$, the second equality follows from set inclusion and the last from Assumption 2. This quantities in the last expression are observed by focussing on the subset of patients that receive the sequence of offer types $q_{n-1}$ and $q_n$.

Thus, $E [\psi (Y_{i,0}) | N_i = n, q_i, z] P (N_i = n | q_i, z)$ is identified. Since the second term equals $P (N_i = n | q_i, z, Y_{i,0} \geq t_{i,j(i,n)})$, which identified and strictly positive, $E [\psi (Y_{i,0}) | N_i = n, q_i, z]$ is identified. The marginal distributions of $Y_{i,0}$ and $Y_{i,j(i,n)}$ conditional on $N_i = n, q_i$ and $z$ are identified because the conditional expectations of $\psi (Y_{i,0})$ and $\psi (Y_{i,j(i,n)})$ are identified for any bounded function $\psi$.

### C.2 Proof of Lemma 2

For any $k \leq n$, Assumptions 1 and 2 imply that the observed probability that $D_{i,j(i,1)} = D_{i,j(i,2)} = \ldots = D_{i,j(i,k)} = 0$ can be re-written as follows:

$$P (D_{i,j(i,1)} = D_{i,j(i,2)} = \ldots = D_{i,j(i,k)} = 0 | q_j^n, z_i) = \int_0^1 \varepsilon^k_D d\varepsilon (\varepsilon_D; q_j, z_i).$$

Observe that $a_k = \int_0^1 \varepsilon^k_D d\varepsilon (\varepsilon_D; q_j, z_i)$ is identified for $k \in \{1, \ldots, n\}$. Moreover, Assumption 3 implies that $a_0 = \int_0^1 1 d\varepsilon (\varepsilon_D; q_j, z_i) = 1$. Therefore, to complete the proof, we need to show that $v_{n-1} (\cdot; q_j, z_i)$ is determined by the values of $a_k = \int_0^1 \varepsilon^k_D d\varepsilon (\varepsilon_D; q_j, z_i)$ for $k \leq n$ where $v_{n-1} (\cdot; q_j, z_i)$ is the $(n - 1)$-st order Fourier-Legendre approximation of $v (\cdot; q_j, z)$. In what
follows, we will drop conditioning on \( z_i \) and \( q_i^n \) for simplicity of notation.

To complete the proof, we write the co-efficients of \((n - 1)\)-st Fourier-Legendre series of \( v(\cdot) \) in terms of \( a_k \). Let \( \Gamma_m (x) \) be the \( m \)-th shifted Legendre Polynomial. Observe that each \( \Gamma_m (\cdot) \) is given by \( \Gamma_m (x) = \sum_{l=0}^{m} \gamma_{m,l} x^l \), with known co-efficients \( \gamma_{m,l} \).\(^{26}\) The \( m \)-th co-efficient in the (shifted) Fourier-Legendre series of \( v(x) \) is given by

\[
c_m = (2m + 1) \int_0^1 \Gamma_m (x) v(x) \, dx
\]

\[
= (2m + 1) \left( \int_0^1 \Gamma_m (x) \, dx - \int_0^1 \int_0^x \Gamma_m (y) \, dy \, dv(x) \right),
\]

where the second equality follows from integration by parts. Observe that \( \int_0^1 \Gamma_m (x) \, dx = \int_0^1 \Gamma_m (x) \Gamma_0 (x) \, dx = 0 \) for \( m > 0 \). Therefore, for \( m > 0 \),

\[
c_m = -(2m + 1) \int_0^1 \int_0^x \Gamma_m (y) \, dy \, dv(x) = -(2m + 1) \int_0^1 \sum_{l=0}^{m} \gamma_{m,l} \frac{1}{l+1} x^{l+1} dv(x)
\]

\[
= -(2m + 1) \sum_{l=0}^{m} \gamma_{m,l} \frac{1}{l+1} a_{l+1}.
\] \( \text{(C.1)} \)

And, finally, we have

\[
c_0 = \int_0^1 \Gamma_0 (x) v(x) \, dx = \int_0^1 v(x) \, dx = v(1) - \int_0^1 y \, dv(x),
\]

where the last equality follows from integration by parts. The term \( v(1) = 1 \) since \( v(\cdot) \) is non-decreasing with image \([0,1]\). Equations (C.1) and (C.2) imply that all \( c_m \) for \( m < n \) can be written in terms of the observed quantities \( a_0, \ldots, a_n \). Therefore, \( v_{n-1} (\cdot) \) is identified.

Let \( \tilde{\Gamma}_m (y) \) be the \( m \)-th unshifted Legendre Polynomial defined over \([-1,1]\) satisfying \( \tilde{\Gamma}_m (y) = \Gamma_m \left( \frac{y+1}{2} \right) \).\(^{27}\) The \((n - 1)\)-st order Fourier-Legendre approximation of \( \tilde{v}(y) = v \left( \frac{y+1}{2} \right) \) is \( \tilde{v}_{n-1} (y) = \sum_{k=0}^{n-1} \tilde{c}_n \tilde{\Gamma}_m (y) \) where, \( \tilde{c}_m = \frac{(2m+1)}{2} \int_{-1}^{1} \tilde{\Gamma}_m (y) \tilde{v} (y) \, dy = c_m \), where the last equality follows after a change of variables \( x = \frac{y+1}{2} \). Since the function \( \tilde{v} (\cdot) \) has a compact domain

\(^{26}\) The shifted Legendre-Polynomials on \([0,1]\) satisfy the orthogonality relationship \( \int_0^1 \Gamma_m (x) \Gamma_n (x) \, dx = \frac{1}{2m+1} \delta_{m,n} \), where \( \delta_{m,n} \) is the Kronecker delta. The first few polynomials are \( \Gamma_0 (x) = 1, \Gamma_1 (x) = 2x - 1, \Gamma_2 (x) = 6x^2 - 6x + 1 \).

\(^{27}\) The unshifted Legendre-Polynomials on \([-1,1]\) satisfy the orthogonality relationship \( \int_{-1}^{1} \Gamma_m (y) \Gamma_n (y) \, dy = \frac{2}{2m+1} \delta_{m,n} \), where \( \delta_{m,n} \) is the Kronecker delta. The first few polynomials are \( \tilde{\Gamma}_0 (y) = 1, \tilde{\Gamma}_1 (y) = y, \tilde{\Gamma}_2 (x) = \frac{1}{2} (3x^2 - 1) \).
and image, we have that \( f_1 \tilde{v}(y)^2 \, dy \) is bounded. Theorem 8.1 in Pollard (1947) shows that the Legendre polynomials form a basis in \( L^2([−1,1]) \), or equivalently, that \( \tilde{v}_n(y) \) converges in the \( L^2 \)-norm to \( \tilde{v}(y) \) as \( n \to \infty \). Therefore, \( \|v_{n-1}(\cdot) - v(\cdot)\|_2 \to 0 \) as \( n \to \infty \). Therefore, \( v(\cdot) \) is identified if the hypotheses of the Lemma are satisfied for all \( n \). Since \( v(\cdot q_j, z_i) \) is increasing in its argument, \( P(D_{i,j} = 1|\nu_i, D = v(\varepsilon_D; q_j, z_i); q_j, z_i) \) is identified.

### C.3 Preliminaries for Theorem 1

**Lemma 3.** Let \( f_n \) and \( g_n \) be sequences of functions such that \( f_n \to f \) and \( g_n \to g \) pointwise. Assume that \( f \) is continuous.

(i) If \( f_n \) converges to \( f \) uniformly in \([a,b]\) and \( g_n(x) \in (a,b) \) for all \( x \), then \( f_n(g_n(x)) \) converges to \( f(g(x)) \) for each \( x \) in the domain of \( g \).

(ii) If \( f_n \) and \( g_n \) respectively converge to \( f \) and \( g \) uniformly in \([a,b]\) and \( \inf_{x \in [a,b]} |g(x)| = k > 0 \), then \( \frac{f_n(x)}{g_n(x)} \) converges to \( \frac{f(x)}{g(x)} \) uniformly in \([a,b]\).

(iii) If \( f_n \) converges to \( f \) uniformly in \([a,b]\) and \( f \) is strictly increasing on \([a,b]\), and the function \( f_n^{-1}(y) \) is defined as \( \inf\{x : f_n(x) > y\} \), then for all \( x \in (a,b) \), \( f_n^{-1}(f(x)) \to x \).

**Proof.** Part (i). By the triangle inequality, we have that

\[
|f_n(g_n(x)) - f(g(x))| \leq |f_n(g_n(x)) - f(g_n(x))| + |f(g_n(x)) - f(g(x))| \\
\leq \sup_{x \in [a,b]} |f_n(y) - f(y)| + |f(g_n(x)) - f(g(x))|.
\]

The first term converges to zero since \( f_n \) converges to \( f \) uniformly in \([a,b]\). The argument of \( f \) in the second term, \( g_n(x) \), converges to \( g(x) \). Since \( f \) is continuous, the second term also converges to zero. Therefore, \( |f_n(g_n(x)) - f(g(x))| \to 0 \) as \( n \to \infty \).

Part (ii). By the triangle inequality, we have that

\[
\sup_{x \in [a,b]} \left| \frac{f_n(x)}{g_n(x)} - \frac{f(x)}{g(x)} \right| \leq \sup_{x \in [a,b]} \left| f_n(x) - f(x) \right| \sup_{x \in [a,b]} \left| \frac{g_n(x)}{g(x)} - \frac{1}{g(x)} \right| \\
+ \sup_{x \in [a,b]} \left| f(x) \right| \sup_{x \in [a,b]} \left| \frac{1}{g_n(x)} - \frac{1}{g(x)} \right| + \sup_{x \in [a,b]} \left| \frac{1}{g(x)} \right| \sup_{x \in [a,b]} \left| f_n(x) - f(x) \right|.
\]
By assumption, \( \sup_{x \in [a,b]} |f_n(x) - f(x)| \) converges to zero and \( \sup_{x \in [a,b]} \left| \frac{1}{g(x)} \right| = k^{-1} \) is finite. Further, \( \sup_{x \in [a,b]} |f(x)| \) if finite because \( f \) is continuous and \([a,b]\) is a compact set. Therefore, the left-hand side converges to zero as required if \( \sup_{x \in [a,b]} \left| \frac{1}{g_n(x)} - \frac{1}{g(x)} \right| \) converges to zero. To show this, observe that

\[
\sup_{x \in [a,b]} \left| \frac{1}{g_n(x)} - \frac{1}{g(x)} \right| \leq \sup_{x \in [a,b]} \left| \frac{1}{g_n(x)} \right| \sup_{x \in [a,b]} \left| \frac{1}{g(x)} \right| \sup_{x \in [a,b]} |g_n(x) - g(x)|
\]

converges to zero. Since \( \lim_{n \to \infty} \sup_{x \in [a,b]} |g_n(x) - g(x)| = 0 \) and \( \sup_{x \in [a,b]} \left| \frac{1}{g(x)} \right| = k^{-1} \) exists by assumption, it is sufficient to show that \( \sup_{x \in [a,b]} \left| \frac{1}{g_n(x)} \right| \) exists. Let \( N \) be such that for all \( n > N \), we have that \( \sup_{x \in [a,b]} |g_n(x) - g(x)| \leq \frac{k}{2} \). Such a value of \( N \) exists because \( g_n \) converges to \( g \) uniformly in \([a,b]\) and \( \inf_{x \in [a,b]} |g(x)| = k > 0 \). Hence, for all \( n > N \), \( \sup_{x \in [a,b]} \left| \frac{1}{g_n(x)} \right| < \frac{2}{k} \), which is finite.

Part (iii). Define \( f_n^{-1}(y) = \inf \{ x : f_n(x) > y \} \). Fix \( x \in (a,b) \). For any \( \varepsilon > 0 \), define \( \bar{\varepsilon} = \min \left\{ \frac{\varepsilon}{2}, x - a, b - x \right\} \) and \( \delta_\varepsilon = \min \{ f(x + \bar{\varepsilon}) - f(x), f(x) - f(x - \bar{\varepsilon}) \} \). Observe that \( \bar{\varepsilon} > 0 \) and \( \delta_\varepsilon > 0 \) because and \( f \) is strictly increasing. Pick \( N \) such that for all \( n > N \) \( \sup_{x' \in [a,b]} |f_n(x') - f(x')| < \delta_\varepsilon \). Such an \( N \) exists because \( f_n \) converges to \( f \) uniformly in \([a,b]\). To complete the proof, we will show that for all \( n > N \), \( f_n^{-1}(f(x)) > x - \varepsilon \) and \( f_n^{-1}(f(x)) < x + \varepsilon \).

Since \( f \) is strictly increasing, for all \( x' < x - \bar{\varepsilon}, f(x') + \delta_\varepsilon < f(x) \). Therefore, for all \( n > N \) and \( x' < x - \bar{\varepsilon}, f_n(x') < f(x) \). Hence, \( f_n^{-1}(f(x)) \geq x - \bar{\varepsilon} > x - \varepsilon \) for all \( n > N \).

Similarly, for all \( x' > x + \bar{\varepsilon}, f(x') - \delta_\varepsilon > f(x) \). Therefore, for all \( n > N \) and \( x' > x + \bar{\varepsilon}, f_n(x') > f(x) \). Hence, \( f_n^{-1}(f(x)) \leq x + \bar{\varepsilon} < x + \varepsilon \) for all \( n > N \). \(\square\)

**Lemma 4.** Let \( g \in L^2([0,1]) \) be continuous and \( s_n(g;x) \) be its Fourier-Legendre approximation of degree \( n \) evaluated at \( x \). For any \([a,b] \subset (0,1)\), the partial average \( S_n(g;x) = \frac{1}{n} \sum_{k=0}^{n-1} s_k(g;x) \) converges to \( g(x) \) uniformly in \([a,b]\).

**Proof.** The result is a corollary of Theorem IV.3.2 in Freud (1971). To apply this result, we will use the cumulative distribution function of the uniform distribution on \([0,1]\) as the function \( \alpha(x) \).

Let \( p_n(da;x) \) for \( n = 0,1,2... \) be the sequence of orthogonal polynomials defined in Theorem
I.1.2 of Freud (1971). It is straightforward to check that, for our chosen \( \alpha (x) \), \( p_n (d\alpha; x) = \sqrt{2m + 1} \Gamma_m (x) \), where \( \Gamma_m (x) \) be the \( m \)-th shifted Legendre Polynomial on \([0, 1]\),\(^{28}\) satisfied the conditions in Theorem I.1.2 because (i) each \( \Gamma_m (x) \) is a polynomial, (ii) the leading co-efficient of \( \Gamma_m (x) \) is positive and (iii) \( \int \Gamma_n (x) \Gamma_m (x)\, dx = \delta_{mn} \) where \( \delta_{mn} \) is the Kronecker delta. Moreover, \( p_n (d\alpha; x) \) is unique as noted in the remark below Theorem I.1.2 in Freud (1971).

Therefore, it remains to show that \( p_n (d\alpha; x) \) satisfies requirement (3.2) in Chapter IV of Freud (1971). As noted following this requirement, it is sufficient to show that for every pair \( x_2 \) and \( x_1 \) in a neighborhood of \( x_0 \in [a, b] \subset (0, 1) \), \( \frac{\alpha(x_2) - \alpha(x_1)}{x_2 - x_1} \geq m > 0 \), for some constant \( m \). This the case because for our chosen \( \alpha (x) \), because the left hand side is identically equal to 1 for every \( x_1, x_2 \in (0, 1) \).

Finally, \( s_k (g; x) \), as defined in equations IV(1.1) and IV(1.2) of Freud (1971) is the \( k \)-th order shifted Fourier-Legendre approximation of \( g \). Therefore, by Theorem IV.3.2 in Freud (1971), \( S_n (g; x) \) converges to \( g (x) \) uniformly in \([a, b] \subset (0, 1) \).

**Lemma 5.** Let \( v_n ' (\cdot; q_j, z) \) be the \( n \)-th order Fourier-Legendre approximation of \( v' (\cdot; q_j, z) \). If the hypotheses of Lemma 2 are satisfied, then \( v_n ' (\cdot; q_j, z) \) is identified for each \( z \in (0, 1) \) and \( q_j \).

**Proof.** We drop the parameters \( z, q_j \) for simplicity of notation as they are held fixed. As argued in the proof of Lemma 2, Assumptions 1 and 2 imply that the quantities \( a_k = \int_0^1 \varepsilon_D \, dv (\varepsilon_D; q_j, z) \) are identified for all \( k \leq n \). Let \( b_m \) be the (shifted) \( m \)-th Fourier-Legendre co-efficient of \( v' (\cdot) \) defined on \([0, 1]\), \( b_m = (2m + 1) \int_0^1 \Gamma_m (x) v' (x)\, dx \) where \( \Gamma_m (\cdot) \) is the \( m \)-th shifted Legendre polynomial on \([0, 1]\). Observe that each \( \Gamma_m (\cdot) \) is given by \( \Gamma_m (x) = \sum_{l=0}^m \gamma_{m,l} x^l \), with known co-efficients \( \gamma_{m,l} \). Therefore, the co-efficients \( b_m = (2m + 1) \sum_{l=0}^m \gamma_{m,l} \int_0^1 x^l v' (x)\, dx = (2m + 1) \sum_{l=0}^m \gamma_{m,l} a_l \), are identified. The second equality follows from the definition of \( a_l \).  

\(^{28}\)The shifted Legendre-Polynomials on \([0, 1]\) satisfy the orthogonality relationship \( \int_0^1 \Gamma_m (x) \Gamma_n (x)\, dx = \frac{1}{\Gamma_m} \delta_{m,n} \) where \( \delta_{m,n} \) is the Kronecker delta. The first few values are \( \Gamma_0 (x) = 1, \Gamma_1 (x) = 2x - 1, \Gamma_2 (x) = 6x^2 - 6x + 1 \).
C.4 Proof of Theorem 1

Identification of $E \left[ Y_{i,0} | \nu_{i,D} = \nu \right]$. Define $y_0 (\nu) = E \left[ Y_{i,0} | \nu_{i,D} = \nu \right] = \int g_0 (\tilde{v}) \, f_{\nu_0 | \nu_{D} = \nu} (\tilde{v}) \, d\tilde{v}$.

For a given $\nu$, fix $z$ such that there exists $\varepsilon_D \in (0, 1)$ with $v (\varepsilon_D ; q_j, z) = \nu$ and drop the conditioning on $z$ in what follows, for simplicity of notation.

Let $s$ and $\tilde{s}$ be a pair of models satisfying the hypotheses of Theorem 1, and let $\{ y_0 (\cdot) , v (\cdot) \}$ and $\{ \tilde{y}_0 (\cdot) , \tilde{v} (\cdot) \}$ be features that are associated with $s$ and $\tilde{s}$ respectively. We will show that if $\{ y_0 (\cdot) , v (\cdot) \} \neq \{ \tilde{y}_0 (\cdot) , \tilde{v} (\cdot) \}$, then there exists $n$, such that if $q^k_j$ is in the support of the distribution of offer types for all $k \leq n$, then the joint distribution of $Y_{i,0}, \{ T_{i,j}, \ldots, T_{i,j} \}$ conditional on $q^k_j$ differs for some $k \leq n$ under models $s$ and $\tilde{s}$.

Consider a value of $\tilde{v} \in (0, 1)$ such that $y_0 (\tilde{v}) \neq \tilde{y}_0 (\tilde{v})$ and $\tilde{v} = v (\tilde{x}; q_j)$ for some $\tilde{x} \in (0, 1)$.

Lemmas 2 and 5 imply that if either $v (\tilde{x}; q_j) \neq \tilde{v} (\tilde{x}; q_j)$ or $v' (\tilde{x}; q_j) \neq \tilde{v}' (\tilde{x}; q_j)$ for some $\tilde{x} \in (0, 1)$, then there exists $N$ such that for all $n > N$ the joint distribution of $\{ T_{i,j}, \ldots, T_{i,j(n)} \}$ conditional on $q^k_j$ for some $k \leq n$ differs for models $s$ and $\tilde{s}$. Therefore, it is sufficient to focus on the case when $v (\tilde{x}; q_j) = \tilde{v} (\tilde{x}; q_j)$ and $v' (\tilde{x}; q_j) = \tilde{v}' (\tilde{x}; q_j)$. Moreover, since $\tilde{x} \in (0, 1)$, we have that $v' (\tilde{x}; q_j) > 0$ (Assumption 4(i)) implying that it is sufficient to show that if $y_0 (v (\tilde{x}; q_j)) \neq \tilde{y}_0 (v (\tilde{x}; q_j))$, then the joint distribution of $Y_{i,0}, \{ T_{i,j}, \ldots, T_{i,j} \}$ conditional on $q^k_j$ differs for some $k \leq n$ under models $s$ and $\tilde{s}$.

We prove this by showing that if $y_0 (v (\tilde{x}; q_j)) \neq \tilde{y}_0 (v (\tilde{x}; q_j))$, then there exists $n$ such that if $q^k_j$ is in the support of the distribution of offer types for all $k \leq n$, then $Y_{i,0}, \{ T_{i,j}, \ldots, T_{i,j} \}$ conditional on $q^k_j$ differs for some $k \leq n$ under models $s$ and $\tilde{s}$.

To do this, we first show that the Fourier-Lebesgue approximation of the function $u(x) = y_0 (v (x; q_j)) v' (x; q_j)$ can be determined from observables. Assumptions 1 and 2 imply that for each $k \leq n$, we can re-write

$$E \left[ Y_{i,0} \times 1 \{ T_i = 0 \} | q^k_j \right] = \int_0^1 E \left[ Y_{i,0} | \nu_D = v (x; q_j) \right] x^k \, dv (x; q_j) = \int_0^1 x^k y_0 (v (x; q_j)) v' (x; q_j) \, dx.$$

The argument in the proof of Lemma 5 implies that the $n$-th order Fourier-Legendre approximation of $u(x; q_j) = y_0 (v (x; q_j)) v' (x; q_j)$, denoted as $u_n (x; q_j)$, is a function of the observables $\{ E \left[ Y_{i,0} \times 1 \{ T_i = 0 \} | q^k_j \right] \}_{k=1}^n$. Similarly, let $\tilde{u}_n (x; q_j)$ be the (shifted) Fourier-
Legendre series associated with model \( s \) with associated feature \( \{ \tilde{y}_0(\cdot), \tilde{v}(\cdot) \} \) such that \( \tilde{v} = v \).

Lemma 4 implies that for any subinterval \([a, b] \subset (0, 1)\), \( \frac{1}{n} \sum_{k=0}^{n-1} u_k(x; q_j) \) converges uniformly to \( u(x; q_j) \) if \( u(x; q_j) \) is square-integrable and continuous. Assumption 4(ii) implies that for any subinterval \([a, b] \subset (0, 1)\), \( \frac{1}{n} \sum_{k=0}^{n-1} u_k(x; q_j) \) converges uniformly to \( u(x; q_j) \) since the product of continuous functions is continuous. To show square-integrability of \( y_0(v(x; q_j))v'(x; q_j) \) observe that

\[
\int_0^1 y_0(v(x; q_j))^2 v'(x; q_j)^2 \, dx = \int_0^1 E[Y_{i,0}] v(x; q_j)^2 v'(x; q_j)^2 \, dx \\
= \int_0^1 E[Y_{i,0}] v^2 \left( v^{-1}(v(q_j); q_j) \right)^2 \, d\nu \leq \int_0^1 E[Y_{i,0}] \, d\nu \int v'(x; q_j)^2 \, dx.
\]

where the second line follows from changes of variables and Holder’s inequality. To show that the right hand side is bounded, observe that Assumption 4(i) implies that \( \int v'(x; q_j)^3 \, dx \) is finite, and that Jensen’s inequality implies

\[
\int_0^1 E[Y_{i,0}] \, d\nu \leq \int_0^1 E[Y_{i,0}^4] \, d\nu = E[Y_{i,0}^4],
\]

which is finite by Assumption 4(ii). Therefore, \( \tilde{u}_n(x; q_j) \) converges uniformly to \( u(x; q_j) \). An identical argument implies that \( \frac{1}{n} \sum_{k=0}^{n-1} \tilde{u}_n(x; q_j) \) converges uniformly to \( \tilde{u}(x; q_j) \) over \( x \in [a, b] \). Since \( \bar{x} \in (0, 1) \), we can pick \( [a, b] \) such that \( \bar{x} \in [a, b] \).

Now, let \( \delta = |y_0(v(\bar{x}; q_j))v'(\bar{x}; q_j) - \tilde{y}_0(v(\bar{x}; q_j))v'(\bar{x}; q_j)| > 0 \). Pick \( n \) such that

\[
\left| y_0(v(\bar{x}; q_j))v'(\bar{x}; q_j) - \frac{1}{n} \sum_{k=0}^{n-1} u_k(\bar{x}; q_j) \right| < \frac{\delta}{2} \quad \text{and} \quad \left| \tilde{y}_0(v(\bar{x}; q_j))v'(\bar{x}; q_j) - \frac{1}{n} \sum_{k=0}^{n-1} \tilde{u}_k(\bar{x}; q_j) \right| < \frac{\delta}{2}.
\]

Such an \( n \) exists because Lemma 4 implies that \( \frac{1}{n} \sum_{k=0}^{n-1} u_k(\bar{x}; q_j) \) and \( \frac{1}{n} \sum_{k=0}^{n-1} \tilde{u}_k(\bar{x}; q_j) \) converge to \( y_0(v(\bar{x}; q_j))v'(\bar{x}; q_j) \) and \( \tilde{y}_0(v(\bar{x}; q_j))v'(\bar{x}; q_j) \) respectively. Therefore, if \( q_j^k \) is in the support of the distribution of offer types for all \( k \leq n \), then

\[
\left| \frac{1}{n} \sum_{k=0}^{n-1} u_k(\bar{x}; q_j) - \frac{1}{n} \sum_{k=0}^{n-1} \tilde{u}_k(\bar{x}; q_j) \right| > 0.
\]

Because each \( u_n(x; q_j) \) and \( \tilde{u}_n(x; q_j) \) is determined by \( E[Y_{i,0} \times 1 \{ T_i = 0 \}] q_j^k \) for \( k \leq n \), we have shows that the joint distribution of \( Y_{i,0}, \{ T_{i,j}, \ldots, T_{i,j} \} \) conditional on \( q_j^k \) differs for some \( k \leq n \) under models \( s \) and \( \tilde{s} \).
Identification of $E[Y_{i,j}|\nu,\varepsilon_{i,j,D} = \varepsilon, q_j]$. Define

$$y_1(\nu, \varepsilon; q_j) = E[Y_{i,j}|\nu,\varepsilon_{i,j,D} = \varepsilon, \varepsilon_D, q_j]$$

$$= \int \int g_1(q_j, \nu, \varepsilon) f_{\varepsilon_D=\varepsilon}(\bar{\varepsilon}) f_{\nu=\nu}(\bar{\nu}) d\nu d\varepsilon.$$

Consider a pair of models $s$ and $\bar{s}$. As argued above, we can restrict to pairs such that $v(x; q_j, z) = \bar{v}(x; q_j, z)$ for all $x \in (0,1)$ and all $z$. For a given $\nu \in (0,1)$ and $\bar{x} \in (0,1)$, and let $\bar{z}$ be such that $v = v(\bar{x}; q_j,\bar{z})$. We will show that if $y_1(v(\bar{x}; q_j,\bar{z}), x; q_j) v'(\bar{x}; q_j,\bar{z}) \neq \bar{y}_1(v(\bar{x}; q_j,\bar{z}), x; q_j) v'(x; q_j,\bar{z})$, then there exists $n$ such that if $q_j^k$ is in the support of the distribution of offer types for all $k \leq n$, then the joint distribution of $Y_{i,j},\{T_{i,j},\ldots,T_{i,j}\}$ conditional on $q_j^k$ and $\bar{z}$ differs for some $k \leq n$ under models $s$ and $\bar{s}$.

Assumptions 1 and 2 imply that for each $k \leq n$, we can re-write the observed quantity

$$E\left[Y_{i,j} \times 1\{T_{i,j} = 1\} | q_j^k, \bar{z}\right]$$

$$= \int_0^1 E\left[Y_{i,j}|\nu_{D} = v(x; q_j, \bar{z}), \varepsilon_{i,j,D} = \varepsilon, q_j\right] x^{k-1} (1-x) dv(x; q_j, \bar{z})$$

$$= \int_0^1 x^{k-1} (1-x) y_1 (v(x; q_j, \bar{z}), x; q_j) v'(x; q_j, \bar{z}) dx.$$  

Arguments similar to those above imply that for any $[a, b] \subset (0,1)$, we can uniformly approximate the function

$$u(x; q_j, \bar{z}) = (1-x) y_1 (v(x; q_j, \bar{z}), x; q_j) v'(v(x; q_j, \bar{z}); q_j, \bar{z})$$

over $x \in [a, b] \subset (0,1)$ with $\frac{1}{n} \sum_{k=0}^{n-1} u_n(x; q_j, \bar{z})$, where $u_n(x; q_j, \bar{z})$ is determined as a function of observed conditional distributions given $\bar{z}$ and $q_j^k$ for $k \leq n$. This claim required continuity and square-integrability of $u(v(x; q_j, \bar{z}); q_j, \bar{z})$ in $x$. Continuity follows because $y_1(\nu, x; q_j)$, $v(x; q_j, \bar{z})$ and $v'(x; q_j, \bar{z})$ are assumed to be continuous (Assumption 4) and the composition and product of continuous functions is continuous. Square integrability follows similarly to
Our main result requires an additional mild restriction on the conditional distribution of mechanism allows for selection in transplanted survival outcomes. Assumption 5.

The results in this subsection explicitly assume that agent $\{\text{conditional on } u\}$ then, as argued above, Lemma 4 implies that there exists $\nu, \varepsilon, D$ such that $\text{finite. Therefore, if}$

$$
\delta = |(1 - \bar{x}) y_1 (v (\bar{x}; q_j, \bar{z}), x; q_j) v' (\bar{x}; q_j, \bar{z}) - (1 - \bar{x}) \bar{y}_1 (v (\bar{x}; q_j, \bar{z}), \bar{x}; q_j) v' (\bar{x}; q_j, \bar{z})|,
$$

then, as argued above, Lemma 4 implies that there exists $n$ such that

$$
\left| \frac{1}{n} \sum_{k=0}^{n-1} u_n (\bar{x}; q_j, \bar{z}) - \frac{1}{n} \sum_{k=0}^{n-1} \bar{u}_n (\bar{x}; q_j, \bar{z}) \right| > 0.
$$

Because each $u_n (\bar{x}; q_j, \bar{z})$ and $\bar{u}_n (\bar{x}; q_j, \bar{z})$ is determined by the conditional expectations $\{E [Y_{i,j} \times 1 \{T_{i,j} = 1\} | q^k_j, \bar{z}]\}_{k=1}^{n}$, we have shown that the joint distribution of $Y_{i,j}, \{T_{i,j}, \ldots, T_{i,j}\}$ conditional on $q^k_j$ and $\bar{z}$ differs for some $k \leq n$ under models $s$ and $\bar{s}$.

**C.5 Dynamic Selection**

The results in this subsection explicitly assume that $Y_{i0}$ denotes survival. Therefore, we will assume that agent $i$ may be assigned object $j$ only if $Y_{i0} > t_{i,j}$. Using waiting time in the mechanism allows for selection in transplanted survival outcomes.

Our main result requires an additional mild restriction on the conditional distribution of $Y_{i0}$:

**Assumption 5.** For any interval $I \subset \mathbb{R}_+$, $P (Y_{i0} \in I | \nu_D)$ is a continuous function of $\nu_D$. 

With this assumption, we show the identification in the presence of dynamic selection:

**Theorem 2.** Suppose that Assumption 5 and the hypothesis of Theorem 1 hold, allowing for \( t_{ij} > 0 \). Then, the probability \( P(D_{ij} = 1|\nu_{i,D} = \nu_D, Y_{i0} \geq t_{ij}) \) and the expectation \( E[\psi(Y_{ij})|\nu_{i,D} = \nu_D, \varepsilon_{ij,D} \geq \varepsilon_D, Y_{i0} \geq t_{ij}] \) are identified for any bounded function \( \psi(\cdot) \), and all \( \varepsilon_D \in (0,1) \) and \( \nu_D \in (0,1) \) such that there exist \( z \) in the support of its distribution with \( \nu_D = v(\varepsilon_D; q_j, z) \) and \( P(Y_{i0} \geq t_{ij}|\nu_D) > 0 \).

The argument is developed in two steps. In the first step, we identify the conditional distribution of \( \nu_D \) for agents that survive until time \( t \) (Lemma 6). The second step takes this conditional distribution and combines it with the arguments that parallel those in Lemma 2 and Theorem 1.

Let \( h_t(v) \) be the cdf of \( \nu_D \) conditional on surviving until \( t \). It is given by \( h_t(v) = \int_v^\infty \frac{P(Y_{i0} \geq t|\nu_D)}{P(Y_{i0} \geq t)} d\nu_D \).

**Lemma 6.** Suppose that the hypothesis of Theorem 1 hold. The function \( h_t(v) \) is identified for every \( t \geq 0 \).

**Proof.** Let \( q_j \) be a donor-type that arrives at the same time as patient \( i \). Because the image of \( v(\cdot, q_j, z) \) is the unit interval (Assumption 3), for any \( \nu_D \in (0,1) \) and \( z \), there exists \( \varepsilon_D \in (0,1) \) such that \( \nu_D = v(\varepsilon_D; q_j, z) \). Theorem 1 implies that for every \( t \geq 0 \), \( P(Y_{i0} \geq t|\nu_D) = E[1 \{Y_{i0} \geq t\}|\nu_D] \) is identified. Thus, \( P(Y_{i0} \geq t) \) and the function \( h_t(v) \) is identified. \( \square \)

Define the cdf of the probability that a patient which survives until \( t_{ij} \) rejects a kidney of type \( q_j \) as \( v_j(\varepsilon_D; q_j, z_i) = h_{t_{ij}} \circ v(\varepsilon_D; q_j, z_i) \).

**Lemma 7.** Suppose that Assumption 5 and the hypothesis of Theorem 1 hold. Then, \( v_j(\varepsilon_D; q_j, z_i) \) and \( v(\varepsilon_D; q_j, z_i) \) are identified for every \( \varepsilon_D \) such that

\[
P(Y_{i0} \geq t_{ij}|\nu_D = v(\varepsilon_D; q_j, z_i)) > 0.
\]

**Proof.** Fix \( \varepsilon_D \) is such that \( P(Y_{i0} \geq t_{ij}|\nu_D = v(\varepsilon_D; q_j, z_i)) > 0 \). Note that \( h_{t_{ij}}(\cdot) \) is differentiable because \( P(Y_{i0} \geq t_{ij}|\nu_D) > 0 \) and Assumption 5 is satisfied. Moreover, it is increasing
in $v$ and has image equal to the unit interval. Therefore, we have that $v_j (\varepsilon_D; q_j, z_i)$ satisfies Assumption 3. By arguments identical to those in Lemma 2, $v_j (\varepsilon_D; q_j, z_i)$ is identified. Assumption 5 implies that $P (Y_{i0} \geq t_{i,j} | \nu_D)$ is also positive in a neighborhood around $\nu_D$ and that $h_{t_{i,j}}$ is strictly increasing at that point. Thus, $v (\varepsilon_D; q_j, z_i) = h_{t_{i,j}}^{-1} \circ v_j (\varepsilon_D; q_j, z_i)$ is identified because the terms on the right hand side are identified.

**Proof of Theorem 2:**

**Proof.** Take any $\varepsilon_D \in (0,1)$ and $\nu_D \in (0,1)$ satisfying the stated hypotheses. As argued in the proof of Lemma 7, $v_j (\cdot; q_j, z_i)$ satisfies Assumption 3 and is identified. Since $v_j (\cdot; q_j, z_i)$ is increasing in its argument, $P (D_{ij} = 1 | \nu_{i,D} = v (\varepsilon_D; q_j, z_i), Y_{i0} \geq t_{i,j})$ is identified.

By the chain rule $\frac{\partial}{\partial \varepsilon} v_j (\varepsilon_D; q_j, z_i) = \frac{\partial}{\partial \nu_j} h_{t_{i,j}} (\nu_D) \frac{\partial}{\partial \varepsilon} v (\varepsilon_D; q_j, z_i)$. Note that $\frac{\partial}{\partial \varepsilon} v (\varepsilon_D; q_j, z_i)$ is continuous, bounded and strictly positive. Also, $\frac{\partial}{\partial \nu_j} h_{t_{i,j}} (\nu_D) = \frac{P (Y_{i0} \geq t_{i,j} \mid \nu_D)}{P (Y_{i0} \geq t_{i,j})}$ is continuous, bounded and strictly positive because the denominator is strictly positive by the assumption that there exists $\nu_D$ with $P (Y_{i0} \geq t_{i,j} \mid \nu_D, x_i) > 0$ and Assumption 5. Therefore, $\frac{\partial}{\partial \varepsilon} v_j (\varepsilon_D; q_j, z_i)$ is continuous and bounded and strictly positive. Therefore, the function $\frac{\partial}{\partial \varepsilon} v_j (\cdot; q_j, z_i)$ it is strictly positive in a neighborhood of $\varepsilon_D$. Arguments identical to those used for proving Theorem 1 imply that $E \left[ Y_{ij} | h_{t_{i,j}} (\nu_{i,D}) = h_{t_{i,j}} (\nu_D), \varepsilon_{ij,D} \geq \varepsilon_D, Y_{i0} \geq t_{i,j} \right]$ is identified. Because $P (Y_{i0} \geq t_{i,j} \mid \nu_D, x_i) > 0$, we have that $h_{t_{i,j}} (\nu_D)$ is strictly increasing at $\nu_D$, the event $h_{t_{i,j}} (\nu_{i,D}) = h_{t_{i,j}} (\nu_D)$ is equivalent to $\nu_{i,D} = \nu_D$. \qed
## Additional Figures and Tables

### Table D.5: Top 10 offers: Balance

<table>
<thead>
<tr>
<th></th>
<th>Age (1)</th>
<th>Diabetes (2)</th>
<th>Female (3)</th>
<th>Weight (4)</th>
<th>Height (5)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>log(1 + # Top 10 Offers in 2 Years)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>KDPI &lt;= 50%</td>
<td>-0.0479</td>
<td>0.00134</td>
<td>-0.00158</td>
<td>-0.269*</td>
<td>0.0253</td>
</tr>
<tr>
<td></td>
<td>(0.0772)</td>
<td>(0.00302)</td>
<td>(0.00277)</td>
<td>(0.108)</td>
<td>(0.0732)</td>
</tr>
<tr>
<td>KDPI &gt; 50% or Missing</td>
<td>-0.0233</td>
<td>-0.00427</td>
<td>0.000269</td>
<td>0.104</td>
<td>0.0137</td>
</tr>
<tr>
<td></td>
<td>(0.0683)</td>
<td>(0.00294)</td>
<td>(0.00276)</td>
<td>(0.101)</td>
<td>(0.0819)</td>
</tr>
<tr>
<td><strong>DSA FE, Year FE, and Blood Type FE</strong></td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
</tr>
<tr>
<td><strong>Control for Pediatric at Listing</strong></td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
</tr>
<tr>
<td><strong>CPRA Category Controls</strong></td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
</tr>
<tr>
<td><strong>F-test p-Value</strong></td>
<td>0.499</td>
<td>0.267</td>
<td>0.787</td>
<td>0.037</td>
<td>0.828</td>
</tr>
<tr>
<td><strong>Number of Observations</strong></td>
<td>128949</td>
<td>127414</td>
<td>128949</td>
<td>127363</td>
<td>126619</td>
</tr>
<tr>
<td><strong>R-Squared</strong></td>
<td>0.026</td>
<td>0.022</td>
<td>0.074</td>
<td>0.038</td>
<td>0.034</td>
</tr>
<tr>
<td><strong>Distribution of # Top 10 Offers in 2 Years</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean</td>
<td>16.92</td>
<td>16.97</td>
<td>16.92</td>
<td>16.91</td>
<td>16.88</td>
</tr>
<tr>
<td>Std. Dev.</td>
<td>22.86</td>
<td>22.92</td>
<td>22.86</td>
<td>22.82</td>
<td>22.79</td>
</tr>
</tbody>
</table>

Notes: * p<0.05, ** p<0.01, *** p<0.001

The sample for all regressions is patients who registered between 2000 and 2008. Dependent variables are as indicated in the column headers. All regressions control for DSA fixed effect, registration year fixed effect, blood type fixed effect, an indicator for pediatric at registration, and indicators for CPRA = 0, 20 <= CPRA < 80, CPRA >= 80, and CPRA missing at registration. Standard errors, clustered by DSA, registration year, and blood type, are in parentheses. F-test tests against the null hypothesis that the coefficients on the instruments are zero.
Table D.6: Scarcity Instruments: Balance

<table>
<thead>
<tr>
<th>Age</th>
<th>Diabetes</th>
<th>Female</th>
<th>Weight</th>
<th>Height</th>
</tr>
</thead>
<tbody>
<tr>
<td>(1)</td>
<td>(2)</td>
<td>(3)</td>
<td>(4)</td>
<td>(5)</td>
</tr>
</tbody>
</table>

Log(1 + No. Donors)

| Patients Waited 0-1 years | -0.319 | 0.00271 | -0.00105 | 0.151 | -0.254 |
|                          | (0.331) | (0.0125) | (0.0115) | (0.516) | (0.328) |
| Patients Waited 1-2 years | 0.135 | -0.0129 | 0.00164 | 0.330 | 0.0594 |
|                          | (0.299) | (0.0117) | (0.0109) | (0.457) | (0.307) |
| Patients Waited 2-3 years | -0.256 | 0.000252 | 0.0130 | -0.290 | -0.0133 |
|                          | (0.272) | (0.0104) | (0.00902) | (0.397) | (0.269) |
| Patients Waited 3-4 years | 0.286 | 0.0160 | -0.0272*** | 0.114 | 0.109 |
|                          | (0.223) | (0.00910) | (0.00800) | (0.348) | (0.225) |
| Patients Waited 4-5 years | -0.0248 | -0.0117 | 0.0120* | -0.393 | -0.212 |
|                          | (0.153) | (0.00603) | (0.00533) | (0.220) | (0.152) |

Log(1 + No. Offers)

| Patients Waited 0-1 years | 0.395* | 0.0165* | -0.00352 | 0.301 | 0.350 |
|                          | (0.195) | (0.00817) | (0.00765) | (0.323) | (0.218) |
| Patients Waited 1-2 years | -0.0375 | 0.0000856 | -0.00111 | -0.228 | -0.174 |
|                          | (0.215) | (0.00847) | (0.00764) | (0.328) | (0.228) |
| Patients Waited 2-3 years | 0.0897 | 0.000332 | -0.00488 | 0.300 | 0.0110 |
|                          | (0.213) | (0.00817) | (0.00698) | (0.315) | (0.223) |
| Patients Waited 3-4 years | -0.123 | -0.0124 | 0.0189** | -0.100 | -0.0956 |
|                          | (0.196) | (0.00766) | (0.00666) | (0.299) | (0.196) |
| Patients Waited 4-5 years | 0.0748 | 0.0125* | -0.0130** | 0.234 | 0.114 |
|                          | (0.133) | (0.00527) | (0.00475) | (0.197) | (0.132) |

Year FE, DSA FE, and blood type FE

| x | x | x | x | x |

Control for Pediatric at Listing

| x | x | x | x | x |

CPRA Category Controls

| x | x | x | x | x |

F-test p-Value

| 0.319 | 0.166 | 0.201 | 0.555 | 0.692 |

Number of Observations

| 87205 | 87200 | 87205 | 86078 | 85500 |

R-Squared

| 0.025 | 0.021 | 0.076 | 0.036 | 0.038 |

Notes: * p<0.05, ** p<0.01, *** p<0.001
The sample for all regressions is adult patients who registered on the waitlist between 1999Q4 and 2005Q4. Each regression is on patient level, where the dependant variable is the patient characteristics in the column header at registration. Each regression has five regressors indexed by \( k = 0, 1, 2, 3, 4 \), where the kth regressor for patient \( i \) is computed as the number of unique donors (offers) such that: the offer is made to patients who are in the same DSA as \( i \), have the same blood type as \( i \), and have waited the same number of years as \( i \); the offer is made between \( 4k+1 \) and \( 4k+4 \) quarters, inclusive, from the quarter when \( i \) registers (e.g. if \( i \) registers in 2002Q1, then the offer must be made between 2003Q2 and 2004Q1 for \( k = 1 \). All regressions control for DSA fixed effect, registration year fixed effect, blood type fixed effect, an indicator for pediatric at registration, and indicators for CPRA = 0, 20 <= CPRA < 80, CPRA >= 80, and CPRA missing at registration. Robust standard errors, clustered by DSA, registration year, and blood type, are in parentheses. F-test tests against the null hypothesis that the coefficients on the five regressors are zero.
Table D.7: Robustness

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<tr>
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<th>(1)</th>
<th>(2)</th>
<th>(3)</th>
<th>(4)</th>
<th>(5)</th>
<th>(6)</th>
<th>(7)</th>
<th>(8)</th>
<th>(9)</th>
<th>(10)</th>
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<tbody>
<tr>
<td>Realized Assignment</td>
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<td></td>
<td></td>
<td>8.13</td>
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<tr>
<td>Random Assignment among</td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>All Patients</td>
<td>7.27</td>
<td>7.87</td>
<td>7.71</td>
<td>7.56</td>
<td>7.91</td>
<td>8.92</td>
<td>7.74</td>
<td>7.16</td>
<td>7.81</td>
<td>7.65</td>
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<tr>
<td>Transplanted Patients</td>
<td>7.60</td>
<td>8.23</td>
<td>8.09</td>
<td>8.12</td>
<td>8.31</td>
<td>9.38</td>
<td>8.09</td>
<td>7.65</td>
<td>8.43</td>
<td>8.19</td>
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<tr>
<td>No Choice</td>
<td>7.99</td>
<td>8.01</td>
<td>7.90</td>
<td>7.83</td>
<td>8.00</td>
<td>8.85</td>
<td>7.86</td>
<td>7.05</td>
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<tr>
<td>Box-Cox $\rho$</td>
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</tr>
<tr>
<td>Survival without Transplant</td>
<td>0.5</td>
<td>0.5</td>
<td>0.5</td>
<td>0.5</td>
<td>0.5</td>
<td>0.4</td>
<td>0.5</td>
<td>0.6</td>
<td>0.6</td>
<td>0.6</td>
</tr>
<tr>
<td>Survival with Transplant</td>
<td>0.6</td>
<td>0.6</td>
<td>0.6</td>
<td>0.6</td>
<td>0.6</td>
<td>0.5</td>
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<td>0.7</td>
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<tr>
<td># Past Donors</td>
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<tr>
<td># Past Offers</td>
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<tr>
<td># Future Donors</td>
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<td>Donor Unobservables</td>
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<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
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<tr>
<td>Other Unobservables</td>
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<td>x</td>
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<td>Adding Cold Ischemic Time</td>
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</tbody>
</table>

Notes: Robustness of the results presented in Figure 4. The baseline specification is presented in column (2). The remaining specifications vary the instruments, the presence of $\eta_j$, or the Box-Cox shape parameters as indicated in the table.