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 aggregate LYFT can be increased to 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: agarwaln@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 without using monetary transfers. Examples include public schools, public housing, and organ allocation. An influential theoretical and a growing empirical literature study the design of these mechanisms. In this literature, notions of efficiency derived from choices are central to evaluating a design. This desideratum often differs from objectives emphasized by policymakers. As examples, school districts emphasize student achievement and organ transplant systems emphasize patient survival.

Because canonical choice-based mechanisms are not designed with these outcomes in mind, it is unclear whether they perform well on this dimension. Choices made by agents who may not be well-informed about the benefits of various options and co-ordination failures may undercut this objective.[1] If so, a planner who can dictate assignments based on benefits estimated using extensive administrative data on outcomes may be able to do better. On the other hand, agents may also have private information about the likely outcomes and using a choice-based mechanism may serve policymakers' objectives.

This paper evaluates the assignment mechanism used to allocate kidneys from deceased donors on the basis of survival outcomes. We compare the performance and distributional consequences of the mechanism to two benchmarks: the survival maximizing assignment and the random assignment. 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 methodological and empirical contributions. First, we build on the literature on Roy selection to analyze a joint model of choices and outcomes in an assignment mechanism. We show how to identify and estimate the effects of counterfactual assignments by using variation generated by the mechanism and instruments that only affect choices but are excluded from outcomes. Second, we estimate the Life-Years from Transplantation (LYFT), defined as the difference between median survival

[1] Moreoever, in the kidney allocation context that we study below, surgeons who advise patients may suffer from agency problems that can misalign decisions relative to maximizing survival outcomes.

with and without a transplant, as a function of patient/donor-specific observed and unobserved characteristics. Third, using these estimates we compare the mechanism used in practice to alternative assignments to assess its performance and identify the scope for further improvements.

Organs from deceased donors are a scarce and valuable resource that need to be allocated efficiently. Approximately 100,000 patients suffering from kidney failure are currently waiting for a life-saving transplant. Only one-sixth receive a transplant in a typical year, and thousands die while waiting.

When a kidney becomes available, patients on the waitlist are offered the organ in a priority order. Patients may choose to reject an offer in order to wait for a more preferable one. This decision may therefore depend on the perceived benefits of a transplant from the offered organ. We consider the decision to accept or refuse an offer along with the potential survival outcomes and then incorporate the potential for selection.

Our model has three components. The first component models the choices patients make as a function of the patients' and organs' attributes as well as time to treatment; the second models post-transplant survival with the offered organ as a function of these variables; and the third governs patient survival without a transplant as a function of patient attributes. This model builds on the approach by Heckman and Navarro (2007) for analyzing sequential choices and dynamic treatment effects, extending it to allow for many treatment-types. Given our focus on evaluating alternative assignments, we allow patient-level unobservables that govern choices to be correlated both with outcomes with and without a transplant, and for patient-donor specific unobservables influencing choices to be correlated with post-transplant outcomes.

The model can generate selection into transplantation along three important margins from the perspective of evaluating assignments. 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 for two reasons. First, patient mortality and waiting times built into the mechanism induce

selection. For example, the mechanism prioritizes patients who have waited longer, thereby selecting patients with high untransplanted survival into transplantation. Second, patient choice may induce selection. Survival without a transplant may be related to choice and organs that are particularly well-suited to a patientmay be more likely to be accepted.

These sources of selection create an identification challenge because they may be driven by unobservables. We prove that our model is non-parametrically identified if two sources of variation are available. The first source of variation is randomness in the offers made to a given patient, conditional on the patient's priority-type in the mechanism. This source of variation allows us to compare the outcomes of patients whose final assignments differed due to variation in which organs were offered to them. Therefore, it identifies a treatment effect – the difference between the survival outcomes for the select group of patients whose assignment is affected by an offer.

An important limitation of using only this first source of variation is that it does not readily allow us to predict survival from counterfactual assignments. Doing so is necessary in order to 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 show that an instrument that shifts choices while holding the (distribution of) outcomes fixed can be used to identify the effects of alternative assignments. A related approach has been used in other settings by Geweke et al. (2003); Heckman and Navarro (2007); Walters (2018); Hull (2018); van Dijk (2019) to correct for selection and to estimate marginal treatment effects (Heckman and Vytlacil, 2005). For our application, we use variation in scarcity across geography and time after showing that our measures are balanced on patient-specific observables. 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. These patterns are also reflected in the final transplants: transplanted patients have a

higher LYFT from the average organ as compared to untransplanted patients. Taken together, these results suggest that prior approaches that do not account for selection on unobservable factors (e.g. Wolfe et al., 2008, 2009) yield biased estimates.

Next, we benchmark this assignment from the perspective of a planner interested in maximizing survival effects as measured by LYFT. We focus on survival as an objective because it is a focal outcome in our empirical context.[2] We compare the observed assignment to alternatives ranging from a random assignment to one that maximizes LYFT by reallocating patients and donors. The latter represents the maximum LYFT achievable by assigning patients to organs. Because distributional constraints may limit the ability to select which patients get a transplant, we also consider alternatives that assigns organs to different patients 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 on observed patient and donor characteristics.

Our results suggest that the mechanism does better than random allocation, but that there is significant room for improvement. A random assignment yields an average LYFT of 7.87, much lower than an average of 8.78 in the mechanism used during our sample period. Compared to a random assignment, the equilibrium assignment selects patients who benefit more from the transplant and matches these patients to donors who are more suitable for them.

Most of this gain in LYFT 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. However, LYFT could be increased to 13.84 by changing the assignment. A significant portion of these gains can be achieved if a planner can dictate assignments using observables in our dataset. The drop from the optimal assignment suggests that choice may not be dispensable if the unobserved types are private information.

These improvements in LYFT have important distributional consequences that may present real-world challenges. Specifically, we find that realizing higher LYFT requires

[2]Observe that there is no natural numeraire good that is transferable in the organ allocation context. This fact poses challenges to utilitarian objectives that are often justified based on a Kaldor-Hicks criteria.

transplanting patients who are relatively healthy and will live longer without a transplant. Such re-distribution is necessary because we find that benefits from transplant and survival without a transplant are strongly correlated, and most of the heterogeneity in LYFT is across patients. Therefore, the planner faces a dilemma between maximizing survival benefits and transplanting urgently sick patients.

Related Literature

This paper contributes to several literatures. We provide an alternative perspective for evaluating assignments to the literature studying assignment mechanisms (Abdulkadiroglu and Sönmez, 2003; Pathak, 2017). This literature typically uses student preferences as the welfare-relevant object. For example, the empirical literature, which has focused on school choice problems, uses a willingness to travel measure for welfare comparisons (see Agarwal and Somaini, 2020, for a survey).

A large theoretical literature has studied the design of living donor kidney exchanges (e.g. Roth et al., 2004, 2007). Despite growth in this market, kidney exchanges account for less than ten percent of all kidney transplants (see Agarwal et al., 2019). The most closely related paper on deceased donor organ assignment is Agarwal et al. (2021), which uses a decision-theoretic notion of welfare by comparing a change in the mechanism to an equivalent increase in donor supply.

Our work contributes an instrumental variables approach to a medical literature that constructs LYFT measures (Wolfe et al., 2008), which are commonly used to guide organ policy design[3] and to calculate cost savings from transplantation. The current state of the art is based on observational studies in part because conducting randomized control trials is both challenging and creates ethical issues. These methods compare survival curves for patients with similar characteristics, ignoring issues related to selection. Few papers within economics study survival outcomes, focusing instead on the total number of transplants (see Teltser, 2019; Dickert-Conlin et al., 2019, for exceptions).

[3]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).

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. 2011; Abdulkadiroglu et al., 2017). The focus of this literature has been to estimate a local average treatment effect. In our context, this estimand would preclude analyzing outcomes from alternative assignments because the set of compliers would change. Our model explicitly incorporates choices in the mechanism and uses a choice shifter in order to estimate the distribution of effects.

The techniques we use build on a large literature studying selection models (Roy, 1951; Heckman and Honore, 1990). Our methods are most closely related to papers that combine outcomes with choice models to correct for selection when estimating treatment effects (e.g. Geweke et al., 2003; Heckman and Navarro, 2007; Walters, 2018; Hull, 2018; van Dijk, 2019). In particular, Geweke et al. (2003) use a Gibbs' sampling in a model that allows for selection on gains. Our use of a Bayesian approach is, as far as we know, new in the literature on estimating causal survival models (e.g. Abbring and Van den Berg, 2003).

Our model is most closely related to Heckman and Navarro's (2007) approach used to study the effects of dropping out of school in any particular grade. An important challenge for our study is that the number of potential donors for each patient vastly exceeds the fixed number of grades for any student. Thus, evaluating counterfactual assignments requires handling match-specific heterogeneity for a large number of potential treatments. We address this issue by using observed characteristics to delineate donor types while allowing for match-specific unobservables to capture heterogeneous gains, which are potentially correlated with choices.

The main difference relative to the aforementioned papers is that we estimate the distribution of treatment effects that include match-specific heterogeneity within observationally identical treatments in order to evaluate alternative assignments.[4] This

[4]Evaluating counterfactual treatment assignment is related to recent work by Kitagawa and Tetenov (2018), who study the statistical properties of assignments that maximize treatment outcomes conditional on observable covariates in a setting without selection on unobservables. A difference in our setting is that treatments within groups are also differentiated.

requires us to simultaneously estimate selection on three margins: baseline outcomes, average outcomes under an assignment, and match-specific effects.

Overview

Section 2 describes the institutions, introduces the data, and presents descriptive evidence. Section 3 explains the model. Section 4 outlines the instruments. Section 5 demonstrates our identification results and specifies the empirical model that we take to the data. Section 6 explores our estimates. Section 7 presents our results on LYFT generated by the mechanism, and section 8 compares it to alernatives. Section 9 concludes.

2 Background, Data, and Descriptive Evidence

This section begins with the basics of kidney transplation before describing the allocation system. We then detail our data and present key descriptive facts to motivate our study.

2.1 Institutional Features

2.1.1 Basics of Kidney Transplantation

Approximately 750,000 patients are afflicted with End-Stage Renal Disease (ESRD) in the United States (USRDS, 2018). Medicare provides these patients with near universal coverage for costs related to ESRD, irrespective of age. This program cost the federal government $35.4 billion in 2016, accounting for 7.2 percent of overall Medicare paid claims (USRDS, 2018) or approximately 1 percent of the federal budget.

Transplantation is considered the best treatment for ESRD and is estimated to extend an average patient's life by approximately seven years for a patient who is more than 50 years old and eleven years for a young adult (Wolfe et al., 2008). In addition, a transplant also saves on expensive dialysis treatment. Current estimates suggest that

each transplant is expected to save \$195,000 – \$400,000 over the life of a transplanted patient, depending on insurance status (Irwin et al., 2012; Held et al., 2016; USRDS, 2018). These estimates are based on survival models that control for patient and donor characteristics and a comparison of healthcare costs for patients with and without a transplant. Our methods improve the estimates of the former set of components by relying on quasi-experimental variation.

There is significant potential for heterogeneity in survival effects along several important dimensions, even amongst compatible patient-donor pairs.[5] First, survival both with and without a transplant can differ based on the patient's health conditions. Some patients may tolerate dialysis better than others. Similarly, the underlying cause of kidney disease and other co-morbidities can affect a patient's post-transplant survival prospects. Second, donor quality can significantly influence transplants. For example, the circumstances of the donor's death, kidney function, and the donor's health prior to death are considered important determinants of organ quality. Finally, there are match-specific factors that affect post-transplant survival. Examples include size and weight match as well as tissue-protein similarity between patient and donor. Our methods will estimate the effects of these factors on life-year benefits.

2.1.2 The Allocation of Deceased Donor Kidneys

The allocation of organs from deceased donors is organized using a prioritized waiting list through which patients receive offers when an organ becomes available and may choose to accept or reject it. This allocation system is co-ordinated using a system called UNet. It collects detailed information about the donor's medical history and organ characteristics and transmits it to biologically compatible patients who are being offered the kidney. Each donor's kidneys are allocated to the highest-priority patients

[5]To receive a kidney transplant, the patient must be considered biologically compatible with the donor. Compatibility requires that a patient does not have a pre-existing immune response to the donated organ's cells. After transplantation, medications can limit new immune responses. We hold medical practices related to determining compatibility and post-transplant management as constant when we measure survival benefits. Danovitch (2009) provides further details about kidney biology and medical practices.

on the waitlist who are willing to accept the organs. Patients whose kidney function is below 20% are eligible to register for the waitlist. During our sample period, waiting time accrued from the registration date. Therefore, patients have incentives to register as soon as they become eligible.

Prior to 2014, patient priority in the kidney assignment system was based primarily on waiting time and tissue-type similarity between the patient and donor. Specifically, each kidney is 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.[6] Within each priority group, the points system is based on tissue type similarity, whether or not the patient is pediatric, patient sensitization, and waiting time (see OPTN, 2014, for details). This allocation system evolved over time with incremental changes aimed at increasing the efficiency of the system (Smith et al., 2012).

A revision to the system aimed at improving survival benefits was implemented on December 4, 2014. The most important change gives greater priority to the patients in the top quintile of expected post-transplant survival for the top quintile predicted organ quality because these patients are believed to have the largest survival benefit from these organs. In addition, the system also increases priority for extremely hard to match patients and reduces emphasis on wait time. We refer the reader to OPTN (2017) for a detailed description of the priorities and points used.

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), 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, kidney waitlist systems have been designed using heuristics aided by simulations and compromises in consideration

[6]The local regions are defined along state boundaries in most cases with exceptions to make sure that a metropolitan area is not split into two regions. This was done, in part, because some regions did not want to disadvantage their patients if others had a high demand for organs. Local allocation also helps reduce the amount of time an organ needs to be preserved using specialized equipment while outside the donor's body.

of distributional effects rather than using a formal optimal design approach (see Stegall et al., 2017, for a historical perspective on the 2014 reforms).

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, survival, and graft failure 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 in UNet and forms submitted by transplant centers from patient follow-ups after a transplant is performed.

We restrict attention to patients who first joined the kidney waiting list between January 1st, 2000 and December 31st, 2010.[7] From this set, we exclude patients who needed multiple organ transplants and those that received a living donor kidney. Correspondingly, we only use data on donor offers and acceptance decisions for these patients.

The data allow us to measure survival outcomes using information on patient death merged from social security records and transplant center reports. These records are consistently populated until December 31st, 2015.[8] 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.

[7]For patients with multiple listings, we keep the earliest registration if the patients never received a transplant; otherwise, we keep the earliest registration with transplant record.

[8]Our data use agreement allows for periodic updates, which we plan to include in future iterations of the paper.

2.2.2 Descriptive Analysis

Patients and Donors

Table 1: Patient Characteristics

	All Patients		Received Deceased Donor Transplant	
	Mean	S.D.	Mean	S.D.
New Patients per Year	15956		8393	
Panel A: Outcomes				
Died by Year Five (%)	27.4	44.6	9.3	29.1
Survived Five Years (%)	64.2	47.9	86.2	34.4
Censored by Year Five (%)	8.4	27.7	4.4	20.6
Transplanted by Year Five (%)	47.2	49.9	89.7	30.4
Panel B: Characteristics				
Age at Registration	51.4	14.2	48.9	15.2
On Dialysis at Registration (%)	77.3	41.9	75.1	43.2
Diabetic Patient (%)	42.9	49.5	33.4	47.2
BMI at Registration	28.2	5.9	27.6	5.7

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 on the waiting list face extreme scarcity, with a significant portion of patients dying while waiting for a transplant. Table 1 describes the sample of patients and their transplant and survival outcomes. An average of 15956 patients from our sample registered each year on the kidney waiting list. Panel A shows that 27.4% of patients who join the list die within five years of registering, while only 47.2% receive a transplant during this time period. The chances of receiving a transplant decline after the first five years as only 54% of the full sample of patients ultimately receive a deceased donor kidney. The remaining patients either still await a kidney or leave the list.

Panel B shows that patients receiving a transplant from a deceased donor 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. These observations are consistent with

long waiting times and the hypothesis that differences in these characteristics correlate with longer survival without a transplant.

Table 2: Donor Characteristics

	All Donors		Any Kidney Discarded			
			Yes		No	
	Mean	S.D.	Mean	S.D.	Mean	S.D.
Number of Donors per Year	6181		1169		5012	
Median Number of Offers per Donor	51		482		40	
Average Number of Offers per Donor	543.5	1927.9	1890.5	3684.3	229.3	946.7
Donor Age	39.2	18.4	52.0	16.6	36.2	17.5
Cause of Death -- Head Trauma (%)	39.7	48.9	19.5	39.6	44.5	49.7
Hypertensive Donor (%)	28.6	45.2	55.4	49.7	22.4	41.7
Donor Creatinine	1.2	1.0	1.4	1.1	1.1	0.9
Non-Heart Beating Donor (%)	7.9	26.9	10.4	30.6	7.3	26.0
KDPI	0.5	0.3	0.8	0.2	0.4	0.3

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 across donors, the mean number of biologically compatible offers is 543.5, but the median is much lower, at 51. This skewed distribution arises because undesirable kidneys are rejected by many patients, 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 an average of 1890.5 patients.

Predictors of organ quality are correlated with number of offers and discards in expected ways. Table 2 summarizes select donor characteristics by the allocation outcome for a donor's kidneys. Donors whose kidney(s) was/were discarded are older, less likely to die of 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. The transplantation community aggregates these and other indicators of quality into the Kidney Donor Profile Index (KDPI), which is the percentile of the estimated risk of graft failure of a donor's organ.[9]

[9]See https://optn.transplant.hrsa.gov/resources/guidance/kidney-donor-profile-index-kdpi-guide-for-clinicians/.

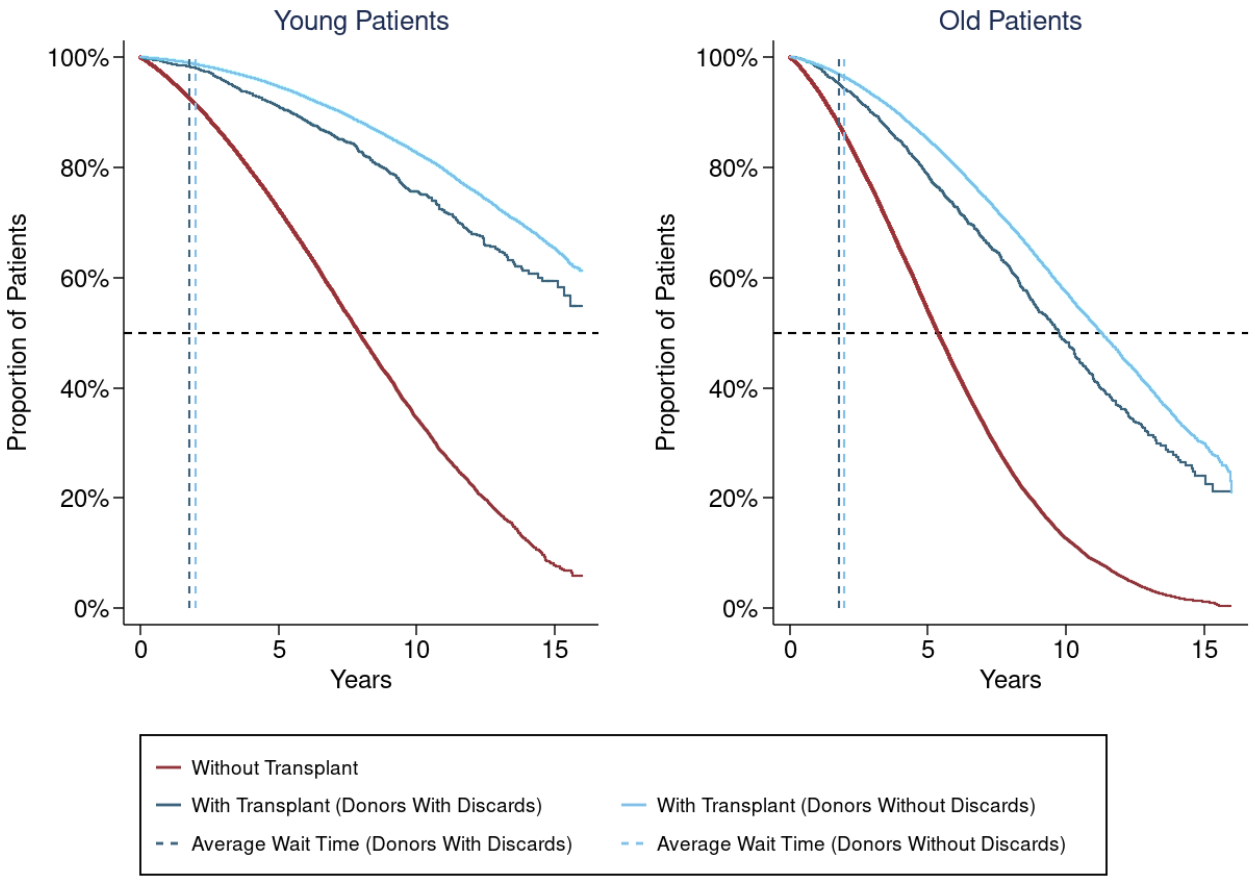
Survival

Our study will 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. As we will show below, ESRD patients who do not receive a transplant have a life expectancy of about half of those that do. 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 (non-parametric) Kaplan-Meier survival curves for patients who receive a transplant and those who do not. We separate the survival curves for young and old patients (above/below the median age of 54) and for patients who received a kidney from donors whose other kidney was discarded. Most deceased donors can donate both kidneys, which can benefit two different patients. Sometimes only one patient accepts a donors' kidneys. In those cases, one kidney is transplanted, and the other one is discarded. We can measure the survival of the patient who accepted the kidney which is likely to be undesirable. The vertical dashed lines depict the average waiting time for organs from the two groups of donors. As expected, 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.

These survival curves show that transplanted patients live significantly longer than patients who do not receive a transplant. Moreover, these survival curves 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

Figure 1: Patient Survival



Notes: The figure shows Kaplan-Meier survival curve for young and old patients (above/below the median age of 54) who registered on the waitlist between 2000 and 2010. Survival with transplant is measured as time since registration.

patients have shorter half-lifes both with and without a transplant. In fact, some young patients survive more than eighteen years, which is rare for an older patient.[10] For both groups of patients, a transplant using an undesirable organ is associated with half-lives that are shorter by about a year or more.

Taken together, these observations point to the potential for choices and assignments to be correlated with survival outcomes. Choices are important because discards occur

[10]In our sample, 61.2% young patients and 21.0% old patients who received a desirable organ survived more than 16 years. We cannot track survival outcomes for any longer than sixteen years because the earliest cohort in our study registered in the year 2000, and our survival data are up to date as of December 31, 2015. This fact also motivates our focus on median survival half-lives instead of expected life-years – the former does not depend on the right-tail of survival outcomes. This focus is also consistent with prior work measuring the life-year benefits from transplantation (see Wolfe et al., 1999, 2008, for example).

only when many patients have refused the organ. Next, we turn to a model that incorporates these features.

3 A Model of Decisions and Outcomes

Our model considers assignment mechanisms in which objects, indexed by j, are assigned to agents, indexed by i. When an object arrives, offers are made to agents on a waiting list who must decide to accept or reject it. These decisions translate into an assignment, and an outcome is realized. We now describe the mechanism, observed outcomes, and the primitives of our model in further detail.

3.1 Assignment Mechanism and Observed Outcomes

Objects arrive sequentially, their index j denotes their arrival order, and the mechanism assigns each one as follows. It orders agents on the waiting list according to a priority score that may be object specific and depend on the time that an agent has waited. Offers are made in priority order and each agent may decide to accept or reject the object. We denote the acceptance of an offer by i of object j with $D_{ij} = 1$. Objects are assigned to the highest priority agents that accept an offer for the object. The mechanism may elicit multiple decisions at once, but agents may not be skipped. Finally, agents that have been assigned an object are removed from the list. Other agents may also leave the list.

Now consider the set of objects that are feasible for a given agent. Holding fixed the decisions of the other agents, define J_i to be an ordered set of objects offered to agent i if the agent refuses all offers made to her and the agent participated in the mechanism indefinitely. Because we allow agents to depart from the list prior to assignment due to death, an agent may only receive a subset of offers which we denote by $\tilde{J}_i$. In our context, for example, patients receive offers until they die. Thus, we focus on the case where the ordered sublist $\tilde{J}_i = (j \in J_i : Y_{0i} \geq t_{ij})$, where t_{ij} is the time between agent i's registration date and donor j arrival. That is, $\tilde{J}_i$ is an ordered set of objects that the

agent would have been offered prior to departure from the list if she refused all objects. The object that an agent is assigned depends both on the feasible set of objects and her decisions. Specifically, let $T_{ij} = 1$ denote agent i being assigned object j. Indexing objects in sequence of arrival, we have that

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

where $D_{ij} = 1$ if agent i accepts object j. Therefore, each agent i is assigned to the first object that she accepts from the set $\tilde{J}_i$.

The outcomes we observe are determined by whether or not an agent is assigned and to which object she is assigned. The observed outcome is

$$Y_i = \sum_{j \in \tilde{J}_i} T_{ij} Y_{ij} + \left(1 - \sum_{j \in \tilde{J}_i} T_{ij}\right) Y_{i0},$$

where Y_{ij} is the outcome of agent i from being assigned object j, and Y_{i0} is the outcome for agent i if the agent is not assigned any object.

This formulation abstracts away from potential truncation of the observed survival outcome for simplicity of notation. That is, if agent i is assigned to object j then we observe Y_{ij}. Otherwise, we observe Y_{i0}. In our empirical context, we observe a censored survival outcome for some set of patients. For these patients, we will be able to deduce that $Y_i > Y_i^C$, where Y_i^C is the censoring time. Our approach will account for the fact that we have access to censored data on outcomes. Throughout, we will make the standard assumption that the duration for censoring is independent of the true 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 agent i if the agent is not assigned any object

is given by

$$Y_{i0} = g_0\left(x_i, \nu_{i,0}\right), \tag{3.1}$$

where $x_i \in \mathbb{R}^{d_x}$ are agent-specific observables; $\nu_{i,0} \in \mathbb{R}$ denotes an agent-specific unobservable; and $Y_{i0} \in \mathbb{R}$.

Assignment Outcome: The outcome of agent i from being assigned object j is given by

$$Y_{ij} = g_1\left(q_j, x_i, \nu_{i,1}, \varepsilon_{ij,1}\right), \tag{3.2}$$

where $x_i \in \mathbb{R}^{d_x}$ is a vector of agent-specific observed characteristics; $q_j \in \mathbb{R}^{d_q}$ denotes the type of object j; $\nu_{i,1} \in \mathbb{R}$ denotes an agent-specific unobservable; $\varepsilon_{ij,1} \in \mathbb{R}$ denotes an unobservable that are agent- and object-specific; and $Y_{ij} \in \mathbb{R}$.

Since Y_{ij} and Y_{i0} 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 of outcomes 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 agent i and object j arrive.

Decision Equation: We model the acceptance decision as

$$D_{ij} = g_D\left(q_j, x_i, z_i, \nu_{i,D}, \varepsilon_{ij,D}\right) \in \{0, 1\} \tag{3.3}$$

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

The choice model nests several primitive models of decisions. It is consistent with both myopic decision rules and a dynamic decision process in which agents do not know which specific organs will be offered in the future, but are likely to base their decisions on the distribution of future offers that they expect. Although we do not need to commit to

a specific model of choice, Agarwal et al. (2021) describe an optimal stopping problem that yields our decision equation as the optimal choice rule. Specifically, an offer is accepted if the (perceived net present) value from accepting the organ exceeds the option value of waiting.[11]

The main difference between X_i and Z_i is that the latter is excluded from the outcome equations described above.[12] 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 the given organ. This exclusion restriction, combined with Assumption 1(i) below, introduces instruments in the model that we will use in the empirical strategy. Identification results and the specific instruments Z_i used in our application are further discussed in Section 4.

Throughout the paper, we will make the following assumptions:

Assumption 1. *(i)* $\{\varepsilon_{ij} = (\varepsilon_{ij,1}, \varepsilon_{ij,D})\}_j$, $\nu_i = (\nu_{i,0}, \nu_{i,1}, \nu_{i,D})$, *and* Z_i *are mutually independent conditional on* x_i *and* $(q_j)_j$.

(ii) The random vector ν_i *is distributed iid across* i.

(iii) The random vector ε_{ij} *is distributed iid across* i *and* j.

Assumption 1(ii) and 1(iii) describes our sampling process. This process allows for dependence between the components of ν_i and the components of ε_{ij}, thereby allowing for Y_{ij} and Y_{i0} to be correlated with each other and with D_{ij}. The independence assumptions imply that agents' outcomes do not depend on other agents' treatment assignment, which implies the stable unit treatment value assumption (SUTVA).

The model and Assumption 1 together impose three main restrictions. First, unobserved agent selectivity, $\nu_{i,D}$ is fixed across all objects and time. Thus, agents can be ordered by unobserved selectivity. Second, selectivity and survival outcomes can be correlated through ν_i, but we abstract away from time-varying information about survival

[11] For example, one model of decisions consistent with our specifications is that $g_D = 1$ if $U_{ij} > V(t_{ij})$ where U_{ij} is the net present value of accepting the offer, $V(t_{ij})$ is the option value of waiting, and t_{ij} is the waiting time of agent i when type j is offered.

[12] We use uppercase letters to denote the random variables that describe the process of sampling from the patient population and reserve lowercase letters to denote their realized values.

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, an agent's decision does not depend directly on the specific decisions of other agents for a given object since ν_i and ε_{ij} are independent of $\nu_{i'}$ and $\varepsilon_{i'j'}$.

In addition, we rule out statistical dependence between J_i and the unobservables $\nu_{i,D}$ and $\varepsilon_{ij,D}$, conditional on x_i:

Assumption 2. *The sequence of offers J_i is conditionally independent of (ν_i, ε_i) given x_i.*

Assumption 2 is satisfied if x_i controls for a sufficiently rich set of agent types such that the remaining variation in potential offers for an agent is independent of unobserved determinants of outcomes and decisions. Thus, J_i can serve as an instrument under this assumption since it affects when and which type of organ is transplanted to a patient but is excludable from the potential outcomes. The assumption parallels the exclusion restriction required for instrument validity. Section 4.1 below argues that it is plausible in our empirical application.

An implication of this assumption is that, agents cannot alter their decisions or their outcomes in response to specific future offers. This rules out foresight over the objects that will be offered in the future as is the case in our empirical setting.[13] Nonetheless, we stress that the model does accommodate forward-looking agents interacting in a dynamic mechanism who strategically refuse offers because the future offers are likely (see Agarwal et al., 2021).

The sequential nature of choices and treatment assignment in our model resembles that of Heckman and Navarro (2007). There are two main differences. First, treatments within a type q_j are heterogeneous in our framework. That is, the decision D_{ij} and the

[13]For the analysis of duration models in particular, the assumption parallels the "no anticipation" assumption in Abbring and Van den Berg (2003) which requires that agents cannot act in order to influence their outcomes in anticipation of a treatment. As Heckman and Navarro (2007) note, this assumption is natural in a fully-specified duration model.

outcome Y_{ij} and may differ from $D_{ij'}$ and $Y_{ij'}$ even if $q_j = q_{j'}$. In our empirical context, 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 problems when objects are highly heterogeneous. 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 in our model.

3.3 Sources of Selection

The model allows for selection into transplantation on three dimensions: untransplanted survival Y_{i0}; average survival across transplants $\bar{Y}_i = \frac{1}{|J|}\sum_j Y_{ij}$; and match-specific survival $Y_{ij} - \bar{Y}_i$. There are two potential sources of selection: selection due to patient choices, selection due to patient mortality.

Selection due to choice occurs if patients' choices D_{ij} are correlated with survival outcomes Y_{i0} or Y_{ij}. Patient choice can induce selection on Y_{i0} if, for example, patients with higher expected survival without a transplant due to unobserved health conditions are more selective. That is, if $E\left(Y_{i0}|\nu_{i,D}, x_i\right)$ varies with $\nu_{i,D}$. Similarly, choice can induce selection on average transplanted survival, $\bar{Y}_i$, if $E\left(Y_{ij}|\nu_{i,D}, x_i\right)$ varies with $\nu_{i,D}$. Choice can also induce selection on match-specific survival if patients are more likely to accept an organ with an idiosyncratic survival benefits $Y_{ij} - \bar{Y}_i$. These sources of selection are generalized versions of Roy (1951) selection.

Selection due to patient mortality occurs because patients with better untransplanted survival outcomes Y_{i0} can stay in the waitlist longer and have a higher chance of receiving a transplant. Moreover, these patients may also have better transplanted survival outcomes due to either time-to-treatment effects or correlation between ν_{i0} and ν_{i1}. Our model features mortality-induced selection because $\tilde{J}_i$ only includes organs that arrive prior to Y_{i0}. This type of selection also depends on the extent to which the mechanism prioritizes offers to patients with longer waiting times.

These sources of selection on unobservable patient characteristics imply that T_{ij} is

potentially correlated with unobserved factors that determine outcomes. Comparing survival with and without a transplant will then yield biased estimates of the causal effect of a transplant. The aim of the instruments discussed in Section 4 is to address the resulting endogeneity concerns.

Finally, note that transplanted and untransplanted patients also differ on observable characteristics for two reasons: the dependence of D_{ij} on x_i, and the dependence of the offer sequence, J_i, on the mechanism's priority rules and the set of patients on the waiting list. For example, priority is given to patients that have sensitive immune systems in order to give them greater transplantation opportunities. These patients may have survival benefits that differ from others.

4 Instruments

Our solution to the selection problems discussed above requires two sources of variation. We describe each of these in turn. Section 5 will formally prove identification under these two sources of variation.

4.1 Conditionally Independent Potential Offers

The first source of variation we will exploit arises from randomness in the objects offered to an agent, relying on Assumption 2. We now 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 on the waiting list, and determinants of the agent's priority and points on the list. It does not depend on the specific decisions made by agent i. Our knowledge of the mechanism allows us to construct rich controls x_i for each patient's priority. Conditional on these 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 (ν_i, ε_i) because it depends primarily on deaths in the local area. And, as we argued in Section 3 above, the decisions of other agents are independent of (ν_i, ε_i) in a natural equilibrium model of the the waiting list.

While we will use the full set of offers to estimate the model, we now use a specific function of J_i to investigate this source of variation. 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 a patient, denoted 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.[14]

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 when the organ arrived. Moreover, the results below use fixed effects to control for differences in a patient's priority, geographical area, and time trends. We therefore need to argue that Assumption 2 is satisfied for this variable conditional on these controls. We claim that the first source of variation is independent of patient i's decisions because specific patients are not considered in organ donation decisions. The second source of variation is also plausibly exogenous because a given patient's decision is unlikely to affect the priority of the patient ranked in the tenth position.[15] Consistent with these claims, Table D.5 in the appendix shows that our measures are not significantly correlated with the vast majority of various patient characteristics (age, diabetes, female, height, and weight).

[14] We include a donor in the calculation irrespective of whether the patient accepted a prior offer or departed from the list during the period. Throughout, we restrict attention to blood type-compatible donors that arrived in the same donor area and assume a fixed waiting time of two years.

[15] The only potential effect is if patient i, in our sample, 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.

Given this exclusion restriction, we now turn to showing how this measure of a patient's potential offers is related to transplants. These correspond to the first-stage relationships in a linear instrumental variables model. Columns (1) to (4) in Table 3 present estimates from linear probability models to examine the relationship between the number of potential top 10 offers from donors that are either above or below median quality (as measured by KDPI) and transplant outcomes. All models include fixed effects for the patient's donor service area (DSA), year of registration, blood type, and determinants of priority.

Table 3 shows that potential offers strongly influence whether or not a patient receives a transplant as well as the type of organ transplanted. 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 depends on the number of potential offers from the corresponding type of donor. Specifically, a patient with a greater number of potential offers from above median quality organs is more likely to receive a transplant from such an organ. Conversely, the probability of a transplant from a below median organ decreases with more offers from above median quality organs. An analogous relationship holds for offers from below median quality donors. The F-statistic is large and much higher than the conventional cutoff of 10 used to assess whether an instrument is strong (Stock and Watson, 2012). Therefore, the evidence points to a strong first-stage relationship.

4.2 A Choice Shifter: Scarcity

The second source of variation that we leverage is based on instruments that alter an agent's acceptance decision but are independent of latent outcomes. In the model, the variables z_i affect the decisions, D_{ij}, but are excluded from the functions $g_1(\cdot)$ and $g_0(\cdot)$. Moreover, Assumption 1(i) requires that, conditional on x_i, (ν_i, ε_i) is distributed independently of z_i. Therefore, these instruments are useful in identifying the model as they can be used to vary the selectivity of patients while holding survival outcomes

Table 3: Top 10 offers: First Stage

	Transplant			
	Any Kidney (1)	Any Kidney (2)	KDPI <= 50% (3)	KDPI > 50% or Missing (4)
log(1 + # Top 10 Offers in 2 Years)				
KDPI <= 50%	0.0322***	0.0334***	0.0439***	-0.0105***
	(0.00441)	(0.00441)	(0.00306)	(0.00287)
KDPI > 50% or Missing	0.0303***	0.0297***	-0.0128***	0.0425***
	(0.00475)	(0.00478)	(0.00314)	(0.00294)
DSA FE, year FE, and blood type FE	x	x	x	x
Control for Pediatric at Listing	x	x	x	x
CPRA Category Controls	x	x	x	x
Patient Characteristics		x	x	x
F-statistic	93.20	92.23	108.0	130.6
Number of Observations	132715	131105	131105	131105
R-Squared	0.210	0.219	0.171	0.065

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.

fixed.

The instruments that we construct for our setting are motivated by the observation that patients face an optimal stopping problem (Agarwal et al., 2021). Therefore, two otherwise identical patients who place different option values on waiting will make different acceptance decisions even when offered the same type of organ. In particular, 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). The scarcity instruments we need to construct must be correlated with decisions but independent of latent outcomes.

We construct two measures of scarcity as choice shifters. The first is a predictor of

offers a patient can expect 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 the relative scarcity of organs in a patient's location while controlling for secular trends across locations. In order to evaluate the assumption that (ν_i, ε_i) are distributed independently of z_i, conditional on x_i, 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). The threat to the instrument therefore needs to be a DSA-specific trend in scarcity that correlates with outcomes for some reason that is not reflected in patient characteristics.

These instruments are relevant to decisions if these ex-post measures are correlated with beliefs about future offer probabilities. This hypothesis is based on the fact that transplant surgeons advise patients on how to respond to an offer and are likely aware of the recent availability of kidneys in their geographical area. 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 two measures of scarcity and a variety of controls. The sample is restricted to the first one hundred offers made for a donor. 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 in the comparison group in the four quarters is negatively correlated with acceptance rates, controlling for patient priority

Table 4: Scarcity Instruments: First Stage

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

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), which is consistent with balance of the scarcity instrument. These patterns are robust to additional controls for donor and match-specific characteristics, although the estimated effect of scarcity on acceptance is slightly lower (columns 5 through 8). Figure D.1 in the appendix shows a residualized binscatter plot suggesting that this relationship is monotonic.

These results indicate that our measure of scarcity has the expected relationship with patient acceptance decisions and that the balance conditions are consistent with the required exclusion restrictions.

5 Identification and Estimation

The previous section introduced two sources of variation that are orthogonal to an agent's latent outcomes Y_{i0} and Y_{ij} – the potential offers that an agent could receive and the scarcity faced by an agent, z_i. We now show that these two sources of variation can be used to identify the decision model and measure the sources of selection discussed in Section 3. Our results condition on the agent type x_i and omit it for simplicity of notation. We assume the analyst observes or can construct the offer sequence J_i,[16] the object types q_j, the choices D_{ij} and the survival outcomes.

The argument proceeds in four parts. First, we show that variation in the offers received by an agent can be used to learn the expected outcomes and certain treatment effects conditional on the value of scarcity, assignment status, and the sequence of offer types. Second, we show that the choice model described in equation (3.3) is identified conditional on scarcity. Third, we combine the variation in scarcity with results from the first part to identify the effect of key unobservables on the distribution of outcomes. The second and third parts consider the case when all objects arrive at the same time as the agent. The fourth part relaxes this restriction. All proofs are in Appendix C.

5.1 Identifying Conditional Expected Outcomes

The first result shows what can be learned about expected outcomes using variation in offers. Let j_i be a realization of J_i and $j_{i,n}$ denote the n-th object offered to i. The sequence of offer types associated with j_i is $q_{j_i} = \left(q_{j_{i,1}}, \ldots, q_{j_{i,|j_i|}}\right)$. Thus, q_{j_i} is a realization from the distribution of offer-types induced by J_i. Our first result shows that variation in the offer-types can identify the average treatment effect on the treated:[17]

[16] In our empirical context, we can construct the offer sequence using knowledge of the mechanism and data on the offers made for each donor. Specifically, for every donor j and patient i, we determine whether the priority of patient i is higher than the lowest priority patient that was offered an organ from the donor. If so, patient i would have received an offer for donor j's organ were she waiting when donor j arrived.

[17] Observe that our model and setting do not allow for always takers since an agent cannot be assigned an object without receiving an offer for one.

Lemma 1. *Suppose that Assumptions 1 and 2 are satisfied. Fix z and q_{j_i}. The quantities $E\left[Y_{ij_{i,n}} \middle| T_{ij_{i,n}} = 1, q_{j_i}, z\right]$ and $E\left[Y_{i0} \middle| T_{ij_{i,n}} = 1, q_{j_i}, z\right]$ are identified for all $n \leq |j_i|$ such that $P\left[T_{ij_{i,n}} = 1 \middle| q_{j_i}, z, Y_{i0} \geq t_{j_{i,n}}\right] > 0$, and $\left(q_{j_{i,1}}, \ldots, q_{j_{i,n}}\right)$ and $\left(q_{j_{i,1}}, \ldots, q_{j_{i,n-1}}\right)$ belong to the support of the distribution of offer-types induced by the distribution of J_i.*

This result shows that we can identify the expected outcomes with and without assignment for agents with an offer-type sequence q_{j_i} who were assigned to the $n-$th object offered. The difference between these two expectations is the treatment effect on the treated. Assignments of agents with the offer-type sequence q_{j_i} allows us to directly observe $P\left[T_{ij_{i,n}} = 1 | q_{j_i}, z, Y_{i0} \geq t_{j_{i,n}}\right]$. Additionally, since we observe the outcome $Y_{ij_{i,n}}$ for agents with $T_{ij_{i,n}} = 1$, we also observe $E\left[Y_{ij_{i,n}} | T_{ij_{i,n}} = 1, q_{j_i}, z\right]$. The challenge is to recover the expected value of Y_{i0} for the group of agents who would have been assigned the $n-$th offer had they received the offer-type sequence q_{j_i}. We construct this quantity using the expected outcomes of unassigned agents with offer-type sequences $\left(q_{j_{i,1}}, \ldots, q_{j_{i,n}}\right)$ and $\left(q_{j_{i,1}}, \ldots, q_{j_{i,n-1}}\right)$. The former group only includes agents with $T_{ij_{i,n}} = 0$ while the latter group includes agents with both values of $T_{ij_{i,n}}$with known probability $P\left[T_{ij_{i,n}} = 1 | q_{j_i}, z, Y_{i0} \geq t_{j_{i,n}}\right]$. These three quantities can be combined to recover $E\left[Y_{i0} | T_{ij_{i,n}} = 1, q_{j_i}, z\right]$.

Although our formal result is stated for the conditional expectations of the outcome variables, we can identify the marginal distributions of Y_{i0} and $Y_{ij_{i,n}}$ conditional on $T_{ij_{i,n}} = 1$, q_{j_i} and z. This follows from Lemma 1 because we can identify the conditional expectation of $\psi\left(Y_{i0}\right)$ and $\psi\left(Y_{ij_{i,n}}\right)$ for any bounded function ψ. This result is similar in spirit to those in the treatment effects literature (e.g. Imbens and Angrist 1994; Heckman et al., 2010). A similar estimand has been the target in Abdulkadiroglu et al. (2017) where offers in a school choice mechanism are used as instruments to estimate treatment effects.

This result allows us to compare expected outcomes under the observed assignments to an alternative in which no agent is assigned. In our application, it allows us to evaluate the additional life-years gained due to the observed deceased donor transplants. The limitation is that these transplants are selected. Specifically, the types of offers an

agent receives and the scarcity faced z introduce selection on the distribution of $\nu_{i,D}$ conditional on transplant. For example, two agents with the same offer-type sequence who are assigned to the $n-$th and the $(n+1)-$st offers likely differ in their selectivity, $\nu_{i,D}$. Identifying $E\left[Y_{ij_{i,n}}|T_{ij_{i,n}}=1,q_{j_i},z\right]$ and $E\left[Y_{i0}|T_{ij_{i,n}}=1,q_{j_i},z\right]$ is therefore not sufficient for evaluating the expected values of $Y_{ij_{i,n}}$ and Y_{i0} under a counterfactual assignment of kidneys to patients. Obtaining counterfactual predictions requires recovering the distribution of outcomes conditional on unobservables, as we do below.

5.2 Identifying the Choice Model

Our next result shows that we can use the variation in offers also to identify the function $g_D(\cdot)$.[18] To fix ideas, focus on donor types whose arrival time coincides with the patient arrival time. That is, if t_{ij} denotes the time difference between donor arrival and patient arrival, we focus on the case when $t_{ij}=0$ so that the distribution of ν_i is not selected due to survival while waiting on the list. This assumption is relaxed in section 5.4 below. We normalize the marginal distributions of $\nu_{i,D}$ and $\varepsilon_{ij,D}$ to be uniform and assume that z is supported in the unit interval. These normalizations are without further loss of generality since $\nu_{i,D}$ and $\varepsilon_{ij.D}$ are univariate because we have not yet placed restrictions on the functional form of $g_D(\cdot)$.

We need to introduce more notation in order to develop our result. For each value of z and donor type q_j, consider two sets of pairs (ν_D,ε_D) such that one set yields $g_D(q_j,z,\nu_D,\varepsilon_D)=0$ and the other yields $g_D(q_j,z,\nu_D,\varepsilon_D)=1$. Figure 2 illustrates the regions for two representative values of $z\in\{z_{low},z_{high}\}$. The function $v(\varepsilon_D;q_j,z)$ separates these two sets.[19] Therefore, identifying the function $v(\varepsilon_D;q_j,z)$ is equivalent to identifying $g_D(\cdot)$.

Our results make the following assumptions on $v(\cdot;q_j,z)$:

[18] The function $g_D(\cdot)$ can be derived from micro-funded binary choice models with mean utilities that depend on $(q_j,x_i,z_i,\nu_{i,D})$ and additive errors (Cosslett, 1983; Matzkin, 1991).

[19] Formally, define $v(\varepsilon_D;q_j,z)=\sup\left\{\nu_D\in[0,1]:g_D(q_j,z,\nu_D,\varepsilon_D)=1\right\}$, where we adopt the convention that the supremum of the empty set is 0.

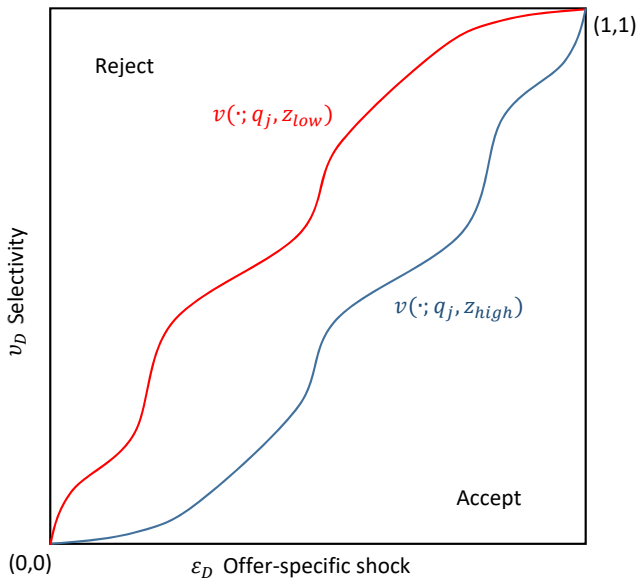


Figure 2: Acceptance and Rejection Regions

Assumption 3. *For each q_j and z, the function $v(\cdot; q_j, z)$ is differentiable and its image is the unit interval.*

This assumption requires that extreme values of ε_D move any agent's decision from accept to reject or vice-versa given a fixed value of z_i and $\nu_D \in (0, 1)$. Because g_D is non-increasing in ν_D and non-decreasing in ε_D, $v(\cdot; q_j, z)$ is a weakly monotone function. We can interpret $v(\varepsilon_D; q_j, z)$ as the fraction of agents that reject an offer of type q_j with probability at least ε_D when faced with scarcity z. Therefore, the assumption requires that agent selectivity cannot overwhelm the effects of idiosyncratic preferences. If it did, then there would be (interior) values of ν_D that would yield a degenerate acceptance probability for a given value of z. In addition, the assumption places a weak smoothness restriction on $v(\cdot; q_j, z)$.

With these assumptions, 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 $t_{ij} = 0$, 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 $\mathbb{P}(D_{ij} = 1 | \nu_{i,D} = \nu_D)$ are identified.*

The main challenge for identification is that there are two latent reasons that drive an agent's decisions, namely $\nu_{i,D}$ and $\varepsilon_{ij,D}$. We must also identify how each of these map to acceptance decisions. For any n, we observe the probability $P\left(D_{ij_{i,1}} = \ldots = D_{ij_{i,k}} = 0 | q_j^n, z\right)$ for all $k \leq n$. Because $v(\varepsilon_D; q_j, z)$ is equal to the fraction of agents that reject an offer of an object with type q_j with probability at most ε_D when faced with scarcity z, we can write

$$P\left(D_{ij_{i,1}} = D_{ij_{i,2}} = \ldots = D_{ij_{i,k}} = 0 | q_j^n, z\right) = \int_0^1 \varepsilon_D^k \mathrm{d}v(\varepsilon_D; q_j, z).$$

Therefore, the quantity $P\left(D_{ij_{i,1}} = D_{ij_{i,2}} = \ldots = D_{ij_{i,k}} = 0 | q_j^n, z\right)$ is the k-th moment of a random variable with cumulative distribution function $v(\cdot; q_j, z)$. The problem of recovering this function is therefore equivalent to solving the Hausdorff moment problem (Casella and Berger, 2002). That is, we need to learn the CDF $v(\cdot; q_j, z)$ with information on its moments. This can be done if infinitely many moments are known.

In fact, our result is stronger: it shows that data with finite n is informative even without variation in the number of offers. Formally, our result implies that $v(\cdot)$ can be well-approximated by observing decisions from a *given* sequence of offer-types q_j^n. We accomplish this by showing that the moments described above determine the n-th order Fourier-Legendre approximation of $v(\cdot)$. Using a result in Pollard (1947), we show that as n becomes large, this approximation converges to the true function $v(\cdot; q_j, z)$ in the L^2 norm.

5.3 Identifying Selection on Unobservables

Next, we turn our attention to identifying the components that determine selection on unobservables. This result requires 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, bounded, and strictly positive function of* $\varepsilon_D \in (0,1)$*.*

(ii) For each z *and* q_j*, the functions* $E(Y_{i0}|\nu_D)$ *and* $E(Y_{ij}|\nu_D, \varepsilon_{ij,D} \geq \varepsilon_D, q_j)$ *are continuous, and the first and second moments of* Y_{i0} *and* Y_{ij} *exist.*

The first part strengthens the monotonicity and differentiability of $v(\varepsilon_D; q_j, z)$ imposed in Assumption 3 by requiring a strictly positive and bounded derivative. Given our interpretation of $v(\cdot)$, observe that $v'(\cdot; q_j, z)$ is the density function of the distribution of the probability with which an agent rejects an offer of an object with type q_j. Therefore, the assumption requires that this density function is bounded and is non-zero for all interior values of ε_D and z. The second part imposes weak regularity assumptions on conditional expectations and the moments of Y_{i0} and Y_{ij}.

With this assumption, we can identify the components resulting in selection:

Theorem 1. *Suppose that Assumption 4 and the hypotheses for Lemma 2 hold for all* n*. Then, the quantities* $E[Y_{i0}|\nu_{i,D} = \nu_D]$ *and* $E[Y_{ij}|\nu_{i,D} = \nu_D, \varepsilon_{ij,D} \geq \varepsilon_D]$ *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* $\nu_D = v(\varepsilon_D; q_j, z)$*.*

This result shows non-parametric identification of the expected value of outcomes conditional on values of selectivity and idiosyncratic preferences. The proof begins by using results in Lemma 1 to identify the conditional expectations given scarcity z, offer-types and assignment. Next, the proof rewrites these quantities in terms of the primitives and uses arguments similar to those in Lemma 2 to recover quantities that depend on both the outcomes model and the choices model. Next, we use the identification results for $v(\cdot)$ in Lemma 2 to recover the objects of interest. For example, Lemma 1 implies

that $E\left(Y_{i0} \times 1\left\{T_i = 0\right\}|q_j^k, z_i\right)$ is identified from variation in offers. This quantity can be re-written as

$$E\left(Y_{i0} \times 1\left\{T_i = 0\right\}|q_j^k, z_i\right) = \int_0^1 E\left(Y_{i0}|\nu_D = v\left(\varepsilon_D; z_i, q_j\right)\right)\varepsilon_D^k \mathrm{d}v\left(\varepsilon_D; z_i, q_j\right).$$

If we observe this quantity for all $k \leq n$, then we can recover the n-th order Fourier-Legendre approximation of $E\left(Y_{i0}|\nu_D = v\left(\varepsilon_D; q_j, z\right)\right)v'\left(\varepsilon_D; q_j, z\right)$ when viewed as a function of ε_D. Under the maintained assumptions, results in Talenti (1986) and Freud (1971) imply that the Cesàro mean of this series converges uniformly to the true function. Finally, since $v'\left(\varepsilon_D; q_j, z\right) > 0$ and bounded and the function $v\left(\varepsilon_D; q_j, z\right)$ is identified (Lemma 2), we can identify $E\left(Y_{i0}|\nu_D\right)$ for all $\nu_D \in (0,1)$ if we can find values of z and ε_D such that $v\left(\varepsilon_D; q_j, z\right) = \nu_D$. The intuition for identifying $E\left(Y_{ij}|\nu_D, \varepsilon_{ij,D} \geq \varepsilon_D\right)$ is similar in spirit, although a little more notationally involved.[20]

In this way, the scarcity instrument z is used to "trace-out" the expected values of Y_{i0} and Y_{ij} conditional on ν_D and ε_D (see also Heckman and Vytlacil, 2005; Lewbel, 2007; Heckman and Navarro, 2007). The instrument does this by changing the set of (ν_D, ε_D) whose treatment status changes in response to the offer instrument (see Figure 2). The proof relies on continuous variation in our scarcity instrument z in order to identify these quantities.[21]

There are two aspects of our result that are worth noting. First, we do not need scarcity instruments to vary at the individual treatment level. In our model, the discrete offer instrument generates variation in treatment assignments. This approach is an alternative to the analysis of generalized Roy models that typically use continuously varying choice shifters that are treatment specific (see Heckman and Navarro, 2007; Hull, 2018, for example). Our assumption that $\nu_{i,D}$ does not vary across j allows us

[20] Again, Lemma 1 implies that $E\left(Y_{ij} \times 1\left\{T_{ij_{i,k}} = 1\right\}|q_j^k, z_i\right)$ is identified. It can be re-written as $\int_0^1 E\left(Y_{ij}|\nu_D = v\left(\varepsilon_D; z_i, q_j\right), \varepsilon_{ij,D} \geq \varepsilon_D\right)\left(1-\varepsilon_D\right)\varepsilon_D^{k-1}\mathrm{d}v\left(\varepsilon_D; z_i, q_j\right)$. As done above, we use this expression to identify $E\left(Y_{ij}|\nu_D = v\left(\varepsilon_D; z_i, q_j\right), \varepsilon_{ij,D} \geq \varepsilon_D\right)\left(1-\varepsilon_D\right)v'\left(\varepsilon_D; q_j, z\right)$ and finally recover $E\left(Y_{ij}|\nu_D, \varepsilon_{ij,D} \geq \varepsilon_D\right)$ by finding a value of z such that $\nu_D = v\left(\varepsilon_D; q_j, z\right)$. One qualitative difference is that identifying $E\left(Y_{i0}|\nu_D\right)$ allows us to use variation in either z or ε_D to trace-out ν_D, whereas the result for $E\left(Y_{ij}|\nu_D, \varepsilon_{ij,D} \geq \varepsilon_D\right)$ must condition on ε_D.

[21] In the case of discrete instruments, it may be possible to obtain bounds on these treatment effects (see Mogstad et al., 2018, for example).

to work with the instrument z_i that varies only across agents i but is fixed across treatments j.[22] Second, we do not rely on values of z that push choice probabilities to degenerate values that obviate the selection problem. Specifically, $E\left[Y_{i0}|\nu_{i,D} = \nu_D\right]$ and $E\left[Y_{ij}|\nu_{i,D} = \nu_D, \varepsilon_{ij,D} \geq \varepsilon_D\right]$ are identified as long as we observe values of z such that $\nu_D = v\left(\varepsilon_D; q_j, z\right)$. Therefore, we do not rely on an "identification at infinity" argument. As is common, however, we can only identify the expected outcomes conditional on the latent variable ν_D for values of ν_D that are spanned by variation in the observable z. Moreover, it is easy to see that if the image of $v\left(\varepsilon_D; \cdot, q_j\right)$ across values in support of z is the unit interval, then we can identify the unconditional values of the latent outcomes, namely $E\left[Y_{i0}\right]$ and $E\left[Y_{ij}\right]$. Recall that this exercise implies identification of the analogous quantities for any bounded transformation $\psi\left(\cdot\right)$ of each of the outcomes, thereby implying identification of the full distribution, not just the mean.

5.4 Dynamic Selection

The results in Lemma 2 and Theorem 1 above apply to objects q_j that arrive at the same time as the agent ($t_{ij} = 0$). We now extend our results to the case when $t_{ij} > 0$. Considering negative values of t_{ij} is not necessary because patients cannot be assigned donors that arrived before them. The case with $t_{ij} > 0$ introduces two issues. First is the direct effect of time to treatment. This can be captured using observable covariates by including the registration date of a patient and the date of an organ's 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_{ij} is no longer unselected. In our empirical example, selectivity may be correlated with survival without a transplant. Therefore, we cannot normalize the marginal distribution to be uniform, except at $t_{ij} = 0$.

We now extend the identification results to account for this type of dynamic selection. While the results in the previous subsection allow Y_{ij} and Y_{i0} to denote arbitrary outcomes of interest, the results in this subsection explicitly assume that Y_{i0} denotes

[22] Relaxing the model to allow $\nu_{i,D}$ to vary across time or treatments would likely require commensurate variation in z_i.

survival. Therefore, we will assume that agent i may be assigned object j only if $Y_{i0} > t_{ij}$. 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 ν_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 $E(Y_{ij} | \nu_{i,D} = \nu_D, \varepsilon_{ij,D} \geq \varepsilon_D, Y_{i0} \geq t_{ij})$ are identified for 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 ν_D for agents that survive until time t. Lemma 6 in Appendix C.5 shows that this function is identified. The proof applies Theorem 1 to show identification of $P(Y_{i0} \geq t | \nu_D) = E[1\{Y_{i0} \geq t\} | \nu_D]$, which implies the identification of the conditional distribution of ν_D for agents that survive until time t.

The second step takes this conditional distribution and combines it with the arguments that parallel those in Lemma 2 and Theorem 1. Specifically, we first identify the function $v(\varepsilon_D; q_j, z_i)$, which delimits the acceptance region in the (ε_D, ν_D)-space, by using the cdf of the probability that a patient who survives until $t_{ij} > 0$ rejects a kidney of type q_j.[23] This result is analogous to Lemma 2 for the case when $t_{ij} > 0$. Then, we use arguments similar to those in Theorem 1.

Taken together with Theorem 1, the result implies the identification of the quantities $E(Y_{ij} | \nu_{i,D} = \nu_D, \varepsilon_{ij,D} \geq \varepsilon_D, Y_{i0} \geq t_{ij})$ and $E(Y_{i0} | \nu_{i,D} = \nu_D, Y_{i0} \geq t_{ij})$ where the conditioning on x_i and q_j has been subsumed for simplicity. These quantities are sufficient

[23] These two quantities were identical when $\nu_{i,D}$ was uniformly distributed on the unit interval, as in the previous subsections. This was the relevant case when $t_{ij} = 0$.

for evaluating the expected survival with and without a transplant for any agent under a counterfactual in which the agent is assigned object j by fiat. Because we have also identified $P\left(D_{ij}=1|\,\nu_{i,D}=\nu_D, Y_{i0}\geq t_{ij}\right)$, we can use Bayes' rule to identify the conditional distribution of υ_D for agents that have or have not received an assignment under the current mechanism by conditioning on their decisions and observed survival time. Combined with $E\left(Y_{ij}|\,\nu_{i,D}=\nu_D, \varepsilon_{ij,D}\geq\varepsilon_D, Y_{i0}\geq t_{ij}\right)$ and $E\left(Y_{i0}|\,\nu_{i,D}=\nu_D, Y_{i0}\geq t_{ij}\right)$, we can obtain the expected survival with and without a transplant conditional on observed decisions and survival duration. This latter quantity allows us to measure the selection induced due to mortality and the use of waiting time.

Theorem 2 applies to any bounded transformation $\psi\left(\cdot\right)$ of the outcomes, which implies identification of the marginal distributions and survival hazard functions of Y_{i0} and Y_{ij}. However, 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_{ij} on Y_{ij} 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.

5.5 Estimation

Although our results above show non-parametric identification of certain treatment effects, 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. Thus, restrictions on the survival process are necessary. Finally, we would like to incorporate correlations between discrete choices and these censored outcomes, resulting in additional covariance terms.

To solve these challenges, we employ a Gibbs' sampling technique to estimate a parametrized

version of equations (3.1) – (3.3).[24]

$$y_{i0} = B\left(Y_{i0};\rho_0\right) = x_i\beta_x + \nu_{i.0} \tag{5.1}$$

$$y_{ij} = B\left(Y_{ij};\rho_1\right) = \chi\left(x_i,q_j\right)\alpha_{x,q} + \alpha_\eta\eta_j + \nu_{i,1} + \varepsilon_{ij,1} \tag{5.2}$$

$$D_{ij} = 1\left\{\chi\left(x_i,q_j\right)\gamma_{x,q} + z_i\gamma_z + \eta_j - \nu_{i,D} + \varepsilon_{ij,D} > 0\right\}, \tag{5.3}$$

where Y_{i0} is survival since registration without a transplant; Y_{ij} is survival since transplantation if patient i is transplanted organ j; $B\left(\cdot;\rho\right)$ denotes a Box-Cox transformation of the argument with parameter ρ (Box and Cox, 1964);[25] $\chi\left(x_i,q_j\right)$ is a flexible function of agent observables x_i and object types q_j; η_j is distributed $\mathcal{N}\left(0,\sigma_\eta^2\right)$ with the parameter σ_η^2 to be estimated; $\varepsilon_{ij} = \left(\varepsilon_{ij,D},\varepsilon_{ij,1}\right)'$ is distributed $\mathcal{N}\left(0,\Sigma_\varepsilon\right)$ where $\Sigma_{\varepsilon,11}$ is normalized to 1; and ν_i is a mean-zero multi-variate normal with a distribution induced by the following factor structure:

$$\nu_{i,1} = \delta_{1,D}\nu_{i,D} + \nu_{i,f} \tag{5.4}$$

$$\nu_{i,0} = \delta_{0,D}\nu_{i,D} + \delta_{0,f}\nu_{i,f} + \tilde{\nu}_{i,0}, \tag{5.5}$$

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 factor structure is without loss given the normality of ν_i.

This empirical model maps the patient and kidney types into characteristic space, which allows us to keep the dimension of the parameter space manageable.[26] It also allows us to include η_j, which represents unobserved heterogeneity in organ quality. This term

[24] It is common to require 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).

[25] The Box-Cox transformation of y with parameter ρ is given by $B\left(Y;\rho\right) = \frac{Y^\rho - 1}{\rho}$. A special case when $\rho = 0$ is $B\left(Y,\rho\right) = \log Y$. We set ρ by comparing an estimated survival curve using the non-parametric Kaplan-Meier estimator to those implied by assuming that $B\left(Y,\rho\right)$ is normally distributed. Robustness of our headline results to our chosen values is presented in Table D.14.

[26] Our framework can also accomodate coarsely defined patient and donor types. In this case, x_i and q_j would be a vector indicating to which a patient or donor belongs. As is common, introducing a very large number of types would result in imprecise results.

can be seen as capturing the cumulative effect of organ characteristics observed by patients and surgeons but not included in the empirical specifications.[27]

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 $\varepsilon_{ij,1}$ and $\varepsilon_{ij,D}$. For example, the factor $\nu_{i,f}$ captures the component of a patient's unobserved frailty that is not correlated with decisions. If $\delta_{0,f}$ is small or negative, then, all else being equal, transplanting a patient with lower frailty (higher $\nu_{i,f}$) results in higher survival benefits. Second, the decision model is similar to the probit binary choice used in Agarwal et al. (2021) for the kidney waitlist. These two considerations direct us to use multivariate normals to model the distributions of ν_i and ε_{ij}. 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 ρ_0 and ρ_1 fixed and conduct robustness analysis to alternative choices.

Directly computing and maximizing the likelihood of this model is difficult because the likelihood for each patient's data depends on the decisions over many donors as well as (potentially censored) survival outcomes. Computing this 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 θ, and the latent variables ν_i, ε_{ij}, and η_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.[28] The distribution this method generates is asymptotically equivalent to that of

[27] Agarwal et al. (2021) argue, using an analogy to measurement error models (see Kotlarski's Theorem in Rao, 1992; Hu and Schennach, 2008), that the distribution of this variable can be identified based on the correlation between acceptances of a given donor's first and second kidney. For consistency with the formal results presented in this paper, we will also estimate models that exclude this term.

[28] These considerations also motivate Geweke et al. (2003) to use a similar parametrization and estimation approach when studying hospital quality in a selection model.

the maximum likelihood estimator since the influence of prior distributions vanishes in a large sample (see van der Vaart, 2000, Theorem 10.1 (Bernstein-von-Mises)). We thus interpret our estimator as equivalent to the maximum likelihood estimator. Details on the method are provided in Appendix B.1.

An advantage of our approach is that it allows a rich set of patient-level covariates x_i and organ types q_j to be included in the model. This richness is important for understanding the extent to which observables can capture the selection on outcomes induced by choices. The cost of this approach is its somewhat heavier reliance on parametric assumptions and computational burden. For example, Hull (2018) studies a semi-parametric model and proposes a indirect inference method that targets a subset of quasi-experimental moments that can be identified directly in a first step. The main drawback of this alternative for our purposes is that the number of moments that need to be estimated in the first step increases with the dimension of the parameter space, making it hard to include the covariates x_i and types q_j.

6 Survival and Choice Estimates

We present estimates from four different specifications. The first specification only relies on offer randomness and does not employ the scarcity instruments. The second specification, which is our preferred one, includes the number of past donors as the scarcity instrument. To assess robustness, we estimate a third specification with our past offers instrument and a fourth one using future donors. All specifications include a rich set of patient and donor covariates to capture medical history and match quality. These include all 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 characteristics used to determine patient priority on the transplant list.

6.1 Choice

Table 5 presents the marginal effects of select characteristics on the probability of acceptance, equation (3.3). The table reports the effects for a one standard deviation increase in a continuous characteristic or a unit change in an indicator.

Our results suggest that proxies for donor quality and match-specific benefits are positively correlated with acceptance. Patients are significantly more likely to accept kidney offers from younger donors and donors who died of head trauma and less likely to accept offers from donors with a history of hypertension. Patients are also significantly more likely to accept kidneys with which they have a perfect tissue-type match. Note that patients are also significantly more likely to accept kidneys which have higher unobservable quality, η_j, suggesting that decisions respond to information that is not perfectly captured by the observable organ characteristics included in the model. This information includes results from various medical tests and physical examination of the kidney.

The last two rows record the scarcity instruments' effects on the probability of 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 three instrumented specifications. These coefficients' robustness across the last three columns suggests that the choice between these two instruments is unlikely to be an important driver of our final results.

6.2 Survival

In Table 6, Panels A and B present estimates for survival without and with a transplant, respectively, as equations (3.1) and (3.2), showing the marginal half-life effects associated with select characteristics.[29]

Observable proxies for baseline patient health predict survival both with and without

[29] We avoid marginal expected life-years in order to limit extrapolation into extremely long survival durations. Our current dataset allows us to observe a patient for 16 years at most. This approach is consistent with the medical literature (e.g. Wolfe et al., 2008).

a transplant. A patient who is older, diabetic, or on dialysis at registration has a significantly shorter half-life without a transplant. These patient characteristics also have lower survival with a transplant, with effects that are slightly larger in magnitude. For example, a diabetic patient's half-life without transplant is lower than a non-diabetic patient by 1.36 years and their half-life with a transplant is lower by 2.98 years.

We also find that the proxies for donor quality, waiting time, and tissue-type similarity 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, which is consistent with the fact that an adverse immune reaction post-transplantation is less likely. These estimates are quite stable across our instrumented specifications.

A comparison of estimates in Tables 5 and 6 indicates 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. Likewise, the kidney unobservable characteristic, η_j, has a significant effect on choice but a small, insignificant effect on survival.

6.3 Selection on Unobservables

An advantage of our framework is that it can be used to measure the correlation between survival and choice induced by unobservable characteristics. The correlation on observable characteristics discussed above suggests this channel may also be important.

Table 7 presents these effects for the three specifications that use the scarcity instruments. The top panel shows how increased selectivity affects acceptance and survival. We measure these effects by raising $\nu_{i,D}$ in equation (3.3) by one standard deviation. The effects on survival are measured by computing the changes on $\nu_{i,0}$ and $\nu_{i,1}$ in-

duced by their estimated correlation with $\nu_{i,D}$. The bottom panel shows the correlation between unobserved match-specific determinants of choice and survival. We present these effects by reporting how one standard deviation higher $\varepsilon_{ij,D}$ impacts choices and post-transplant half-lives.

We find that selective patients typically survive longer without a transplant and benefit less from the typical transplant. Therefore, there is positive selection into treatment on the patient-specific component of survival benefits. A one standard deviation rise in $\nu_{i,D}$ selectivity lowers the probability of acceptance by 3.9 percentage points. This magnitude is of a similar order as the effect of a kidney from a donor with a history of hypertension. Comparing the specifications shows that our conclusion is not sensitive to instrument choice.

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. This suggests there is limited positive selection into specific treatments based on unobservable match-level benefits.

Table 5: Choice Estimates

	(1)	(2)	(3)	(4)
Patient Characteristics				
Diabetic	-0.003	-0.005	-0.005	-0.006
	(0.000)	(0.001)	(0.001)	(0.001)
CPRA	-0.008	-0.008	-0.008	-0.013
	(0.000)	(0.000)	(0.000)	(0.001)
On Dialysis at Registration	0.001	0.003	0.003	0.003
	(0.001)	(0.001)	(0.001)	(0.001)
Age at Registration	0.002	0.004	0.004	0.003
	(0.000)	(0.001)	(0.001)	(0.001)
Donor Characteristics				
Age < 18	0.140	0.153	0.154	0.152
	(0.008)	(0.008)	(0.008)	(0.009)
Age 18-35	0.079	0.098	0.098	0.134
	(0.008)	(0.008)	(0.008)	(0.010)
Age 50+	-0.060	-0.071	-0.069	-0.072
	(0.002)	(0.003)	(0.003)	(0.003)
Cause of Death - Head Trauma	0.057	0.065	0.064	0.068
	(0.006)	(0.007)	(0.007)	(0.007)
History of Hypertension	-0.025	-0.029	-0.028	-0.029
	(0.001)	(0.001)	(0.001)	(0.002)
Unobservable (η_j)	0.000	0.224	0.219	0.219
	(0.000)	(0.002)	(0.002)	(0.002)
Offer Characteristics				
Perfect Tissue Type Match	0.146	0.143	0.145	0.114
	(0.008)	(0.009)	(0.009)	(0.008)
Log Waiting Time (Years)	0.010	0.026	0.016	0.024
	(0.000)	(0.001)	(0.001)	(0.001)
Scarcity				
Log(1+#Past Donors)		-0.010		
		(0.001)		
Log(1+#Past Offers)			-0.020	
			(0.001)	
Log(1+#Future Donors)				-0.009
				(0.001)
Instruments	No Instruments	# Past Donors	# Past Offers	# Future Donors

Notes: This Table presents selected estimates of the marginal effect on the probability of acceptance of a one standard deviation increase in each continuous covariate and a unit increase in each binary covariate. Marginal effects are computed at the median value of observable covariates, integrating over the distribution of all unobservables. We generate 250000 draws and burn-in the first 50000 draws. We thin the chain by selecting every 10 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. See Appendix Table D.11 through D.13 for detailed estimates.

Table 6: Survival Estimates

	(1)	(2)	(3)	(4)
	Panel A: Survival without Transplant			
Patient Characteristics				
Diabetic	-1.380	-1.361	-1.361	-1.378
	(0.030)	(0.030)	(0.030)	(0.030)
CPRA	0.089	0.089	0.089	0.082
	(0.031)	(0.031)	(0.031)	(0.030)
On Dialysis at Registration	-1.019	-1.013	-1.013	-0.902
	(0.042)	(0.041)	(0.041)	(0.039)
Age at Registration	-1.070	-1.060	-1.060	-1.052
	(0.025)	(0.025)	(0.025)	(0.024)
	Panel B: Survival with Transplant			
Patient Characteristics				
Diabetic	-2.959	-2.980	-2.977	-3.212
	(0.099)	(0.113)	(0.111)	(0.110)
CPRA	-0.027	-0.026	-0.031	-0.064
	(0.098)	(0.098)	(0.097)	(0.096)
On Dialysis at Registration	-2.384	-2.395	-2.389	-2.075
	(0.118)	(0.125)	(0.123)	(0.115)
Age at Registration	-3.183	-3.192	-3.181	-3.416
	(0.118)	(0.126)	(0.124)	(0.121)
Donor Characteristics				
Age < 18	1.595	1.604	1.647	0.784
	(0.906)	(0.916)	(0.916)	(0.830)
Age 18-35	-0.267	-0.282	-0.249	-0.547
	(0.973)	(0.981)	(0.980)	(0.930)
Age 50+	3.383	3.381	3.296	0.635
	(2.243)	(2.252)	(2.241)	(1.864)
Cause of Death - Head Trauma	0.662	0.665	0.691	0.673
	(0.313)	(0.316)	(0.314)	(0.317)
History of Hypertension	-0.340	-0.342	-0.357	-0.410
	(0.122)	(0.124)	(0.123)	(0.125)
Unobservable (η_j)		0.107	0.181	0.194
		(0.183)	(0.177)	(0.176)
Offer Characteristics				
Perfect Tissue Type Match	2.272	2.269	2.322	1.946
	(0.944)	(0.959)	(0.954)	(0.896)
Log Waiting Time (Years)	-0.487	-0.543	-0.539	-0.646
	(0.062)	(0.168)	(0.161)	(0.155)
Instruments	No Instruments	# Past Donors	# Past Offers	# Future Donors

Notes: Select estimates of the marginal effect on half-life of a one standard deviation increase in each continuous covariate and a unit increase in each binary variable. Marginal effects are computed at the median value of observable covariates, integrating over the distribution of all unobservables. The specifications have the same patient, donor, and offer covariates as in Table 5 other than the scarcity instruments. Standard errors are in parentheses. See Appendix Table D.7 through D.10 for detailed estimates.

Table 7: Correlation Table

	(1)	(2)	(3)
Selectivity ($v_{i,D}$)			
Probability of Acceptance	-0.039	-0.039	-0.044
	(0.001)	(0.001)	(0.001)
Post-Transplant Survival	0.008	-0.025	-0.121
	(0.138)	(0.134)	(0.136)
Survival without a Transplant	0.330	0.323	0.229
	(0.060)	(0.059)	(0.059)
Match value ($\varepsilon_{ij,D}$)			
Probability of Acceptance	0.068	0.066	0.068
	(0.001)	(0.001)	(0.001)
Post-Transplant Survival	0.022	0.122	0.091
	(0.258)	(0.251)	(0.253)
Instruments	# Past Donors	# Past Offers	# Future Donors

Notes: Estimates of how one standard deviation increase in choice unobservables affects acceptance and survival probabilities. Survival durations are calculated using half-lives. Survival effects from changes in $\varepsilon_{ij,D}$ are computed using the expected change 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. Columns (1) through (3) use specifications corresponding to columns (2) through (4) in Tables 6 and 5.

7 Estimated LYFT

7.1 Calculating Life Years from Transplant

Our model measures survival benefits for every potential transplant. 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. This measure is widely used in the literature on organ transplantation (Wolfe et al., 2008) and is arguably the focal outcome for patients with kidney failure.

Specifically, for each pair (i, j), we define LYFT conditional on a set of covariates $I_{ij} = \{x_i, q_j, D_{ij}, \eta_j, \nu_{i,D}, \nu_{i,f}\}$ in our model as follows:

$$LYFT\left(I_{ij}\right) = M(Y_{ij}|I_{ij}, Y_{i0} \geq t_{ij}) - M(Y_{i0}|I_{ij}, Y_{i0} \geq t_{ij}), \tag{7.1}$$

where $M(Y|X)$ is the median of random variable Y conditional on X and t_{ij} is the time between patient i's registration and the arrival of kidney j. We then use a Gibbs' sampler to compute the expectation of $LYFT\left(I_{ij}\right)$ by drawing η_j, $\nu_{i,D}$, and $\nu_{i,f}$ from their conditional distributions given observables, decisions, and observed survival outcomes.[30] Therefore, this measure accounts for selection on unobservables induced by the mechanism.

In order to assess the role of selection due to choices and due to other unobservables, we also calculate expected $LYFT\left(I_{ij}\right)$ given only the observables x_i and q_j. In this case, we integrate $LYFT\left(I_{ij}\right)$ over the unconditional distributions of $\eta_j, \nu_{i,D}$, $\nu_{i,f}$ and D_{ij}.

7.2 Life Years from Transplant in the Mechanism

Table 8 presents the average estimated LYFT over all realized transplants. The first row presents the average LYFT accounting for patient- and kidney-specific unobservables

[30] The sampler provides us with simulated draws of $LYFT\left(I_{ij}\right)$ from its distribution. To do this, we generate a chain that fixes the parameters at the estimate $\hat{\theta}$. We generate 200,000 draws, burn-in the first half, and take one of every 1,000 draws.

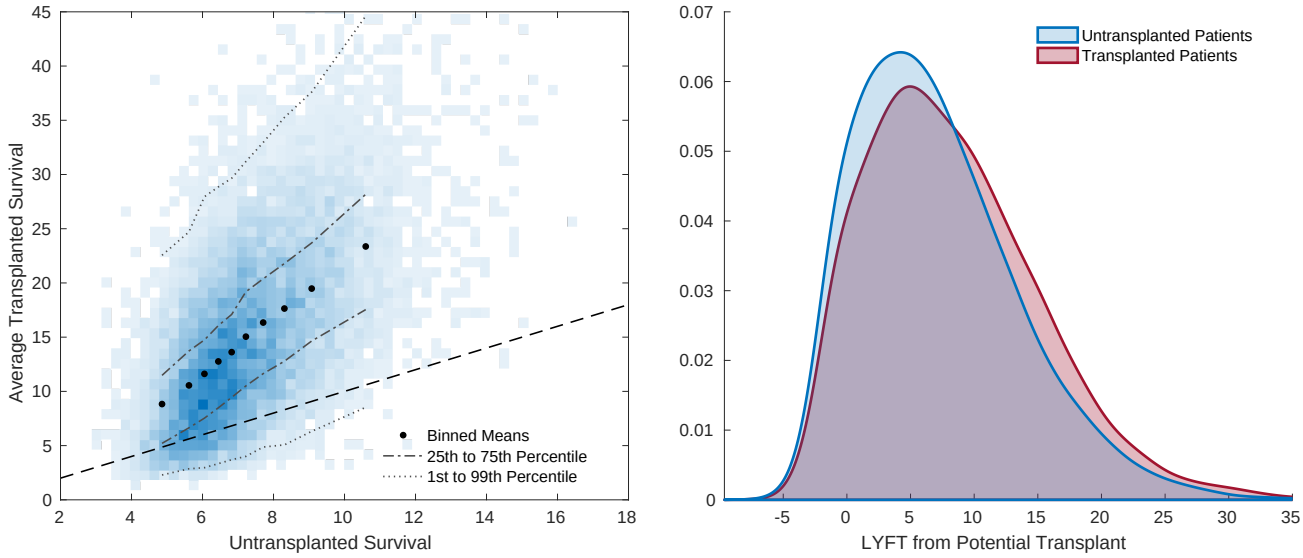
and the decision to accept. The second row presents the results conditioning only on the observables. The columns correspond to the specifications in Tables 5 and 6. The average LYFT from our preferred specification is 8.64 years (column 2). Ignoring selection on unobservables yields an an average LYFT of 7.94 years. This difference suggests there is positive selection on LYFT of patients into transplantation based on unobservables. Column (1) reports analogous estimates from a specification that does not use quasi-experimental variation from our scarcity instruments. The estimated LYFT is biased and about two-thirds of a year less than our preferred estimate. This suggests observational methods such as those in Wolfe et al. (1999) may underestimate gains from transplantation.

Table 8: Life-Years from Transplant

	(1)	(2)	(3)	(4)
Life Years from Transplant				
Accounting for Unobservables	7.98	8.64	8.63	8.63
Observables Only	7.95	7.94	7.83	7.71
Untransplanted Survival				
All Patients	6.89	6.95	6.95	6.86
Transplanted Patients	7.24	7.21	7.21	7.17
Post-Transplant Survival	15.22	15.84	15.84	15.80
Instruments	No Instruments	# Past Donors	# Past Offers	# Future Donors

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 6 Panel B and Table 5.

The second pair of rows of Table 8 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. This selection on untransplanted survival aggregates selection due to both choice and the mechanism.



(a) Transplanted and Untransplanted Survival

(b) LYFT by Transplant Status

Figure 3: Patient Selection

7.3 Selection and LYFT

The selection on LYFT and untransplanted survival reported in Table 8 above can take place along two margins: patients who are transplanted and kidneys to which they are matched. This subsection further investigates these sources.

7.3.1 Patient Selection

To understand the importance of patient selection, we present the relationship between (median) untransplanted survival and the average (median) transplanted survival from all potential donors for each patient. Figure 3(a) presents the joint density between these two quantities overlayed with a binscatter plot. Transplanted and untransplanted survival are strongly correlated with a slope of the conditional mean that is larger than one. Therefore, patients who are expected to live longer without a transplant also have the largest life-year gains from a transplant. This result implies strong complementarities between baseline health and transplantation.

When combined with the observation in Table 8 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. However, there are additional components of patient selection, from choice and from the priorities in the mechanism.

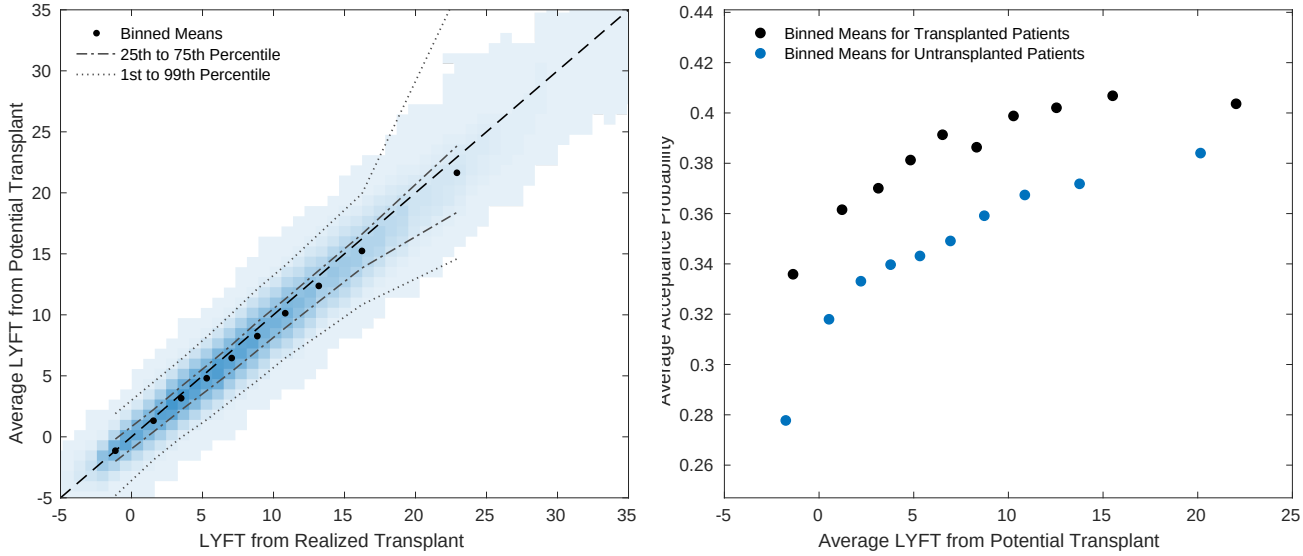
The overall selection on LYFT by observed transplant status is presented in Figure 3(b). This figure plots the distribution of predicted LYFT across all potential transplants. This distribution is shifted to the right for transplanted patients. The mean LYFT for this group is 1.2 years more than the untransplanted group.

Taken together, these results show that the mechanism selects patients with larger average LYFT and that some of this selection comes from transplanting patients who are relatively healthy at baseline. One way in which the mechanism achieves this is by making patients wait, which induces positive dynamic selection.

7.3.2 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 4(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.

The estimates of the choice and survival equations reported in Section 6 suggest that part of this advantageous matching comes from the correlation of patients' acceptance decisions with LYFT. Figure 4(b) summarizes this relationship in 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. This pattern is expected given that acceptance is necessary for a transplant to occur.



(a) Transplanted Survival from Potential and Realized Donors

(b) LYFT and Choice

Figure 4: Patient-Kidney Matching

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). A regression of acceptance probability on average LYFT, controlling for patient and donor fixed effects, underlines this point.[31] A one standard deviation increase in the match-specific component of LYFT raises the probability of acceptance by 0.59 percent.

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.

7.3.3 Patient Selection vs. Rematching

Figure 4(a) also provides insight into which of these two assignment margins dominates. The heterogeneity in survival across patients swamps the heterogeneity across donors

[31] Specifically, 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.

within a patient. In fact, a decomposition of the total variance in LYFT into patient-specific, donor-specific, and match-specific components (the latter is 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.[32]

These results suggest that the potential for increasing life-years by improving the match between patients and donors without changing which patients are transplanted (re-matching) is limited. Therefore, distributional constraints may 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, suggesting a potential trade-off between equity and efficiency.

8 Potential for Further Increasing LYFT

We now turn to evaluating the performance of the mechanism on LYFT and quantifying the importance of patient selection versus rematching. We do this by comparing the average LYFT achieved by the realized assignment to alternatives, ranging from a random assignment to one that maximizes LYFT. To ease computation throughout this exercise, we restrict the sample to the set of patients who registered in 2005.[33]

As we argued in Section 2, extending patients' lives is a prima facie objective of the medical profession. However, a planner's objective may depend on other factors, or the weights the planner places on life year gains may depend on how urgently sick a patient is. To address this issue, we will compare the types of patients who are transplanted under the alternative assignments. The results provide insight into the trade off between maximizing LYFT and distributional or ethical motivations for evaluating an assignment.

[32] The standard deviation in LYFT is 6.68 years, which is the Pythagorean sum of the three components.

[33] One of our exercises requires simulating the mechanism, a task not necessary during estimation. The year 2005 is the earliest year for which we were able to do so reliably.

We focus on results from our preferred specification. These results are not sensitive to choice of instrument and to varying the Box-Cox shape parameters of our specification. Detailed estimates from the alternative specifications are provided in Table D.14.

8.1 Comparison with Benchmark Assignments

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

- **Random assignment** is simulated by sorting patients in a random order and successively assigning patients to kidneys at random from the set of feasible kidneys. For a kidney to be feasible for a patient, it must be biologically compatible and 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, $\nu_{i,D}$, $\nu_{i,f}$, η_j, each patient's arrival and 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.[34]

Comparison to the random assignment allows us to measure the increase in LYFT achieved by the mechanism. Both selecting patients and advantageously matching kidneys to patients drove the difference. To decompose these sources, we evaluate an alternative that allocated kidneys randomly among patients who were actually transplanted:

[34]Specifically, we simulate the unobservables $\nu_{i,D}$, $\nu_{i,f}$, η_j from the distribution of these random variables conditional on the estimated parameters and the decisions observed in the data. We also draw a death date from the estimated untransplanted survival distribution. Call a simulated draw for each patient/donor pair $LYFT^s_{ij}$. 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 $\max_a \sum_{i,j} a_{ij} LYFT_{ij}$ 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 **random amongst transplanted** assignment is simulated by sorting *transplanted* patients in a random order and successively assigning only these 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 the LYFT gain over the random allocation achieved by the mechanism targeting patients from the LYFT gain 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. Kidneys are assigned in a random order, and priority is computed as in the mechanism.

Comparing the realized assignment to the optimal assignment reveals the maximum theoretical gain in LYFT that could be achieved over the existing 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 is computed by maximizing the total LYFT from all transplants under 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.[35]

The theoretical bounds based on optimal assignments use information about factors that induce selection, $\nu_{i,D}$, $\nu_{i,f}$, and η_j. However, the factors $\nu_{i,D}$ and $\nu_{i,f}$ may not be observed by the planner and may be hard to elicit in a mechanism. Similarly, η_j may

[35] As in calculations of average $LYFT\,(I_{ij})$, we simulate the unobservables from their conditional distributions given the data of these random variables to generate draws $LYFT^s_{ij}$. We then solve the problem in footnote 34 above with the additional constraint $a_{ij} = 0$ if i was not transplanted in the data.

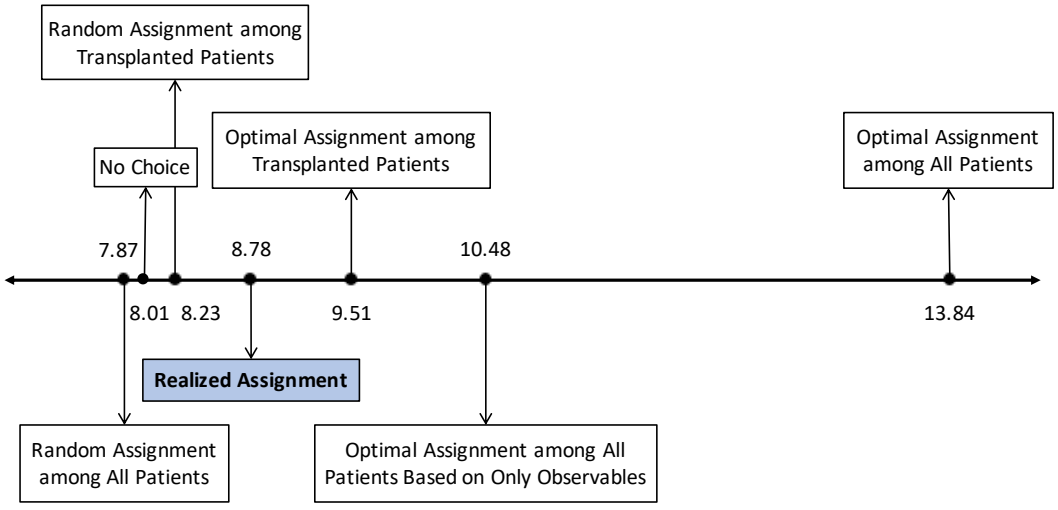


Figure 5: LYFT Under Counterfactual Assignments

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. For tractibility, we assume the planner has foresight on when patients arrive and depart and when kidneys arrive.[36],[37]The solution describes the highest possible LYFT that can be achieved by a planner who can dictate assignments based on this information.

Figure 5 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 8 above.

The realized assignment achieves about 0.92 years or 11.6% improvement in average LYFT over random assignment. Both selecting patients and matching patients to kidneys are important. If the transplanted patients were assigned a random kidney, then

[36]Relaxing foresight would require solving a dynamic assignment problem with uncertainty about the future.

[37]We modify the problem in footnote 34 by replacing $LYFT_{ij}^{s}$ with its expectation given x_i and q_j. The factors $\nu_{i,D}$, $\nu_{i,f}$, and η_j are drawn from their unconditional distributions.

the increase would only be 4.4 months. This quantity represents the rise relative to random assignments that is accounted for by patient selection. The remainder is due to patient-kidney matching.

Patient choice is a key contributor to the mechanism's gains in LYFT over random assignment. The no choice assignment results in similar LYFT as the random assignment. Thus, assignment to patients based on the existing priority rules without allowing patients to decline kidneys would achieve only 15.8% of the LYFT increase achieved by the realized assignment.[38]

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 13.84 years, about 5.1 years longer than the LYFT achieved in the realized assignment. Bias in estimates based on observational studies would miss the potential for these gains.[39] 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 4(a), most of the improvements in the optimal allocation come from changing the set of patients who are transplanted.

Finally, we find that a planner who can dictate assignments using the observable characteristics could achieve a significant fraction, but not all, of the potential increase. The average LYFT under the optimal assignment based on observables is 10.48 years. Although this is 3.4 years less than the theoretical maximum, it is about 1.7 years more than the average LYFT achieved by the mechanism. The observables in our model have been either used to determine priority or considered explicitly in proposed reforms.[40] Therefore, in principle, average LYFT could be substantially raised by targeting transplants using observed characteristics rather than choices.

[38] We obtain very similar results irrespective of whether we use the priorities in place before or after 2014. Thus, the change in the mechanism does not achieve the gains we identify.

[39] An assignment proposed 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.

[40] This claim is based on the minutes of the OPTN Kidney Transplantation Committee. Determining whether other health conditions can be used in the assignment system is beyond the scope of this paper.

Table 9: Characteristics of Transplanted Patients

	All Patients	Random Assignment		No Choice		Realized Assignment		Optimal Assignment	
		Transplanted Patients	LYFT	Transplanted Patients	LYFT	Transplanted Patients	LYFT	Transplanted Patients	LYFT
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
Age < 18	3.1%	3.2%	14.81	5.9%	13.70	5.4%	14.89	5.4%	18.83
Age 18 - 35	11.6%	12.3%	11.46	11.9%	11.61	13.0%	12.45	16.8%	15.35
Age 36 - 59	54.8%	55.8%	8.07	53.0%	8.17	54.7%	8.86	57.5%	13.70
Age >= 60	30.5%	28.8%	5.20	29.2%	5.11	26.9%	5.61	20.3%	11.68
Diabetic	41.4%	40.2%	5.94	37.7%	5.80	33.3%	6.51	31.6%	12.14
On Dialysis at Registration	83.0%	82.3%	7.63	82.0%	7.74	80.2%	8.50	80.7%	13.64
0 HLA Mismatches	-	0.0%	8.00	15.5%	8.18	12.5%	8.59	8.1%	15.55
0 DR Mismatches	-	4.2%	8.55	35.6%	8.11	21.9%	8.86	13.1%	14.87
HLA Mismatches	-	4.75	-	3.62	-	3.92	-	3.80	-
Untransplanted Survival	6.68	6.75	-	6.72	-	6.81	-	7.27	-

8.2 The Planner's Dilemma

An important conclusion from Figure 5 is that LYFT could be increased by up to 57.6%. However, achieving this would require changing the set of patients who are transplanted. We now argue that this change shifts the demographics and health conditions of transplanted patients, creating a potential barrier due to distributional considerations and the need to weigh patient urgency.

The LYFT increases, from random assignment to the mechanism and finally to the optimal solutions, require transplanting relatively healthy patients. Table 9 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 who are transplanted under the realized assignment are healthier than average – they are 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.

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. Under the no choice assignment, 15.5% of assignments are to zero-mismatch patients. Only 12.5% of assignments are to such patients under the realized assignment and 8.1% under the optimal assignment. Yet

choice also dramatically changes the selection of who is transplanted, shifting the age distribution towards younger patients and those with longer untransplanted survival. Both factors are correlated with higher LYFT. 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 over random assignment in part 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 3(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.

This stark trade-off represents a moral dilemma for several reasons. First, society may have a moral imperative to transplant sick patients who may soon die, even if doing so implies reducing total life years gained from transplantation. Second, there are concerns about discriminating based on patient characteristics. Our results suggest an optimal assignment should target transplants for younger patients, though proposed age-based priorities have conflicted with concerns about age discrimination when previous reforms were being considered.

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 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 a total of 5.1 years per kidney. Approximately 14.4% of these benefits could be attained if it were possible to dictate assignments based on observed patient and donor characteristics. 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. Specifically, we find that survival with and without a transplant is strongly correlated and that most of the heterogeneity in benefits from a transplant is across patients rather than match-specific. Therefore, the planner faces a dilemma between transplanting the sick and transplanting those for whom life will be extended the longest.

Through this work we open several important avenues for further research. First, our current approach evaluates benchmark assignments rather than the equilibria of alternative mechanisms that allow agents to express choice. It would be useful to combine recent approaches for analyzing equilibria of alternative mechanisms with a model of outcomes. Such a model would allow us to consider the selection induced via choices in a counterfactual environment. Second, we focus on an aggregate measure of LYFT that abstracts away from distributional considerations. Formalizing these constraints and incorporating them into the design problem is valuable. Solving these two challenges would allow a design approach that better speaks to the considerations central to policymaking. This trade-off between equity and efficiency, which is particularly central to designing mechanisms when outcomes are the target, deserves further research in other contexts as well.

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Appendix for "Choices and Outcomes in Assignment Mechanisms"

Nikhil Agarwal, Charles Hodgson, Paulo Somaini.

A Data Appendix

A.1 Obtainting 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 begining 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. 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 – 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.[41] 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 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 Janurary 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

[41] We use transplant data through December 31, 2015 to be consistent with the sample period during which we observe patient survival.

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 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

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

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 patiens that registered prior to 2000.

We consider a sample of offers made betwee January 1, 2000 and December 31, 2010 that could have resulted in transplants between our patient and donor samples. The

Table A.2: Sample Selection: Donors

	Number of Donors
Deceased donors offered to any kidney waitlist patients between 2000 and 2010	71,738
Exclude deceased donors offered through non-standard kidney allocations	67,993
Restrict to deceased donors offered to patients in the sample	61,453

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

	Number of Offers
Offers made between 2000 and 2010 from donors in the sample	14,888,539
Exclude non-responsive offers	14,239,214
Restrict to offers made to patients in the sample	8,444,106

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 with an observed survival duration needs to be computed. The 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 Decmeber 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. We expect that this censoring date does not induce selection bias that might confound our survival analysis.

Table A.4: Censor Reason

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

B Estimation Appendix

B.1 Gibbs' Sampler

Recall that our model is given by

$$\begin{aligned}
y_{i0} &= B\left(Y_{i0};\rho_0\right) = x_i\beta_x + \nu_{i,0} \\
y_{ij} &= B\left(Y_{ij};\rho_1\right) = \chi\left(x_i,q_j\right)\alpha_{x,q} + \alpha_\eta\eta_j + \nu_{i,1} + \varepsilon_{ij,1} \\
D_{ij} &= 1\left\{y_{ij,D} = \chi\left(x_i,q_j\right)\gamma_{x,q} + z_i\gamma_z + \eta_j + \nu_{i,D} + \varepsilon_{ij,D} > 0\right\},
\end{aligned}$$

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 η_j, ν_i and ε_{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 obserables 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 ν_i as

$$\begin{aligned}\nu_{i,D} &= f_{i,1} \\ \nu_{i,f} &= f_{i,2} \\ \nu_{i,0} &= \beta_{\nu 1} f_{i,1} + \beta_{\nu 2} f_{i,2} + \tilde{\varepsilon}_{i0}\end{aligned}$$

where $f_{i,1}$, $f_{i,2}$ and ε_{i0} are each independently distributed mean-zero normal random variables with variances σ_1^2, σ_2^2 and $\sigma_{\tilde{\varepsilon},0}^2$. This structure places no restrictions on the covariance matrix Σ_ν. Similarly, we write ε_{ij} as

$$\begin{aligned}\varepsilon_{ij,1} &= \alpha_\varepsilon f_{ij,3} + \tilde{\varepsilon}_{ij,1} \\ \varepsilon_{ij,D} &= f_{ij,3} + \tilde{\varepsilon}_{ij,D}\end{aligned}$$

where $f_{ij,3}$, $\tilde{\varepsilon}_{ij,1}$ and $\tilde{\varepsilon}_{ij,D}$ are independently distributed mean-zero normal random variables with variances σ_3^2, $\sigma_{\tilde{\varepsilon},1}^2$ and $\sigma_{\tilde{\varepsilon},D}^2$. We normalize the variances σ_3^2, and $\sigma_{\tilde{\varepsilon},D}^2$ to 1. Finally, we set

$$\eta_j = f_{j,4}$$

with variance σ_4^2. The main difference between $f_\cdot$and $\tilde{\epsilon}_\cdot$ 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 co-efficients in each equation, $\beta = (\beta_x, \beta_{\nu 1}, \beta_{\nu 2})$, $\alpha = (\alpha_{x,q}, \alpha_\eta, \alpha_{\nu 1}, \alpha_\varepsilon)$, $\gamma = (\gamma_{x,q}, \gamma_z)$, and the variances $\sigma_{\tilde{\varepsilon},0}^2 = V(\tilde{\varepsilon}_{i0})$, $\sigma_{\tilde{\varepsilon},1}^2 = V(\tilde{\varepsilon}_{ij,1})$ and $\sigma_l^2 = V(f_l)$ where $l \in \{1, 2, 4\}$ is the l-th factor.

For simplicity of notation, we will collect the coefficients in the vector θ and the standard deviations in the vector σ, with $\sigma_{\tilde{\varepsilon}}$ and σ_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 α, β and γ 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 σ^2_l 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, θ^0, σ^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+1}_{i0}, y^{k+1}_{ij} for censored observations and $y^{k+1}_{ij,D}$ for observed decisions given θ^k, σ^k and f^k from truncated normal distributions.

2. **Sample Coefficients:** Sample θ^{k+1} given y^{k+1}, f^k, the standard deviations σ^k and the prior distribution from a multi-variate normal distribution.

3. **Sample Variances:** Sample $\sigma^{2,k+1}_{\tilde{\varepsilon},0}$ and $\sigma^{2,k+1}_{\tilde{\varepsilon},1}$ given y^{k+1}, f^k, the parameters θ^{k+1} and the prior distribution from a inverse-Wishart distribution.

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

5. **Sample Factor Variances:** Sample $\sigma^{2,k+1}_l$ for $l \in \{1, 2, 4\}$ given f^{k+1} 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. We visually inspect the chains and 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 θ, f and σ:

(a) For each i, j pair such that D_{ij} is observed, the distribution of $y_{ij,D}$ conditional on γ, f and D_{ij} is a one-sided truncated with mean $\mathbb{E}\left[g_{ij,D}|\gamma, f_{ij}\right]$ 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 β and f is a one-sided truncated normal with mean $\mathbb{E}\left[y_{i0}|\beta, f_{i1}, f_{i2}\right]$ and standard deviation $\sigma_{\tilde{\varepsilon},0}$. The distribution of y_{i0} is truncated below at the censoring duration.

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

2. Posterior distributions of the co-efficients α, β and γ given y, f, σ and the priors. Since y_{i0}, y_{ij} and $y_{ij,D}$ are mutually independent conditional on f, the parameters α, β and γ 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 (see 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, σ and the priors. As argued 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. (see 2014).

4. Posterior distributions of f given y, θ and σ:

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

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

and σ_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 $f_{i,1} + \frac{1}{\alpha_{\nu 1}}\tilde{\varepsilon}_{ij,1} = \frac{1}{\alpha_{\nu 1}}[y_{ij} - (\chi(x_i, q_j)\alpha_{x,q} + \alpha_\eta\eta_j + +f_{i,2} + \alpha_\varepsilon f_{ij,3})]$ if $T_{ij} = 1$. These residuals have prior mean zero and variances $\sigma_1^2 + \frac{\sigma^2_{\tilde{\varepsilon},0}}{\beta^2_{\nu 1}}$, $\sigma_1^2 + \sigma^2_{\tilde{\varepsilon},1}$ and $\sigma_1^2 + \frac{\sigma^2_{\tilde{\varepsilon},1}}{\alpha^2_{\nu 1}}$ repectively. The mean is the precision-weighted average of the residuals conditioned on, and the variance is the inverse of the sum of σ_1^{-2} and the precisions of each residual.

(b) The distribution of $f_{i,2}$ is analogous, where we condition on the residual $\frac{1}{\beta_{\nu 2}}(y_{i0} - (x_i\beta_x + \beta_{\nu 1}f_{i,1}))$ and σ_2 throughout; and on the residual $y_{ij} - [\chi(x_i, q_j)\alpha_{x,q} + \alpha_\eta\eta_j + \alpha_{\nu 1}f_{i,1}]$ if $T_{ij} = 1$.

(c) The distribution of $f_{ij,3}$ is analogous, where we conditions on α_ε 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 $\frac{1}{\alpha_\varepsilon}(y_{ij} - [\chi(x_i, q_j)\alpha_{x,q} + \alpha_\eta\eta_j + f_{i,2}])$ if $T_{ij} = 1$. Observe that σ_3 is normalized to 1.

(d) The distribution of $f_{j,4}$ is analogous, where we condition on σ_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 $\frac{1}{\alpha_\eta}(y_{ij} - [\chi(x_i, q_j)\alpha_{x,q} + f_{i,2} + \alpha_\varepsilon f_{ij,3}])$ if $T_{ij} = 1$.

5. The variances σ_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 $q_n = \left(q_{j_{i,1}}, \ldots, q_{j_{i,n}}\right)$ and $q_{n-1} = \left(q_{j_{i,1}}, \ldots, q_{j_{i,n-1}}\right)$, which are truncated from q_{j_i} to the first n and $n-1$ offers respectively; and the vector $\tilde{D}_{i,n} = \left(D_{ij_{i,1}}, \ldots, D_{ij_{i,n}}\right)$. The definitions of $\tilde{J}_i$ and $T_{ij_{i,n}}$ imply that $T_{ij_{i,n}} = 1$

if and only if $\tilde{D}_{i,n-1} = 0, D_{ij_{i,n}} = 1$ and $Y_{0i} \geq t_{j_{i,n}}$. Assumption 2 implies that $P\left[T_{ij_{i,n}} = 1|q_{j_i}, z, Y_{0i} \geq t_{j_{i,n}}\right] = P\left[\tilde{D}_{i,n-1} = 0, D_{ij_{i,n}} = 1|q_{j_i}, z, Y_{0i} \geq t_{j_{i,n}}\right]$ is equal to the observed quantity $P\left[T_{ij_{i,n}} = 1|q_n, z, Y_{0i} \geq t_{j_{i,n}}\right]$, and is therefore identified. Similarly, if $P\left[T_{ij_{i,n}} = 1|q_{j_i}, z, Y_{0i} \geq t_{j_{i,n}}\right] > 0$, then Assumption 2 implies that

$$E\left[Y_{ij_{i,n}}|T_{ij_{i,n}} = 1, q_{j_i}, z\right] = E\left[Y_{ij_{i,n}}|\tilde{D}_{in-1} = 0, D_{ij_{i,n}} = 1, q_{j_i}, z, Y_{0i} \geq t_{j_{i,n}}\right]$$

is identified because it is equal to $E\left[Y_{ij_{i,n}}|T_{ij_{i,n}} = 1, q_n, z\right]$. Therefore, it remains to show that $E\left[Y_{i0}|T_{ij_{i,n}} = 1, q_{j_i}, z\right]$ is identified. First, re-write

$$\begin{aligned}
&E\left[Y_{i0}|T_{ij_{i,n}} = 1, q_{j_i}, z\right] \Pr\left[T_{ij_{i,n}} = 1|q_{j_i}, z, Y_{0i} \geq t_{j_{i,n}}\right] \\
=&E\left[Y_{i0}|T_{ij_{i,n}} = 1, q_n, z\right] \Pr\left[T_{ij_{i,n}} = 1|q_n, z, Y_{0i} \geq t_{j_{i,n}}\right] \\
=&E\left[Y_{i0}|\tilde{D}_{i,n-1} = 0, D_{ij_n} = 1, q_n, z, Y_{0i} \geq t_{j_{i,n}}\right] \Pr\left[\tilde{D}_{in-1} = 0, D_{ij_n} = 1|q_n, z, Y_{0i} \geq t_{j_{i,n}}\right] \\
=&E\left[Y_{i0}|\tilde{D}_{i,n-1} = 0, q_n, z, Y_{0i} \geq t_{j_{i,n}}\right] \Pr\left[\tilde{D}_{i,n-1} = 0|q_n, z, Y_{0i} \geq t_{j_{i,n}}\right] - E\left[Y_{i0}|\tilde{D}_{i,n} = 0, q_n, z, Y_{0i} \geq t_{j_{i,n}}\right] \mathrm{P} \\
=&E\left[Y_{i0}|\tilde{D}_{i,n-1} = 0, q_{n-1}, z, Y_{0i} \geq t_{j_{i,n}}\right] \Pr\left[\tilde{D}_{i,n-1} = 0|q_{n-1}, z, Y_{0i} \geq t_{j_{i,n}}\right] \\
&- E\left[Y_{i0}|\tilde{D}_{i,n} = 0, q_n, z, Y_{0i} \geq t_{j_{i,n}}\right] \Pr\left[\tilde{D}_{i,n} = 0|q_n, z, Y_{0i} \geq t_{j_{i,n}}\right]
\end{aligned}$$

where the last expression is observed. The first equality above follows from Assumption 2, the second equality is definitional, the third equality follows from set inclusion and the last from Assumption 2. Thefore, since $\Pr\left[T_{ij_{i,n}} = 1|q_{j_i}, z, Y_{0i} \geq t_{j_{i,n}}\right]$ is identified and strictly positive, $E\left[Y_{i0}|T_{ij_{i,n}} = 1, q_{j_i}, z\right]$ is identified.

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:

$$P\left(D_{i,j_{i,1}} = D_{i,j_{i,2}} = \ldots = D_{i,j_{i,k}} = 0|q_j^n, z_i\right) = \int_0^1 \varepsilon_D^k \mathrm{d}v\left(\varepsilon_D; q_j, z_i\right).$$

Observe that $a_k = \int_0^1 \varepsilon_D^k \mathrm{d}v\left(\varepsilon_D; q_j, z_i\right)$ is identified for $k \in \{1, \ldots, n\}$. Moreover, 3(i) and (ii) together imply that

$$a_0 = \int_0^1 1 \mathrm{d}v\left(\varepsilon_D; q_j, z_i\right) = 1.$$

Therefore, to complete the proof, we need to show that $v_{n-1}\left(\cdot; q_j, z_i\right)$ is determined by the values of $a_k = \int_0^1 \varepsilon_D^k \mathrm{d}v\left(\varepsilon_D; q_j, z_i\right)$ for $k \leq n$ where $v_{n-1}\left(\cdot; q_j, z_i\right)$ is the $(n-1)$-st order Fourier-Legendre approximation of $v\left(\cdot; q_j, z\right)$. In what follows, we will drop conditioning on z_i and q_j^n for simplicity of notiation.

To complete the proof, we write the co-efficients of $(n-1)$ −st Fourier-Legendre series of $v\left(\cdot\right)$ in terms of a_k. Let $\Gamma_m\left(x\right)$ be the m-th shifted Legendre Polynomial. Observe that each $\Gamma_m\left(\cdot\right)$ is given by

$$\Gamma_m\left(x\right) = \sum_{l=0}^{m} \gamma_{m,l} x^l,$$

with known co-efficients $\gamma_{m,l}$.[42]The $m-$th co-efficient in the (shifted) Fourier-Legendre series of $v\left(x\right)$ is given by

$$\begin{aligned} c_m &= \left(2m+1\right) \int_0^1 \Gamma_m\left(x\right) v\left(x\right) \mathrm{d}x \\ &= \left(2m+1\right) \left[\int_0^1 \Gamma_m\left(x\right) \mathrm{d}x - \int_0^1 \int_0^x \Gamma_m\left(y\right) \mathrm{d}y \mathrm{d}v\left(x\right) \right], \end{aligned}$$

where the second equality follows from integration by parts. Observe that $\int_0^1 \Gamma_m\left(x\right) \mathrm{d}x =$

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

$\int_0^1 \Gamma_m(x)\,\Gamma_0(x)\,\mathrm{d}x = 0$ for $m > 0$. Therefore, for $m > 0$,

$$\begin{aligned} c_m &= -(2m+1)\int_0^1\int_0^x \Gamma_m(y)\,\mathrm{d}y\mathrm{d}v(x) \\ &= -(2m+1)\int_0^1\int_0^x \sum_{l=0}^m \gamma_{m,l} y^l \mathrm{d}y\mathrm{d}v(x) \\ &= -(2m+1)\int_0^1 \sum_{l=0}^m \gamma_{m,l}\frac{1}{l+1}x^{l+1}\mathrm{d}v(x) \\ &= -(2m+1)\sum_{l=0}^m \gamma_{m,l}\frac{1}{l+1}\int_0^1 x^{l+1}\mathrm{d}v(x) \\ &= -(2m+1)\sum_{l=0}^m \gamma_{m,l}\frac{1}{l+1}a_{l+1}. \end{aligned} \tag{C.1}$$

And, finally, we have

$$\begin{aligned} c_0 &= \int_0^1 \Gamma_0(x)\,v(x)\,\mathrm{d}x \\ &= \int_0^1 v(x)\,\mathrm{d}x \\ &= v(1) - \int_0^1 x\mathrm{d}v(x), \end{aligned} \tag{C.2}$$

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 $\tilde{\Gamma}_m(y) = \Gamma_m\left(\frac{y+1}{2}\right)$.[43] The $(n-1)$-st order Fourier-Legendre approximation of $\tilde{v}(y) = v\left(\frac{y+1}{2}\right)$ is $\tilde{v}_{n-1}(y) = \sum_{k=0}^{n-1}\tilde{c}_m\tilde{\Gamma}_m(y)$ 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

[43] The unshifted Legendre-Polynomials on $[-1,1]$ satisfy the orthogonality relationship $\int_0^1 \tilde{\Gamma}_m(y)\,\tilde{\Gamma}_n(y)\,dy = \frac{2}{2n+1}\delta_{m,n}$ where $\delta_{m,n}$ is the Kronecker delta. The first few polynomials are $\tilde{\Gamma}_0(y) = 1$, $\tilde{\Gamma}_1(y) = y$, $\tilde{\Gamma}_2(x) = \frac{1}{2}\left(3x^2 - 1\right)$.

$\tilde{v}(\cdot)$ has a compact domain and image, we have that $\int_{-1}^{1} \tilde{v}(y)^2 \, dy$ 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, $\mathbb{P}(D_{ij} = 1 | \nu_{i,D} = v(\varepsilon_D; q_j, z_i); q_j, z_i)$ 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

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

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 sequential definition of continuity implies that 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

$$\begin{aligned}\sup_{x\in[a,b]}\left|\frac{f_n(x)}{g_n(x)}-\frac{f(x)}{g(x)}\right| \leq & \sup_{x\in[a,b]}|f_n(x)-f(x)|\sup_{x\in[a,b]}\left|\frac{1}{g_n(x)}-\frac{1}{g(x)}\right| \\ & + \sup_{x\in[a,b]}|f(x)|\sup_{x\in[a,b]}\left|\frac{1}{g_n(x)}-\frac{1}{g(x)}\right| \\ & + \sup_{x\in[a,b]}\left|\frac{1}{g(x)}\right|\sup_{x\in[a,b]}|f_n(x)-f(x)|.\end{aligned}$$

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 $\sup_{x\in[a,b]}|g(x)-g_n(x)|\leq\frac{k}{2}$. 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 $n>N$, $\sup_{x\in[a,b]}\left|\frac{1}{g_n(x)}\right| < \left(\frac{k}{2}\right)^{-1}$, 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 $\tilde{\varepsilon}=\min\left\{\frac{\varepsilon}{2}, x-a, b-x\right\}$ and $\delta_{\tilde{\varepsilon}}=\min\left\{f(x+\tilde{\varepsilon})-f(x), f(x)-f(x-\tilde{\varepsilon})\right\}$. Observe that $\tilde{\varepsilon}>0$ and $\delta_{\tilde{\varepsilon}}>0$ because and f is strictly increasing. Pick N such that for all $n>N$ $\sup_{x'\in[a,b]}|f_n(x')-f(x')|<\delta_{\tilde{\varepsilon}}$. 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 $x'<x-\tilde{\varepsilon}$, $f(x')+\delta_{\tilde{\varepsilon}}<f(x)$. Therefore, for all $n>N$ and $x'<x-\tilde{\varepsilon}$, $f_n(x')<f(x)$. Hence, $f_n^{-1}(f(x))\geq x-\tilde{\varepsilon}>x-\varepsilon$ for all $n>N$. Similarly, for all $x'>x+\tilde{\varepsilon}$, $f(x')>f(x)$. Therefore, for all $n>N$ and $x'>x+\tilde{\varepsilon}$,

$f_n(x') - \delta_{\tilde{\varepsilon}} > f(x)$. Hence, $f_n^{-1}(f(x)) \leq x + \tilde{\varepsilon} < x + \varepsilon$ for all $n > N$. □

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 $[a,b] \in (0,1)$, 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 $p_n(d\alpha;x)$ 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 $[0,1]$,[44]

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) $\int \Gamma_n(x)\Gamma_m(x)\,\mathrm{d}x = \delta_{mn}$ where δ_{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 a direct corollary

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

of 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

$$a_k = \int_0^1 \varepsilon_D^k \mathrm{d}v(\varepsilon_D;q_j,z_i)$$

are identified for all $k \leq n$. Let b_m be the (shifted) $m-$th Fourier-Legendre co-efficient of $v'(\cdot)$ defined on $[0,1]$

$$b_m = (2m+1)\int_0^1 \Gamma_m(x)\, v'(x)\,\mathrm{d}x$$

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

$$\begin{aligned} b_m &= (2m+1)\sum_{l=0}^{m} \gamma_{m,l} \int_0^1 x^l v'(x)\,\mathrm{d}x \\ &= (2m+1)\sum_{l=0}^{m} \gamma_{m,l} a_l, \end{aligned}$$

are identified. The second equality follows from the definition of a_l. □

C.4 Proof of Theorem 1

Identification of $E(Y_{i0}|\nu)$. Define $y_0(\nu) = E(Y_{i0}|\nu)$. For a given ν, 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. As in the main text, we are also conditioning and dropping x_i from the 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_j^k is in the support of the distribution of offer types for all $k \leq n$, then the joint distribution of $Y_{i0}, \left\{T_{ij_{i,1}}, \ldots, T_{ij_{i,k}}\right\}$ conditional on q_j^k differs for some $k \leq n$ under models s and $\tilde{s}$.

Consider a value of $\bar{\nu} \in (0,1)$ such that $y_0(\bar{\nu}) \neq \tilde{y}_0(\bar{\nu})$ and $\bar{\nu} = v(\bar{x}; q_j)$ for some $\bar{x} \in (0,1)$. Lemmas 2 and 5 imply that if either $v(\bar{x}; q_j) \neq \tilde{v}(\bar{x}; q_j)$ or $v'(\bar{x}; q_j) \neq \tilde{v}'(\bar{x}; q_j)$ for some $\bar{x} \in (0,1)$, then there exists N such that for all $n > N$ the joint distribution of $\left\{T_{ij_{i,1}}, \ldots, T_{ij_{i,n}}\right\}$ conditional on q_j^k for some $k \leq n$ differs for models s and $\tilde{s}$. Therefore, it is sufficient to focus on the case when $v(\bar{x}; q_j) = \tilde{v}(\bar{x}; q_j)$ and $v'(\bar{x}; q_j) = \tilde{v}'(\bar{x}; q_j)$. Moreover, since $\bar{x} \in (0,1)$, we have that $v'(\bar{x}; q_j) > 0$ (Assumption 4(i)) implying that it is sufficient to show that that if $y_0(v(\bar{x}; q_j)) v'(\bar{x}; q_j) \neq \tilde{y}_0(v(\bar{x}; q_j)) v'(\bar{x}; q_j)$, then the joint distribution of $Y_{i0}, \left\{T_{ij_{i,1}}, \ldots, T_{ij_{i,k}}\right\}$ conditional on q_j^k differs for some $k \leq n$ under models s and $\tilde{s}$.

We prove this by showing that if $y_0(v(\bar{x}; q_j)) v'(\bar{x}; q_j) \neq \tilde{y}_0(v(\bar{x}; q_j)) v'(\bar{x}; q_j)$, 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$Y_{i0}, \left\{T_{ij_{i,1}}, \ldots, T_{ij_{i,k}}\right\}$ conditional on q_j^k 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

$$\begin{aligned} E\left(Y_{i0} \times 1\{T_i = 0\} | q_j^k\right) &= \int_0^1 E\left(Y_{i0} | \nu_D = v(x; q_j)\right) x^k \mathrm{d}v(x; q_j) \\ &= \int_0^1 x^k y_0(v(x; q_j)) v'(x; q_j) \mathrm{d}x. \end{aligned}$$

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 $\left\{E\left(Y_{i0} \times 1\{T_i = 0\}\,|q_j^k\right)\right\}_{k=1}^{n}$. Similarly, let $\tilde{u}_n(x;q_j)$ be the (shifted) Fourier-Legendre series associated with model $\tilde{s}$ 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(i) and (ii) imply continuity of $u(x,q_j)$ 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

$$\begin{aligned}\int_0^1 y_0(v(x;q_j))^2\,v'(x;q_j)^2\,\mathrm{d}x &= \int_0^1 E(Y_{i0}|v(x;q_j))^2\,v'(x;q_j)^2\,\mathrm{d}x \\ &\leq \sup_x |v'(x;q_j)| \int_0^1 E(Y_{i0}|v(x;q_j))^2\,v'(x;q_j)\,\mathrm{d}x \\ &= \sup_x |v'(x;q_j)| \int_0^1 E(Y_{i0}|\nu)^2\,\mathrm{d}\nu,\end{aligned}$$

where the second equality follows from a change of variables. Observe that Assumption 4(i) holds that $\sup_x |v'(x;q_j)|$ is finite. The term $\int_0^1 E(Y_{i0}|\nu)^2\,\mathrm{d}\nu$ is finite since

$$\begin{aligned}\int_0^1 E(Y_{i0}|\nu)^2\,\mathrm{d}\nu &= V(E[Y_{i0}|\nu]) + E(E(Y_{i0}|v))^2 \\ &= V(E[Y_{i0}|\nu]) + E(Y_{i0})^2 \\ &\leq V(Y_{i0}) + E(Y_{i0})^2,\end{aligned}$$

where the inequality follows from the law of total variance. 4(ii) implies that the right hand side is bounded. Therefore, $\bar{u}_n(x,q_j)$ 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

$$\left| y_0(v(\bar{x};q_j))\,v'(\bar{x};q_j) - \frac{1}{n}\sum_{k=0}^{n-1} u_k(\bar{x};q_j) \right| < \frac{\delta}{2}$$

and

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

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

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

Because each $u_n\left(\bar{x};q_j\right)$ and $\tilde{u}_n\left(\bar{x};q_j\right)$ is determined by the conditional expectations $\left\{E\left(Y_{i0}\times 1\left\{T_i=0\right\}|q_j^k\right)\right\}_{k=1}^{n}$, we have shows that the joint distribution of $Y_{i0},\left\{T_{ij_{i,1}},\ldots,T_{ij_{i,k}}\right\}$ conditional on q_j^k differs for some $k\leq n$ under models s and $\tilde{s}$.

Identification of $E\left(Y_{ij}|\nu_D,\varepsilon_{ij,D}\geq\varepsilon_D,q_j\right)$. Define $y_1\left(\nu_D,\varepsilon_D;q_j\right)=E\left(Y_{ij}|\nu_D,\varepsilon_{ij,D}\geq\varepsilon_D,q_j\right)$. Consider a pair of models s and $\tilde{s}$. As argued above, we can restrict to pairs such that $v\left(x;q_j,z\right)=\tilde{v}\left(x;q_j,z\right)$ 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 $\nu=v\left(\bar{x};q_j,\bar{z}\right)$. We will show that if $y_1\left(v\left(\bar{x};q_j,\bar{z}\right),\bar{x};q_j\right)v'\left(\bar{x};q_j,\bar{z}\right)\neq\tilde{y}_1\left(v\left(\bar{x};q_j,\bar{z}\right),\bar{x};q_j\right)v'\left(\bar{x};q_j,\bar{z}\right)$, 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 $Y_{ij},\left\{T_{ij_{i,1}},\ldots,T_{ij_{i,k}}\right\}$ conditional on q_j^k and $\bar{z}$ differs for some $k\leq n$ under models s and $\tilde{s}$.

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

$$\begin{aligned}
&E\left(Y_{ij_k}\times 1\left\{T_{ij_k}=1\right\}|q_j^k,\bar{z}\right)\\
=&\int_0^1 E\left(Y_{ij_n}|\nu_D=v\left(x;q_j,\bar{z}\right),\varepsilon_{ij,D}\geq x,q_j\right)x^{k-1}\left(1-x\right)\mathrm{d}v\left(x;q_j,\bar{z}\right)\\
=&\int_0^1 x^{k-1}\left(1-x\right)y_1\left(v\left(x;q_j,\bar{z}\right),x;q_j\right)v'\left(x;q_j,\bar{z}\right)\mathrm{d}x.
\end{aligned}$$

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

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

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 the argument above because

$$\begin{aligned}
&\int_0^1 (1-x)^2 y_1(v(x; q_j, \bar{z}), x; q_j)^2 v'(x; q_j, \bar{z})^2 \, \mathrm{d}x \\
&\leq \sup_x |v'(x; q_j)| \int_0^1 ((1-x) E(Y_{ij} | v(x; q_j, \bar{z}), \varepsilon_{ij,D} \geq x))^2 v'(x; q_j, \bar{z}) \, \mathrm{d}x \\
&= \sup_x |v'(x; q_j)| \int_0^1 \int_0^1 E(Y_{ij} | v(x; q_j, \bar{z}), \varepsilon)^2 1\{\varepsilon \geq x\} v'(x; q_j, \bar{z}) \, \mathrm{d}x d\varepsilon \\
&= \sup_x |v'(x; q_j)| \int_0^1 \int_0^1 E(Y_{ij} | \nu, \varepsilon)^2 1\{v(\varepsilon; q_j, \bar{z}) \geq \nu\} \, \mathrm{d}\nu d\varepsilon \\
&\leq \sup_x |v'(x; q_j)| \int_0^1 \int_0^1 E(Y_{ij} | \nu, \varepsilon)^2 \, \mathrm{d}\nu d\varepsilon,
\end{aligned}$$

where the second equality follows from a change of variables and the fact that $v(x; q_j, z)$ is strictly monotonic in x. As above, Assumption 4(i) implies that $\sup_x |v'(x; q_j, z)|$ is finite and

$$\begin{aligned}
\int_0^1 E(Y_{ij} | \nu, \varepsilon)^2 \, \mathrm{d}\nu &= V(E[Y_{ij} | \nu, \varepsilon]) + E(E(Y_{ij} | v, \varepsilon))^2 \\
&= V(E[Y_{ij} | \nu, \varepsilon]) + E(Y_{ij})^2 \\
&\leq V(Y_{ij}) + E(Y_{ij})^2,
\end{aligned}$$

Therefore, if $\delta = |(1-\bar{x}) y_1(v(\bar{x}; q_j, \bar{z}), \bar{x}; q_j) v'(\bar{x}; q_j, \bar{z}) - (1-\bar{x}) \tilde{y}_1(v(\bar{x}; q_j, \bar{z}), \bar{x}; q_j) v'(\bar{x}; q_j, \bar{z})|$, then, as argued above, Lemma 4 implies that there exists n such that

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

Because each $u_n(\bar{x}; q_j, \bar{z})$ and $\tilde{u}_n(\bar{x}; q_j, \bar{z})$ is determined by the conditional expectations $\left\{ E\left(Y_{ij_k} \times 1\{T_{ij_k} = 1\} | q_j^k, \bar{z} \right) \right\}_{k=1}^n$, we have shown that thejoint distribution of

$Y_{ij}, \left\{T_{ij_{i,1}}, \ldots, T_{ij_{i,k}}\right\}$ conditional on q_j^k and $\bar{z}$ differs for some $k \leq n$ under models s and $\tilde{s}$.

C.5 Dynamic Selection

Let $h_t(v)$ be the cdf of ν_D conditional on surviving until t. It is given by

$$h_t(v) = \int^v \frac{\Pr(Y_{i0} \geq t | \nu_D)}{\Pr(Y_{i0} \geq t)} d\nu_D.$$

Observe that the types ν_D of surviving patients will be uniformly distributed only if the event $Y_{i0} \geq t$ is independent of ν_D.

Lemma 6. *Suppose that the hypothesis of Theorem 1 hold.* The function $h_t(v) = \int^v \frac{\Pr(Y_{i0} \geq t|\nu_D)}{\Pr(Y_{i0} \geq t)} d\nu_D$ is identified for every $t \geq 0$.

Proof. Let q_j be a donor-type that arrives at the same time 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 $t \geq 0$, $\mathbb{P}(Y_{i0} \geq t|\nu_D) = \mathbb{E}[1\{Y_{i0} \geq t\} | \nu_D]$ is identified. Thus, $\Pr(Y_{i0} \geq t)$ and the function $h_t(v)$ is identified. □

Define the cdf of the probability that a patient which survives until t_j rejects a kidney of type q_j as $v_j(\varepsilon_D; q_j, z_i) = h_{t_j} \circ v(\varepsilon_D; q_j, z_i)$. Our next result shows that $v_j(\varepsilon_D; q_j, z_i)$ and $v(\varepsilon_D; q_j, z_i)$ are identified under additional smoothness assumptions on the distribution of $Y_{i0}|\nu_D$. Lemma 2 discussed the identification of the function $v(\varepsilon_D; q_j, z_i)$ for donor types q_j with $t_j = 0$. We extend this result to the case when $t_j > 0$.

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 ε_D such that*

$$\mathbb{P}(Y_{i0} \geq t_j | \nu_D = v(\varepsilon_D; q_j, z_i)) > 0.$$

Proof. Fix ε_D is such that $\mathbb{P}(Y_{i0} \geq t_j|\nu_D = v(\varepsilon_D; q_j, z_i)) > 0$. Note that $h_{t_j}(\cdot)$ is differentiable because $\mathbb{P}(Y_{i0} \geq t_j|\nu_D) > 0$ 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 $\mathbb{P}(Y_{i0} \geq t_j | \nu_D)$ is also positive in a neighborhood around ν_D and that h_{t_j} is strictly increasing at that point. Thus, $v(\varepsilon_D; q_j, z_i) = h_{t_j}^{-1} \circ v_j(\varepsilon_D; q_j, z_i)$ is identified because the terms on the right hand side are identified. □

We are now ready to prove our main identification result for donor types that arrive at any $t_j > 0$:

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, $\mathbb{P}(D_{ij} = 1 | \nu_{i,D} = v(\varepsilon_D; q_j, z_i), Y_{i0} \geq t_j)$ is identified.

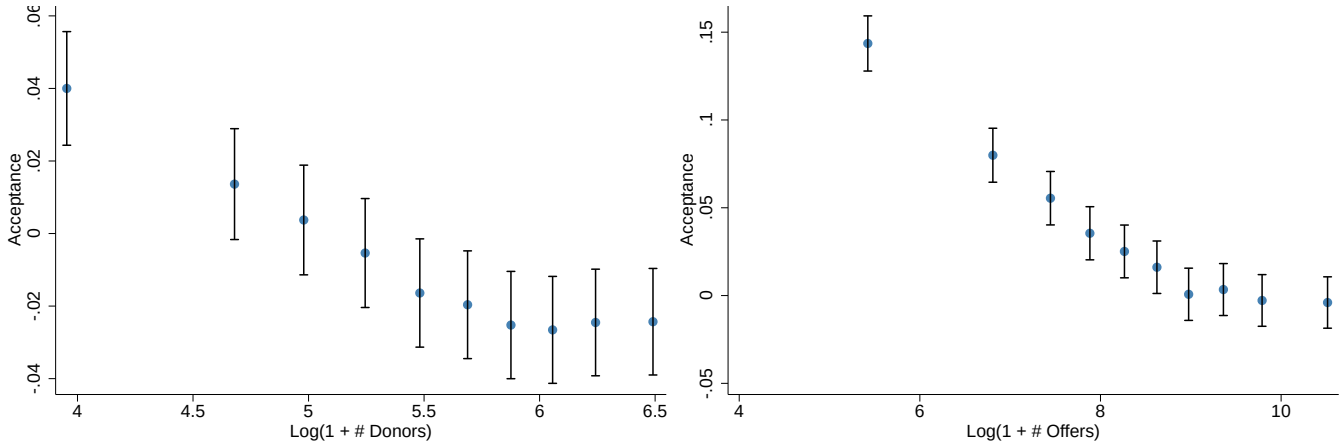
By the chain rule $\frac{\partial}{\partial \varepsilon} v_j(\varepsilon_D; q_j, z_i) = \frac{\partial}{\partial \nu} h_{t_j}(\nu_D) \frac{\partial}{\partial \varepsilon} v(\varepsilon_D; q_j, z_i)$. Note that $\frac{\partial}{\partial \varepsilon} v(\varepsilon_D; q_j, z_i)$ is continuous, bounded and strictly positive. Also, $\frac{\partial}{\partial \nu} h_{t_j}(\nu_D) = \frac{\Pr(Y_{i0} \geq t_j | \nu_D)}{\Pr(Y_{i0} \geq t_j)}$ is continous, bounded and strictly positive because the denominator is strictly positive by the assumption that there exists ν_D with $\mathbb{P}(Y_{i0} \geq t_j | \nu_D, x_i) > 0$ 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 neiborhood of ε_D. Arguments identical to those used for proving Theorem 1 imply that $\mathbb{E}\left(Y_{ij} | h_{t_j}(\nu_{i,D}) = h_{t_j}(\nu_D), \varepsilon_{ij,D} \geq \varepsilon_D, Y_{i0} \geq t_j\right)$ is identified. Because $\mathbb{P}(Y_{i0} \geq t_j | \nu_D, x_i) > 0$, we have that $h_{t_j}(\nu_D)$ is strictly increasing at ν_D, the event $h_{t_j}(\nu_{i,D}) = h_{t_j}(\nu_D)$ is equivalent to $\nu_{i,D} = \nu_D$. □

D Additional Figures and Tables

Table D.5: Top 10 offers: Balance

	Age	Diabetes	Female	Weight	Height
	(1)	(2)	(3)	(4)	(5)
log(1 + # Top 10 Offers in 2 Years)					
KDPI <= 50%	-0.0479	0.00134	-0.00158	-0.269*	0.0253
	(0.0772)	(0.00302)	(0.00277)	(0.108)	(0.0732)
KDPI > 50% or Missing	-0.0233	-0.00427	0.000269	0.104	0.0137
	(0.0683)	(0.00294)	(0.00276)	(0.101)	(0.0819)
DSA FE, Year 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	0.499	0.267	0.787	0.037	0.828
Number of Observations	128949	127414	128949	127363	126619
R-Squared	0.026	0.022	0.074	0.038	0.034

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 indictors 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.



(a) Donors (b) Offers

Figure D.1: Scarcity Instrument: First Stage

Notes: Figures are plotted using binsreg (Cattaneo et al., 2019) with the same specification as Columns (5) and (6) in Table 4. Dependent variable is acceptance of an offer. Independent variables include DSA fixed effect, offer year fixed effect, number of years waited at offer fixed effect, blood type fixed effect, patient characteristics, donor characteristics, and match characteristics.

Table D.6: Scarcity Instruments: Balance

	Age	Diabetes	Female	Weight	Height
	(1)	(2)	(3)	(4)	(5)
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**	-0.1000	-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 indictors 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: Survival Estimates

	(1)	(2)	(3)	(4)
Panel A: Survival without Transplant				
Constant	0.286	0.282	0.282	0.240
	(0.052)	(0.052)	(0.052)	(0.050)
Patient Characteristics				
Diabetic	-0.054	-0.054	-0.054	-0.055
	(0.001)	(0.001)	(0.001)	(0.001)
CPRA	0.018	0.018	0.018	0.016
	(0.006)	(0.006)	(0.006)	(0.006)
CPRA >= 0.8	0.000	-0.001	-0.001	-0.004
	(0.008)	(0.008)	(0.008)	(0.008)
CPRA = 0	0.003	0.003	0.003	0.002
	(0.002)	(0.002)	(0.002)	(0.002)
CPRA - 0.8 if CPRA >= 0.8	-0.073	-0.067	-0.067	-0.035
	(0.053)	(0.053)	(0.053)	(0.051)
Intial CPRA Missing	-0.129	-0.129	-0.129	-0.120
	(0.005)	(0.005)	(0.005)	(0.004)
Prior Transplant	-0.041	-0.041	-0.041	-0.041
	(0.005)	(0.005)	(0.005)	(0.005)
On Dialysis at Registration	-0.039	-0.039	-0.039	-0.035
	(0.002)	(0.002)	(0.002)	(0.002)
Blood Type A	0.006	0.006	0.006	0.005
	(0.003)	(0.003)	(0.003)	(0.004)
Blood Type O	0.019	0.020	0.020	0.018
	(0.003)	(0.003)	(0.003)	(0.003)
Blood Type B	0.027	0.029	0.029	0.027
	(0.004)	(0.004)	(0.004)	(0.004)
Age at Registration	0.002	0.002	0.002	0.002
	(0.001)	(0.001)	(0.001)	(0.001)
Age - 18 if Age >= 18	-0.003	-0.003	-0.003	-0.004
	(0.001)	(0.001)	(0.001)	(0.001)
Age - 35 if Age >= 35	-0.002	-0.002	-0.002	-0.002
	(0.000)	(0.000)	(0.000)	(0.000)
Age - 50 if Age >= 50	0.000	0.000	0.000	0.000
	(0.000)	(0.000)	(0.000)	(0.000)
Age - 65 if Age >= 65	-0.001	-0.001	-0.001	0.000
	(0.000)	(0.000)	(0.000)	(0.000)
BMI at Departure	0.004	0.004	0.004	0.005
	(0.003)	(0.003)	(0.003)	(0.003)
BMI - 18.5 if BMI >= 18.5	0.001	0.001	0.001	0.000
	(0.003)	(0.003)	(0.003)	(0.003)
BMI - 25 if BMI >= 25	-0.004	-0.004	-0.004	-0.004
	(0.001)	(0.001)	(0.001)	(0.001)
BMI - 30 if BMI >= 30	-0.001	-0.001	-0.001	-0.001
	(0.001)	(0.001)	(0.001)	(0.001)
BMI Missing	0.050	0.050	0.050	0.076
	(0.053)	(0.053)	(0.053)	(0.052)
Serum Albumin	0.043	0.042	0.042	0.042
	(0.002)	(0.002)	(0.002)	(0.002)
Serum Albumin - 3.7 if >= 3.7	0.011	0.011	0.011	0.012
	(0.004)	(0.004)	(0.004)	(0.005)
Serum Albumin - 4.4 if >= 4.4	-0.060	-0.059	-0.059	-0.060
	(0.005)	(0.005)	(0.005)	(0.005)
Serum Albumin Missing	0.148	0.147	0.147	0.148
	(0.008)	(0.008)	(0.008)	(0.008)
Log Dialysis Time at Registration (Years)	-0.015	-0.015	-0.015	-0.015
	(0.000)	(0.000)	(0.000)	(0.001)
Log Dialysis Time at Registration x 1{> 5 years}	0.000	0.000	0.000	0.006
	(0.005)	(0.005)	(0.005)	(0.005)
Unobservable Characteristics				
Selectivity		0.014	0.014	0.009
		(0.003)	(0.002)	(0.002)
Survival		0.014	0.018	0.067
		(0.039)	(0.037)	(0.032)

Table D.8: Survival Estimates (Continued)

	Panel B: Survival with Transplant			
Constant	0.646	1.778	1.758	0.628
	(0.089)	(0.237)	(0.237)	(0.092)
Patient Characteristics				
Diabetic	-0.097	-0.233	-0.234	-0.100
	(0.003)	(0.010)	(0.010)	(0.004)
CPRA	-0.010	-0.011	-0.013	-0.011
	(0.017)	(0.041)	(0.041)	(0.017)
CPRA >= 0.8	0.004	-0.011	-0.012	0.004
	(0.021)	(0.051)	(0.051)	(0.022)
CPRA = 0	0.003	0.007	0.007	0.003
	(0.005)	(0.012)	(0.012)	(0.005)
CPRA - 0.8 if CPRA >= 0.8	-0.072	-0.235	-0.242	-0.075
	(0.144)	(0.347)	(0.347)	(0.144)
Intial CPRA Missing	-0.008	-0.033	-0.031	-0.011
	(0.009)	(0.028)	(0.027)	(0.010)
Prior Transplant	-0.013	-0.044	-0.046	-0.016
	(0.015)	(0.040)	(0.040)	(0.016)
On Dialysis at Registration	-0.063	-0.185	-0.185	-0.064
	(0.004)	(0.010)	(0.010)	(0.004)
Blood Type A	-0.007	-0.019	-0.020	-0.008
	(0.007)	(0.017)	(0.017)	(0.007)
Blood Type O	0.001	0.003	0.000	0.000
	(0.007)	(0.018)	(0.018)	(0.007)
Blood Type B	-0.008	-0.005	-0.006	-0.009
	(0.008)	(0.019)	(0.019)	(0.008)
Age at Registration	-0.007	-0.013	-0.013	-0.006
	(0.002)	(0.004)	(0.004)	(0.002)
Age - 18 if Age >= 18	0.006	0.013	0.013	0.005
	(0.002)	(0.005)	(0.005)	(0.002)
Age - 35 if Age >= 35	-0.006	-0.017	-0.017	-0.007
	(0.001)	(0.003)	(0.003)	(0.001)
Age - 50 if Age >= 50	-0.002	-0.004	-0.004	-0.002
	(0.001)	(0.002)	(0.002)	(0.001)
Age - 65 if Age >= 65	-0.001	-0.002	-0.002	-0.001
	(0.001)	(0.002)	(0.002)	(0.001)
BMI at Departure	0.010	0.011	0.010	0.010
	(0.005)	(0.013)	(0.013)	(0.005)
BMI - 18.5 if BMI >= 18.5	-0.007	-0.001	-0.001	-0.007
	(0.005)	(0.014)	(0.014)	(0.005)
BMI - 25 if BMI >= 25	-0.003	-0.012	-0.012	-0.003
	(0.002)	(0.004)	(0.004)	(0.002)
BMI - 30 if BMI >= 30	-0.003	-0.004	-0.004	-0.003
	(0.001)	(0.003)	(0.003)	(0.001)
BMI Missing	0.205	0.271	0.268	0.205
	(0.091)	(0.235)	(0.235)	(0.090)
Serum Albumin	0.026	0.061	0.062	0.028
	(0.006)	(0.015)	(0.015)	(0.006)
Serum Albumin - 3.7 if >= 3.7	0.028	0.079	0.079	0.029
	(0.011)	(0.027)	(0.027)	(0.011)
Serum Albumin - 4.4 if >= 4.4	-0.056	-0.138	-0.138	-0.059
	(0.010)	(0.027)	(0.027)	(0.010)
Serum Albumin Missing	0.105	0.252	0.253	0.112
	(0.020)	(0.053)	(0.053)	(0.021)
Log Dialysis Time at Registration (Years)	-0.016	-0.042	-0.042	-0.016
	(0.001)	(0.004)	(0.004)	(0.001)
Log Dialysis Time at Registration x 1{> 5 years}	-0.070	-0.171	-0.171	-0.069
	(0.012)	(0.029)	(0.029)	(0.012)

Table D.9: Survival Estimates (Continued)

Donor Characteristics				
Age < 18	0.021	0.116	0.119	0.023
	(0.024)	(0.065)	(0.065)	(0.025)
Age 18-35	-0.017	-0.022	-0.020	-0.016
	(0.029)	(0.074)	(0.074)	(0.029)
Age 50+	0.020	0.235	0.230	0.017
	(0.055)	(0.152)	(0.152)	(0.055)
Cause of Death - Anoxia	0.003	-0.001	0.000	0.004
	(0.009)	(0.023)	(0.023)	(0.010)
Cause of Death - Stroke	0.002	0.000	0.001	0.003
	(0.009)	(0.023)	(0.023)	(0.009)
Cause of Death - CNS	0.010	0.041	0.039	0.009
	(0.019)	(0.050)	(0.050)	(0.019)
Cause of Death - Head Trauma	0.018	0.049	0.051	0.020
	(0.009)	(0.023)	(0.023)	(0.009)
Creatinine 0.5-1.0	-0.005	-0.002	0.000	-0.004
	(0.007)	(0.018)	(0.018)	(0.007)
Creatinine 1.0-1.5	-0.013	-0.023	-0.021	-0.012
	(0.007)	(0.018)	(0.018)	(0.007)
Creatinine >= 1.5	-0.012	-0.027	-0.028	-0.013
	(0.008)	(0.020)	(0.020)	(0.008)
Expanded Criteria Donor (ECD)	-0.019	-0.047	-0.049	-0.020
	(0.006)	(0.015)	(0.015)	(0.006)
Donation After Cardiac Death (DCD)	-0.003	-0.007	-0.009	-0.004
	(0.005)	(0.013)	(0.013)	(0.005)
Male	0.001	0.001	0.001	0.001
	(0.003)	(0.007)	(0.007)	(0.003)
History of Hypertension	-0.012	-0.026	-0.027	-0.013
	(0.004)	(0.009)	(0.009)	(0.004)
Offer Characteristics				
Perfect Tissue Type Match	0.053	0.162	0.167	0.054
	(0.025)	(0.067)	(0.067)	(0.026)
2 A Mismatches	-0.002	0.017	0.017	-0.002
	(0.016)	(0.039)	(0.039)	(0.016)
2 B Mismatches	0.001	-0.018	-0.019	0.001
	(0.017)	(0.042)	(0.043)	(0.017)
2 DR Mismatches	0.000	-0.006	-0.006	0.000
	(0.016)	(0.040)	(0.040)	(0.017)
ABO Compatible	-0.008	-0.011	-0.013	-0.009
	(0.012)	(0.030)	(0.030)	(0.012)
Regional Offer	-0.007	-0.014	-0.012	-0.007
	(0.014)	(0.036)	(0.036)	(0.014)
Local Offer	0.035	0.073	0.080	0.038
	(0.021)	(0.057)	(0.057)	(0.022)
Log Waiting Time (Years)	-0.003	-0.008	-0.008	-0.003
	(0.002)	(0.005)	(0.005)	(0.002)
Log Waiting Time x 1{Over 1 Year}	-0.003	-0.026	-0.025	-0.005
	(0.008)	(0.021)	(0.021)	(0.008)
Log Waiting Time x 1{Over 2 Years}	-0.021	-0.055	-0.055	-0.027
	(0.011)	(0.032)	(0.032)	(0.013)
Perfect Tissue Type Match x Prior Transplant	-0.003	0.012	0.012	-0.003
	(0.032)	(0.082)	(0.082)	(0.032)
Perfect Tissue Type Match x Diabetic Patient	-0.008	-0.011	-0.010	-0.007
	(0.008)	(0.019)	(0.019)	(0.008)
Perfect Tissue Type Match x Patient Age	0.000	-0.002	-0.002	0.000
	(0.000)	(0.001)	(0.001)	(0.000)
Perfect Tissue Type Match x CPRA	-0.016	0.011	0.012	-0.015
	(0.027)	(0.068)	(0.068)	(0.027)
Perfect Tissue Type Match x 1{CPRA > 80%}	-0.015	-0.039	-0.039	-0.015

Table D.10: Survival Estimates (Continued)

	(0.031)	(0.078)	(0.078)	(0.031)
Perfect Tissue Type Match x ECD Donor	0.019	0.044	0.042	0.018
	(0.011)	(0.030)	(0.030)	(0.011)
Perfect Tissue Type Match x DCD Donor	-0.014	-0.044	-0.045	-0.014
	(0.020)	(0.050)	(0.050)	(0.020)
Perfect Tissue Type Match x Local Offer	-0.027	-0.055	-0.056	-0.028
	(0.020)	(0.052)	(0.052)	(0.020)
Perfect Tissue Type Match x ABO Compatible	0.023	0.046	0.047	0.024
	(0.015)	(0.038)	(0.038)	(0.015)
Local Offer x 1{2 A Mismatches}	-0.001	-0.022	-0.022	-0.002
	(0.016)	(0.040)	(0.040)	(0.016)
Local Offer x 1{2 B Mismatches}	0.000	0.022	0.022	0.000
	(0.017)	(0.043)	(0.043)	(0.017)
Local Offer x 1{2 DR Mismatches}	-0.010	-0.011	-0.011	-0.010
	(0.017)	(0.040)	(0.040)	(0.017)
Local Offer x 1{Donor Age < 18}	-0.036	-0.097	-0.099	-0.037
	(0.017)	(0.046)	(0.046)	(0.018)
Local Offer x 1{Donor Age 18-35}	-0.017	-0.032	-0.033	-0.018
	(0.013)	(0.035)	(0.035)	(0.013)
Local Offer x 1{Donor Age 50+}	-0.010	-0.022	-0.022	-0.010
	(0.014)	(0.035)	(0.035)	(0.014)
Patient Age x 1{Donor Age < 18}	0.000	0.000	0.000	0.000
	(0.000)	(0.001)	(0.001)	(0.000)
Patient Age x 1{Donor Age 18-35}	0.001	0.003	0.003	0.002
	(0.001)	(0.002)	(0.002)	(0.001)
Patient Age x 1{Donor Age 50+}	-0.001	-0.008	-0.008	-0.001
	(0.002)	(0.004)	(0.004)	(0.002)
Patient Age - 35 if Age >= 35 x 1{Donor Age 18-35}	-0.001	-0.002	-0.002	-0.001
	(0.001)	(0.002)	(0.002)	(0.001)
Patient Age - 35 if Age >= 35 x 1{Donor Age 50+}	0.002	0.008	0.008	0.001
	(0.002)	(0.005)	(0.005)	(0.002)
Unobserved Covariates				
Selectivity		0.001	-0.002	-0.004
		(0.012)	(0.012)	(0.004)
Survival		1.000	1.000	1.000
		(0.000)	(0.000)	(0.000)
Match Value		0.002	0.009	0.002
		(0.019)	(0.019)	(0.008)
Donor Quality	0.002	0.003	0.005	0.002
	(0.001)	(0.005)	(0.005)	(0.002)
Instruments	No Instruments	# Past Donors	# Past Offers	# Future Donors

Notes: Estimates of the survival equations are presented. The sample includes 7938854 offers made between 2000 and 2009 to patients in the sample. The chain length is 250000, which includes a burn-in of 50000 draws. We thin the chain by taking every 10 draws. All columns control for dummies for DSA fixed effect, blood type fixed effect, and registration year fixed effect. Future donors (offers) is defined as the number of donors (offers) in the next 4 quarters (see Table 4 for detailed definition). Standard errors are in parenthese.

Table D.11: Choice Estimates

	(1)	(2)	(3)	(4)
Constant	-4.080	-4.895	-4.071	-5.266
	(0.175)	(0.356)	(0.356)	(0.381)
Patient Characteristics				
Diabetic	-0.043	-0.090	-0.093	-0.111
	(0.006)	(0.011)	(0.011)	(0.013)
CPRA	-0.721	-1.072	-1.089	-1.504
	(0.032)	(0.064)	(0.065)	(0.067)
CPRA >= 0.8	-0.198	-0.181	-0.132	-0.162
	(0.043)	(0.086)	(0.087)	(0.087)
CPRA = 0	0.053	0.110	0.110	0.110
	(0.009)	(0.018)	(0.019)	(0.019)
CPRA - 0.8 if CPRA >= 0.8	-1.322	-3.142	-3.258	-2.839
	(0.287)	(0.563)	(0.576)	(0.569)
Intial CPRA Missing	0.632	1.178	1.177	1.214
	(0.024)	(0.047)	(0.048)	(0.045)
Prior Transplant	-0.319	-0.468	-0.478	-0.578
	(0.028)	(0.052)	(0.053)	(0.057)
On Dialysis at Registration	0.012	0.050	0.054	0.060
	(0.007)	(0.013)	(0.013)	(0.014)
Blood Type A	-0.307	-0.425	0.053	-0.477
	(0.036)	(0.063)	(0.065)	(0.063)
Blood Type O	-0.553	-0.827	-0.183	-0.871
	(0.037)	(0.066)	(0.068)	(0.065)
Blood Type B	-0.149	-0.652	-0.328	-0.713
	(0.040)	(0.067)	(0.067)	(0.070)
Age at Registration	0.057	0.084	0.083	0.082
	(0.003)	(0.005)	(0.005)	(0.005)
Age - 18 if Age >= 18	-0.055	-0.085	-0.083	-0.082
	(0.003)	(0.006)	(0.006)	(0.006)
Age - 35 if Age >= 35	0.002	0.008	0.009	0.008
	(0.002)	(0.004)	(0.004)	(0.004)
Age - 50 if Age >= 50	-0.003	-0.005	-0.005	-0.006
	(0.001)	(0.002)	(0.002)	(0.003)
Age - 65 if Age >= 65	-0.002	-0.002	-0.003	-0.003
	(0.002)	(0.004)	(0.004)	(0.004)
BMI at Departure	-0.011	-0.010	-0.009	0.019
	(0.009)	(0.018)	(0.018)	(0.019)
BMI - 18.5 if BMI >= 18.5	0.007	0.000	-0.002	-0.033
	(0.010)	(0.020)	(0.020)	(0.021)
BMI - 25 if BMI >= 25	-0.007	-0.011	-0.011	-0.008
	(0.003)	(0.007)	(0.007)	(0.007)
BMI - 30 if BMI >= 30	-0.006	-0.008	-0.008	-0.012
	(0.003)	(0.005)	(0.005)	(0.006)
BMI Missing	-0.593	-0.888	-0.860	-0.283
	(0.164)	(0.328)	(0.333)	(0.354)
Serum Albumin	0.001	-0.003	-0.002	0.018
	(0.011)	(0.022)	(0.022)	(0.024)
Serum Albumin - 3.7 if >= 3.7	0.067	0.102	0.102	0.115
	(0.020)	(0.040)	(0.041)	(0.042)
Serum Albumin - 4.4 if >= 4.4	-0.089	-0.149	-0.151	-0.159
	(0.021)	(0.041)	(0.042)	(0.041)
Serum Albumin Missing	0.082	0.138	0.146	0.216
	(0.039)	(0.078)	(0.078)	(0.084)
Log Dialysis Time at Registration (Years)	0.008	0.025	0.027	0.025
	(0.002)	(0.004)	(0.004)	(0.005)
Log Dialysis Time at Registration x 1{> 5 years}	-0.008	0.050	0.039	0.025
	(0.022)	(0.044)	(0.044)	(0.050)
Donor Characteristics				
Age < 18	1.169	1.930	1.957	1.907
	(0.052)	(0.081)	(0.081)	(0.085)
Age 18-35	0.752	1.359	1.380	1.728
	(0.059)	(0.095)	(0.094)	(0.098)
Age 50+	-1.176	-1.987	-1.990	-2.010
	(0.085)	(0.132)	(0.132)	(0.143)

Table D.12: Choice Estimates (Continued)

Cause of Death - Anoxia	0.097	0.156	0.153	0.203
	(0.054)	(0.089)	(0.086)	(0.089)
Cause of Death - Stroke	0.409	0.678	0.677	0.736
	(0.053)	(0.088)	(0.086)	(0.090)
Cause of Death - CNS	-0.589	-0.894	-0.876	-0.988
	(0.100)	(0.158)	(0.154)	(0.175)
Cause of Death - Head Trauma	0.573	0.969	0.967	0.998
	(0.052)	(0.089)	(0.085)	(0.089)
Creatinine 0.5-1.0	0.757	1.281	1.273	1.270
	(0.038)	(0.068)	(0.067)	(0.058)
Creatinine 1.0-1.5	0.523	0.898	0.890	0.912
	(0.040)	(0.072)	(0.071)	(0.060)
Creatinine >= 1.5	-0.577	-0.927	-0.926	-0.839
	(0.041)	(0.076)	(0.074)	(0.064)
Expanded Criteria Donor (ECD)	-0.733	-1.203	-1.174	-1.247
	(0.034)	(0.053)	(0.054)	(0.052)
Donation After Cardiac Death (DCD)	-0.438	-0.734	-0.737	-0.765
	(0.028)	(0.049)	(0.048)	(0.055)
Male	0.108	0.184	0.184	0.175
	(0.016)	(0.027)	(0.027)	(0.027)
History of Hypertension	-0.351	-0.591	-0.588	-0.598
	(0.023)	(0.029)	(0.029)	(0.036)
Offer Characteristics				
Perfect Tissue Type Match	1.208	1.828	1.876	1.520
	(0.053)	(0.091)	(0.092)	(0.091)
2 A Mismatches	-0.027	-0.039	-0.040	-0.110
	(0.013)	(0.021)	(0.020)	(0.025)
2 B Mismatches	0.020	0.031	0.031	-0.022
	(0.013)	(0.023)	(0.023)	(0.026)
2 DR Mismatches	-0.086	-0.143	-0.147	-0.152
	(0.012)	(0.018)	(0.018)	(0.024)
ABO Compatible	-0.492	-0.820	-0.854	-0.866
	(0.039)	(0.066)	(0.067)	(0.066)
Regional Offer	0.477	0.812	0.817	0.134
	(0.017)	(0.025)	(0.025)	(0.030)
Local Offer	1.698	2.783	2.798	2.508
	(0.030)	(0.046)	(0.045)	(0.050)
Log Waiting Time (Years)	-0.022	0.034	0.025	0.053
	(0.004)	(0.007)	(0.007)	(0.007)
Log Waiting Time x 1{Over 1 Year}	0.161	0.441	0.322	0.419
	(0.016)	(0.027)	(0.027)	(0.028)
Log Waiting Time x 1{Over 2 Years}	0.174	0.484	0.235	0.352
	(0.023)	(0.042)	(0.043)	(0.043)
Perfect Tissue Type Match x Prior Transplant	-0.262	-0.639	-0.658	-0.598
	(0.076)	(0.148)	(0.148)	(0.147)
Perfect Tissue Type Match x Diabetic Patient	0.009	-0.037	-0.034	-0.016
	(0.023)	(0.042)	(0.042)	(0.042)
Perfect Tissue Type Match x Patient Age	0.002	0.005	0.005	0.005
	(0.001)	(0.002)	(0.002)	(0.001)
Perfect Tissue Type Match x CPRA	0.059	0.084	0.087	0.365
	(0.073)	(0.137)	(0.137)	(0.143)
Perfect Tissue Type Match x 1{CPRA > 80%}	0.086	0.057	0.031	0.123
	(0.077)	(0.147)	(0.147)	(0.156)
Perfect Tissue Type Match x ECD Donor	-0.765	-1.229	-1.266	-1.033
	(0.043)	(0.069)	(0.069)	(0.071)
Perfect Tissue Type Match x DCD Donor	-0.556	-0.957	-0.963	-1.057
	(0.076)	(0.128)	(0.127)	(0.129)
Perfect Tissue Type Match x Local Offer	0.147	0.379	0.360	0.699
	(0.039)	(0.061)	(0.060)	(0.063)
Perfect Tissue Type Match x ABO Compatible	0.482	0.850	0.875	0.908
	(0.048)	(0.085)	(0.085)	(0.082)
Local Offer x 1{2 A Mismatches}	-0.007	-0.016	-0.015	0.042
	(0.014)	(0.023)	(0.023)	(0.027)

Table D.13: Choice Estimates (Continued)

Local Offer x 1{2 B Mismatches}	-0.114	-0.181	-0.183	-0.177
	(0.015)	(0.025)	(0.026)	(0.028)
Local Offer x 1{2 DR Mismatches}	-0.149	-0.207	-0.211	-0.243
	(0.014)	(0.020)	(0.020)	(0.026)
Local Offer x 1{Donor Age < 18}	-0.868	-1.416	-1.426	-1.284
	(0.033)	(0.054)	(0.055)	(0.058)
Local Offer x 1{Donor Age 18-35}	-0.414	-0.706	-0.703	-0.960
	(0.031)	(0.050)	(0.049)	(0.052)
Local Offer x 1{Donor Age 50+}	0.100	0.134	0.134	0.024
	(0.028)	(0.043)	(0.043)	(0.050)
Patient Age x 1{Donor Age < 18}	-0.013	-0.021	-0.021	-0.023
	(0.001)	(0.001)	(0.001)	(0.001)
Patient Age x 1{Donor Age 18-35}	0.003	0.003	0.003	0.000
	(0.001)	(0.002)	(0.002)	(0.002)
Patient Age x 1{Donor Age 50+}	0.015	0.028	0.028	0.032
	(0.002)	(0.004)	(0.004)	(0.004)
Patient Age - 35 if Age >= 35 x 1{Donor Age 18-35}	-0.011	-0.016	-0.016	-0.014
	(0.002)	(0.003)	(0.003)	(0.003)
Patient Age - 35 if Age >= 35 x 1{Donor Age 50+}	-0.006	-0.014	-0.014	-0.016
	(0.003)	(0.004)	(0.004)	(0.004)
Scarcity				
Log(1+#Past Donors)		-0.289		
		(0.018)		
Log(1+#Past Offers)			-0.297	
			(0.008)	
Log(1+#Future Donors)				-0.268
				(0.019)
Instruments	No Instruments	# Past Donors	# Past Offers	# Future Donors

Notes: Estimates of the choice equation are presented. The sample includes 7938854 offers made between 2000 and 2009 to patients in the sample. The chain length is 250000, which includes a burn-in of 50000 draws. We thin the chain by taking every 10 draws. All columns control for dummies for DSA fixed effect, blood type fixed effect, and registration year fixed effect. Future donors (offers) is defined as the number of donors (offers) in the next 4 quarters (see Table 4 for detailed definition). Standard errors are in parenthese.

Table D.14: Robustness

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
Realized Assignment	8.13	8.78	8.72	8.73	8.88	10.07	8.66	8.08	8.93
Random Assignment among									
All Patients	7.27	7.87	7.71	7.56	7.91	8.92	7.74	7.16	7.81
Transplanted Patients	7.60	8.23	8.09	8.12	8.31	9.38	8.09	7.65	8.43
No Choice	7.99	8.01	7.90	7.83	8.00	8.85	7.86	7.05	7.56
Optimal Assignment among									
Transplanted Patients	10.45	10.48	10.35	10.37	10.47	11.91	10.25	9.30	10.39
All Patients Based on Only Observables	8.74	9.51	9.42	9.29	9.59	10.95	9.39	8.71	9.69
All Patients	10.48	13.84	13.68	13.58	13.83	15.97	13.74	12.16	12.90
Box-Cox ρ									
Survival without Transplant	0.5	0.5	0.5	0.5	0.5	0.5	0.4	0.5	0.6
Survival with Transplant	0.6	0.6	0.6	0.6	0.6	0.5	0.6	0.7	0.6
Instruments									
# Past Donors		x			x	x	x	x	x
# Past Offers			x						
# Future Donors				x					
Donor Unobservables	x	x	x	x		x	x	x	x
Other Unobservables		x	x	x	x	x	x	x	x

Notes: Robustness of the results presented in Figure 5. The baseline specification is presented in column (2). The remaining specifications vary the instruments, the presence of η_j, or the Box-Cox shape parameters as indicated in the table.