

Who Benefits from Higher Treatment Rates? Selection and Survival on the U.S. Kidney Transplant Waitlist

Stephen KASTORYANO*

January 19, 2026

Abstract

The paper examines how expanding treatment availability in waitlist-based allocation systems can affect who receives treatment, when they receive it, and with what consequences. It focuses on the U.S. kidney transplant system, where biological variation in ABO blood type induces quasi-random differences in transplant rates. I develop a strategy to identify selection on treatment timing within a dynamic causal framework, combining information on pre- and post-transplant survival. The main empirical finding is that higher transplant rates disproportionately skew kidney allocation toward healthier individuals rather than uniformly increasing transplants across all health types. Surprisingly, when considering overall survival, the returns to increasing transplant rates appear remarkably small: a 1 percent increase over five years raises survival by just 0.04 percent at most, seven years after entry.

Keywords: Selection effects, Dynamic treatment effects, Kidney transplant, Survival models.

JEL Codes: I11, I18, C22, C41

*University of Reading, IZA, e-mail: s.p.kastoryano@reading.ac.uk.

Thanks to Jad Beyhum, Petyo Bonev, Jaap Abbring, Giovanni Mastrobuoni, Sang Yoon (Tim) Lee, and Christoph Breunig for valuable comments and help. The data reported here have been supplied by the Hennepin Healthcare Research Institute (HHRI) as the contractor for the Scientific Registry of Transplant Recipients (SRTR). 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 SRTR or the U.S. Government. The research protocol was approved by the IRB of the University of Reading.

1 Introduction

Medical treatments and social programs – education, welfare, labour market, housing – are often allocated through waitlists. Causal evaluations of these programs and treatments strive to ensure selection is held fixed among treated and control groups. However, after changing a policy, selection into treatment may not remain invariant. Faced with a new information setting imposed by regulators, agents may re-optimize their behaviour in response to changing incentives (Rosenzweig and Wolpin, 2000). These pre-treatment changes can alter the composition of individuals receiving treatment (Chassang et al., 2012). When this is the case, understanding the selection processes may be as important as evaluating the effect of the program or treatment itself.

This issue is particularly salient in the U.S. kidney transplant system. As of 2022, approximately 140,000 candidates were on the national waitlist, with around 40,000 added each year but only 25,000 removed after a successful transplantation (Lentine et al., 2023). Despite chronic scarcity, 25% of viable kidneys were discarded in 2021,¹ a share far exceeding that of other high-income countries.² These patterns suggest a complex allocation process shaped not only by biology and medical need, but also by prevailing policies and institutional discretion. This raises an important question: when organ availability increases, who benefits? Does an increase in supply improve access uniformly, or does it systematically favor certain types of candidates?

This paper studies how variation in treatment availability affects selection and outcomes in waitlist-based allocation systems. Focusing on the U.S. kidney transplant setting, it exploits quasi-random variation in blood types (AB,A,B,O) as a plausibly exogenous shifter of the transplant rates. Leveraging this variation, I develop a simple strategy to identify selection on treatment timing within a dynamic causal framework. The approach combines information on pre-treatment survival with survival outcomes and observed characteristics of candidates treated immediately after waitlist entry. The results show that higher transplant rates skew access toward healthier individuals rather than expanding access uniformly. Yet, despite the sizeable difference in transplant rates and disparities in treatment timing, overall survival gains from increased transplant rates are surprisingly small. The findings underscore how institutional incentives and selection dynamics can mediate the effects of policy expansions in constrained allocation systems.

The structure of the dynamic causal framework, first proposed in Abbring and Van den Berg (2005), is common to many waitlist allocation systems: individuals enter a state at time 0 and are randomized to a *regime* at baseline. The regime is an intention-to-treat

¹The U.S. discard rates have been increasing since 2015.

²France had a kidney discard rate of 9.1% from 2004-2014, the United Kingdom has a rate ranging from 10% to 12% and Eurotransplant, a consortium of eight countries including Germany, reported a rate of about 8% (Aubert et al., 2019; Bernstein, 2023). The main reason for this difference is that the U.S. system is unusually transparent compared to these other regions, leaving more room for discretion in which organs to accept in the hands of transplant centers and candidates on waitlists (Rasmussen et al., 2018).

variable which dictates a stochastic propensity for the timing of a future *treatment* among agents. Treatment is then realized at various points depending on regime assignment and individual survival. In the context of the U.S. kidney transplant waitlist, individuals enter the waitlist when their kidney function falls below a clinical threshold. As a regime variable, the paper uses a previously underexploited source of biological randomness: individual ABO blood type. Conditional on health and socio-demographic covariates, including race, blood type is assumed to be as-good-as-random with respect to survival. Due to compatibility constraints, however, blood type directly affects the likelihood and timing of transplant offers – satisfying the necessary conditions for this causal setup. In this application, the outcome is survival and ‘treatment’ is defined as receiving a transplant, which, due to dynamic selection, is only observed for candidates who receive one before dying.

The first part of the empirical analysis yields four main findings: (1) as expected, AB candidates who are compatible with any blood type are, on average, 2.5 times more likely to receive a transplant within the first five years of entering the waitlist than O candidates, who almost always need to receive a transplant from an O donor, (2) AB candidates exhibit lower *pre-transplant survival* than O candidates, (3) this pre-transplant effect remains when controlling for health variables determining the prioritization of a candidate on the waitlist and is also robust to controlling for additional baseline health, socio-economic and location variables,³ (4) transplant rates and pre-transplant survival exhibit a one-to-one inverse relationship across blood types.

Drawing on the identification strategy, the discussion outlines two mechanisms that could generate these effects. One possibility is that a higher transplant rate skews allocation toward healthier individuals earlier in their waitlist spells – a selection effect tied to the timing of treatment.⁴ In some cases, like ours, this mechanism can also be inferred if candidates who receive a transplant shortly after entering the waitlist exhibit different survival rates across regimes. An alternative mechanism is that higher transplant rates induce behavioral responses, such as moral hazard, that reduce survival. I discuss how to interpret the pre-transplant effect under each mechanism. Under our preferred interpretation, consistent with the literature, the effect arises because transplant centers tend to prioritize higher-health candidates. In that case, and assuming moral hazard responses to the regime are limited, the results show that a 1% increase in the transplant rate leads to a 0.1% increase in transplants to individuals who would have survived even without receiving a kidney during the first five years on the waitlist.

³These pre-transplant differences are stable across time horizons, specifications, and subsamples – including by race, donor type, and before/after a 2014 reform to the allocation mechanism. We also offer additional evidence looking at survival and transplant patterns for all blood-type groups (AB, A, B, O) to rule out biological or socio-economic explanations for pre-transplant differences in survival.

⁴The discussion of selection must be qualified here. Given a sufficiently large set of pertinent observed variables, selection can always be explained. In this paper our discussion of selection focuses on how much selection remains after controlling for the official variables determining prioritization on the waitlist, and after conditioning on a large set of observed demographic, health, and location variables in the dataset.

The second contribution is to offer additional insights into the selection mechanism. Using double/debiased machine learning methods the analysis shows: (1) AB candidates transplanted in the first three months on the waitlist have a 5 percentage point higher survival probability after three years than their O-type counterparts transplanted at the same period, (2) the functional health of AB transplant recipients, an observable proxy for higher-health, is significantly higher than for O-types during the early waitlist period, but not thereafter, (3) as duration on the waitlist elapses, younger and healthier candidates with tissue compatibility more difficult to match are at an increased chance of receiving a transplant. These findings support a selection mechanism in which higher transplant rates accelerate transplants for higher-health candidates, and favour healthy individuals waiting longer.

Despite large differences in transplant rates, *overall survival* differences across blood types are surprisingly modest. A 1% increase in the transplant rate over the first five years on the waitlist implies only a 0.04% increase in survival after 7 years, and 0.01% after 12 years. One explanation, consistent with previous results in the paper, is that healthier candidates, who are already more likely to survive, disproportionately benefit from higher transplant rates. For these individuals, short delays in receiving a transplant may have little effect on outcomes. Meanwhile, among the sickest candidates, earlier transplants confer limited gains due to high graft failure risks. The largest benefits of earlier transplants may therefore be concentrated in the middle of the health distribution. If relatively few of these intermediate candidates are induced to receive a transplant under the high-transplant regime, then overall survival differences between AB and O blood groups may remain modest.

The final section of the paper considers which agents – candidates, physicians, or transplant centers – face incentives most likely to generate the observed selection patterns. The discussion draws on relevant literature ([Chan and Roth, 2024](#); [Agarwal et al., 2020](#); [Axelrod et al., 2017, 2018](#); [Stith and Hirth, 2016](#)) and on simulations from a canonical search model ([Mortensen, 1986](#)). Evidence points to transplant centers as the primary actors, particularly given historical evaluation metrics and financial incentives that favor the selection of healthier recipients.⁵ The paper also considers how candidates’ strategic behavior could contribute to these patterns, though stronger assumptions on information acquisition are needed to support this channel.

The discussion and results in this paper contribute to several fields. The methodological setup builds on a rich history of dynamic treatment effects. It illustrates how the framework of [Abbring and Van den Berg \(2005\)](#) can be leveraged to draw out insights

⁵These results, of course, do not necessarily imply a sub-optimal allocation of kidneys from a welfare perspective. If, as supported by [Agarwal et al. \(2020\)](#), the relative and absolute life-year gains of a transplant are larger for higher health candidates, then this skewed distribution may be optimal. This paper focuses on survival although we do discuss at times the dual outcomes of quantity and quality of life. Surveys show that transplants improve the latter relative to dialysis, the next best alternative ([Axelrod et al., 2018](#)).

about selection into treatment and propose testable hypotheses about selection mechanisms. The empirical results also contribute to recent theoretical work focused on optimal allocation mechanisms for the deceased donor waitlist which considers ABO blood compatibility issues (Sönmez et al., 2020; Kim and Li, 2022). This paper also contributes to various important theoretical and empirical insights focused on candidate option value of a transplant and discretion to reject offers (Zhang, 2010; Agarwal et al., 2018, 2021) and the first evaluation of transplants on survival using causal methods in Agarwal et al. (2020). Finally, the results also relate to research underlining the relevance of market dynamics in the supply of kidneys (Chan and Roth, 2024; Stith and Hirth, 2016; Dickert-Conlin et al., 2019; Teltser, 2019). In particular, this paper shows that increases in the supply of kidneys do not necessarily translate into proportional increases in transplants across candidates of varying health. Without changes to the incentive structure, new donations may continue to be allocated to relatively healthy, recently listed candidates. These implications are discussed in light of recent institutional reforms to transplant center evaluations in the U.S. system.

The remainder of the paper proceeds as follows. It begins in the next section by briefly introducing the kidney transplant allocation system. It then displays the descriptive patterns of transplant rates and survival which form the basis for the analysis of selection mechanisms. Section 3 introduces the dynamic causal setting, describes and identifies regime and selection effect, and describes the estimation approach. Section 4 describes the data and Section 5 discusses results.

2 Kidney Transplant Allocation System

In the United States, the allocation of deceased donor kidneys is designed, coordinated, and administered by the Organ Procurement and Transplantation Network (OPTN). A person becomes eligible to register on the kidney transplant waitlist and begin accruing time once their kidney function falls below 20mL/min.⁶ Upon entry, OPTN collects detailed information on the candidate’s health, immunological profile, and other characteristics relevant for kidney compatibility.

When a potential kidney becomes available, OPTN gathers extensive biological information on the kidney and the donor’s medical history. Through its automated UNet system, OPTN calculates a priority order for all compatible candidates. The offer process aims to balance equity and efficiency, assigning priority based on a strict set of criteria. Unlike some other organs (e.g., livers or hearts), priority is not given to those in most urgent need.

Prior to December 2014, the main prioritization criteria were blood and tissue type compatibility, the candidate’s age, waitlist duration, and geographic proximity to the center holding the deceased donated kidney (Smith et al., 2012).

⁶More precisely, when the Glomerular Filtration Rate (GFR) is 20 or below.

This allocation system was criticized for leading to excessive kidney discards, lost graft-years,⁷ high re-transplant rates, and inequities in access for minority patients. In response, a new allocation policy was implemented in December 2014. Key changes included awarding additional points to highly sensitized patients (those with high antibody levels) and prioritizing healthier candidates for the longest-lasting kidneys. This latter prioritization is guided by an Estimated Post-Transplant Survival (EPTS) score, which depends on age, diabetes status, dialysis status and duration, and whether they previously received a transplant.⁸ Under the new system, less emphasis is placed on waitlist time and geographic proximity.⁹

In terms of ABO blood type prioritisation, B and O blood type candidates are prioritised for identical blood type kidneys unless perfect zero-antigen tissue compatibility exists with AB (or A for O donors) blood types (Israni et al., 2014). This policy, unchanged by the 2014 reform, aims to compensate for the lower supply of B and O kidneys. Crucially for this paper, the UNet algorithm determines kidney offers based on observed variables, all health-related components of which are available in the data.

There also exists some discretion on the part of candidates and their physicians concerning which kidney they are willing to accept. Many kidneys are automatically filtered out based on pre-specified criteria regarding donor age, health, and kidney function. These acceptance criteria are recorded in the data. Even after passing these filters, additional offers are frequently rejected (Gordon et al., 2020). Using data from January 1, 2010 to December 31, 2013, Agarwal et al. (2018) show that among kidneys passing initial screens, only 0.9% of offers are accepted, and just 70% of those are eventually transplanted.¹⁰ The low acceptance rate is largely due to highly undesirable kidneys being offered to thousands of patients.¹¹ Rejections have no impact on future offers. As a result, nearly 25% of kidneys are ultimately discarded.¹²

3 Evaluation Framework

This section first presents descriptive patterns in transplant and survival outcomes that motivate the analysis of selection mechanisms. It then introduces the evaluation design

⁷For example, candidates with shorter life expectancy might receive kidneys capable of lasting significantly longer.

⁸More specifically, candidates in the top 20% of EPTS scores are prioritized for kidneys from donors in the top 20% of the Kidney Donor Profile Index (KDPI).

⁹In 2021, geographic criteria were further refined to assign points on a linearly decreasing scale (with notches) based on distance between the candidate’s transplant center and the donor hospital (Potluri and Bloom, 2022).

¹⁰This discrepancy arises primarily from final crossmatch screenings, which identify immunological incompatibilities likely to cause graft failure.

¹¹Factors influencing kidney quality include donor health, age, gender, anatomy, and infectious disease history. See Danovitch (2012) for an overview.

¹²There is also considerable variation in acceptance behavior across the roughly 250 transplant centers (Wey et al., 2017).

using the framework of [Abbring and Van den Berg \(2005\)](#), adapting it to the kidney transplant setting. A final subsection outlines a simple estimation strategy.

3.1 AB vs. O Transplant Rates and Survival Probabilities

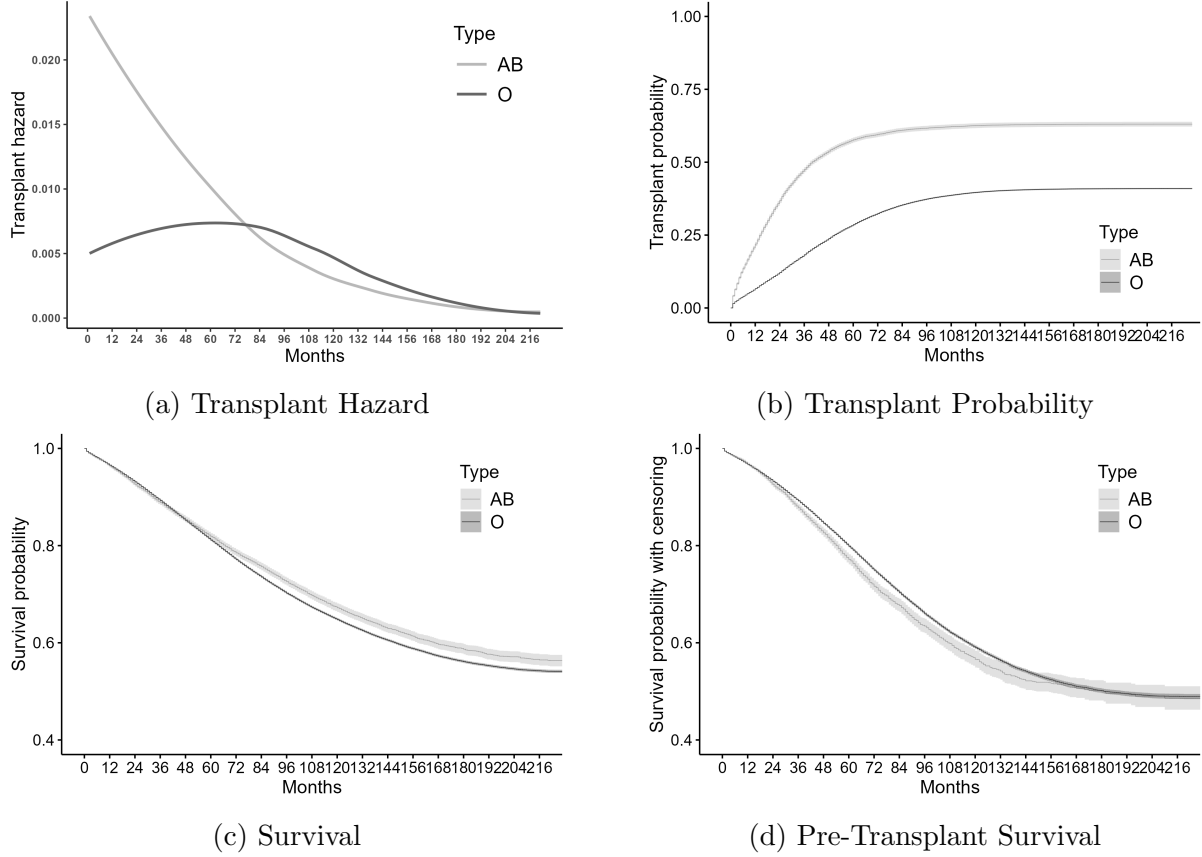


Figure 1: Survival of candidates on the kidney transplant waitlist

Includes candidates entering waitlist after 01 June 2002. Based on selected sample of Scientific Registry of Transplant Recipients data described in [Appendix A](#).

Observations: $N_O = 170,463$, $N_{AB} = 13,393$.

Figure 1 presents the central patterns under study using administrative data from the Organ Procurement and Transplantation Network (OPTN).¹³ Figure 1a shows the transplant hazard from deceased donors for AB blood types – universal recipients – and O blood types, who can only receive from O donors.¹⁴ Due to compatibility constraints, AB candidates face a consistently higher transplant hazard during the first four years on the waitlist, resulting in a transplant probability (Figure 1b) that is roughly twice as high as that of O candidates over the first five years.

Despite the large differences in transplant probabilities, and relatively steep declines in survival over time, Figure 1c reveals a striking and previously undocumented fact:

¹³Data from the OPTN/Scientific Registry of Transplant Recipients (SRTR) include socio-economic and medical histories of all waitlist candidates and donors.

¹⁴Earlier economic interest focused on kidney exchange programs for living donors ([Roth et al., 2004](#); [Agarwal et al., 2019](#)), but these represent a minority; over 70% of kidney transplants are from deceased donors.

overall survival rates for AB and O candidates are nearly identical for the first five years, diverging only modestly thereafter. This raises the question of whether transplants have limited effects on survival, or whether selection into transplantation is shaping outcomes.

Figure 1d reveals a further striking pattern. It plots the probability of survival for AB-types and O-types after censoring the duration to death at the moment a candidate receives a kidney transplant. Here, we observe a clear divergence: AB candidates, who have a higher transplant rate, exhibit lower survival prior to transplant compared to O candidates.

3.2 Regime and Potential Treatment Duration

We now turn to a framework from Abbring and Van den Berg (2005) and explain how it can be leveraged to draw out insights about positive or negative dynamic selection into treatment. We follow individuals from entry into the initial state ($t = 0$), which corresponds in our setting to waitlist entry. In other settings it could be the moment of contracting an illness, becoming unemployed, or advertising a new product on the market. At $t = 0$, agents are assigned to a regime $Z \in \{0, 1\}$ – e.g., $Z = 1$ for AB-type candidates and $Z = 0$ for O-type candidates – which affects their transplant hazard. Z is observed by the econometrician and may be partially or fully known to agents.

The regime Z is not a direct treatment assignment but an intention-to-treat variable that shifts the stochastic propensity of future treatment. We define S^z as the potential duration to treatment under regime $Z = z$, and $T^{z,s}$ as the potential duration to death, for treatment occurring at s . The objects of interest in this study will compare the probability of survival beyond a fixed horizon τ across regimes. As is standard, we define $T^{z,\infty}$ as the potential outcome under no treatment. In this context, it corresponds to the time to death for candidates who remain untreated in regime z .¹⁵

In the data, we observe (Z, S, T, X) for cases where $T \geq S$, with X representing covariates measured at baseline.

3.2.1 A strategy to assess pre-transplant and selection effects

Our objective is to assess whether differences in pre-transplant survival across regimes reflect a selection effect on the timing of treatment. The strategy unfolds in three steps.

Step 1: First, we assume there is a variable which randomly varies the transplant rate at time 0. In this paper, that amounts to assuming blood types, conditional on a set of observed variables, are random with respect to potential transplant and survival times. Formally, we assume, denoting by $\{T^{z,s}\}, \{S^z\}$ the sets of all potential variables, that,

$$(\{T^{z,s}\}, \{S^z\}) \perp\!\!\!\perp Z \mid X = x$$

¹⁵We focus on survival probabilities rather than expected durations because a large share of survival times are censored, so the right tail of the exit distribution will be poorly approximated.

Step 2: If this assumption holds, then we can evaluate the following pre-transplant value for different time horizons of τ ,

$$\beta_{\tau,z',z,x} = \prod_{t=0}^{\tau} \Pr(T > t | S > t, T \geq t, X = x, Z = z') - \prod_{t=0}^{\tau} \Pr(T > t | S > t, T \geq t, X = x, Z = z) \quad (1)$$

This is the empirical difference in pre-transplant survival at τ between the high transplant rate z' (eg. AB blood group) and the low transplant rate z (eg O blood group) when conditioning on variables $X = x$.

A negative value of $\beta_{\tau,z',z,x}$ indicates that the high-hazard regime z' has lower pre-transplant survival than the low-hazard regime z by time τ (as observed in our data; see Figure 1d). Two distinct mechanisms could produce a negative pre-transplant survival gap.

- **Mechanism 1 (Regime effects on pre-transplant survival):**

$$\mathbb{E}[T^{z',\infty}] < \mathbb{E}[T^{z,\infty}]$$

This condition implies that individuals assigned to the higher-transplant-rate regime (z') would, in the absence of treatment, have lower survival prospects than those in the lower-transplant-rate regime (z).

- **Mechanism 2 (Regime effects on treatment timing):**

$$\frac{\partial \Pr(S^{z'} \leq s \mid X = x, Z = z', U = u) - \Pr(S^z \leq s \mid X = x, Z = z, U = u)}{\partial u} > 0$$

where $u \in \mathbb{R}$ represents a scalar unobserved health index, with survival probability increasing in u . This condition captures a regime effect on the *timing* of treatment. It implies that the advantage of being in the higher transplant-rate regime ($Z = z'$) – in terms of receiving a transplant by time s – is increasing in unobserved health status u . In other words, healthier candidates are disproportionately more likely to benefit from faster access to transplants under the higher-rate regime.

The first mechanism is not a selection effect. It represents a direct causal effect of the transplant regime on survival for candidates who have not yet been treated. In our context, $\mathbb{E}[T^{z',\infty} - T^{z,\infty}] < 0$ would hold if a higher transplant rate leads to a lower survival for candidates waiting for a transplant on the waitlist. Such an effect may reflect moral hazard if candidates adjust their actions based on the anticipated likelihood of receiving a transplant.

The second mechanism, by contrast, reflects a selection effect in the timing of treatment rather than a causal regime effect on untreated survival. In this case, $\mathbb{E}[\beta_{\tau,z',z,x}] < 0$ can hold even if $T^{z',\infty} = T^{z,\infty}$. This form of selection can arise either from the behaviour

of transplant centres or from the choices of candidates. If transplant centres are the primary agents allocating kidneys, the condition for Mechanism 2 holds when the distribution of transplants is more heavily skewed toward healthier candidates in the high transplant-rate regime than in the low one. From the perspective of candidate choice, the same condition would hold if healthier individuals are less likely to postpone accepting a transplant in the high-rate regime than in the low-rate regime, relative to less healthy candidates. Although delaying transplant is risky, it may be optimal for sicker candidates if they expect to receive a higher-quality kidney in the future. Appendix F presents a simple one-period stylised model to build intuition for these survival dynamics.

Step 3: The third step tests for Mechanism 2, selection effects, on a restricted subset of candidates. If kidneys are allocated proportionally with respect to underlying health across the high and low transplant-rate regimes, then post-transplant survival for candidates treated at period $t = 1$ should be similar across regimes. If it is not, this suggests that selection effects are already at work early in the waitlist process. This comparison, however, loses validity for $t > 1$ if selection or Mechanism 1 effects occur at $t = 1$, since selective transplantation and/or survival alters the composition of the remaining not-yet-treated candidates across regimes. Nonetheless, as we show in the results section, descriptive comparisons of the characteristics of transplanted candidates over time can still shed light on the evolving nature of selection when it is present. This final step provides qualitative insight into the presence of selection in the initial periods on the waitlist. This last step can on its own speak qualitatively to the question of whether selection is present, at least in initial periods on the waitlist. It does not, however, yield a formally interpretable causal parameter for the selection effect.

3.2.2 Interpreting Regime and Selection Effects

While the previous steps clarify the direction of selection effects, when present, they do not directly relate $\beta_{\tau, z', z, x}$ to a quantifiable causal estimand. This subsection provides identification results and guidance on interpreting the pre-transplant effect.

We begin by considering the case in which *Mechanism 1* (regime effects on survival) operates in isolation, with the selection effect *Mechanism 2* (regime effects on treatment timing) absent. Suppose, in addition, that we observe all variables relevant to transplant allocation. Under the sequential unconfoundedness assumption,

$$\{T^{z,s}\} \perp\!\!\!\perp \mathbf{1}(S = s) \mid S \geq t, T \geq t, Z = z,$$

the pre-transplant survival difference $\beta_{\tau, z', z, x}$ identifies the causal effect of regime assignment on untreated survival:

$$\beta_{\tau, z', z, x} = \Pr(T^{1,\infty} > \tau \mid X = x) - \Pr(T^{0,\infty} > \tau \mid X = x).$$

This result follows from Robins' G-computation formula (Robins, 1986).¹⁶

Next, consider the case where *Mechanism 2* operates and *Mechanism 1* is absent. The causal interpretation of $\beta_{\tau,z',z,x}$ is then more subtle and best understood period by period. For $\tau = 0$, we can write:

$$\begin{aligned}\beta_{0,z',z,x} &= \Pr(T > 0 \mid S > 0, T \geq 0, X = x, Z = z') - \Pr(T > 0 \mid S > 0, T \geq 0, X = x, Z = z) \\ &= \Pr(T^\infty > 0 \mid S^{z'} \neq 0, X = x) - \Pr(T^\infty > 0 \mid S^z \neq 0, X = x),\end{aligned}$$

using the fact that $T^{z',\infty} = T^{z,\infty} = T^\infty$ under the assumption of no regime effects on pre-transplant survival. This difference reflects the difference in survival across regimes among candidates who are not selected for transplant at $t = 0$.

We can gain sharper identification under an additional *monotonicity* assumption in treatment timing: $S^{z'} \leq S^z$. Under this condition, any candidate who receives a transplant in period 0 under the low-transplant regime ($Z = z$) also receives a transplant in that period under the high-transplant regime ($Z = z'$). Thus, the observed difference isolates candidates who are treated early under $Z = z'$ but not under $Z = z$. The survival difference then reflects selection in transplant timing. This monotonicity assumption is plausible if transplant centers are the primary decision-makers and transplant offers increase mechanically with regime Z . It may be less tenable if differences arise from candidate-side optimization over unobserved health states.

The interpretation for general $\tau > 0$ follows analogously:

$$\begin{aligned}\beta_{\tau,z',z,x} &= \prod_{t=0}^{\tau} \Pr(T^\infty > t \mid S^{z'} \neq t, T^\infty \geq t, X = x, Z = z') \\ &\quad - \prod_{t=0}^{\tau} \Pr(T^\infty > t \mid S^{z'} \neq t, T^\infty \geq t, X = x, Z = z)\end{aligned}\tag{2}$$

An alternative measure of pre-transplant effects may have considered,

$$\beta_{\tau,z',z,x} = \Pr(T > \tau \mid S > \tau, X = x, Z = z') - \Pr(T > \tau \mid S > \tau, Z = z)\tag{3}$$

However, this alternative is problematic in two respects. If Mechanism 1 is present without Mechanism 2, this measure will not recover the causal regime effect on pre-transplant survival. This is because the sequential unconfoundedness assumption on treatment $\{T^{z,s}\} \perp\!\!\!\perp \mathbb{1}(S = s) \mid S \geq t, T \geq t, Z = z$ asserts that transplant is sequentially random conditional on survival. The latter measure requires a far stronger assumption of randomness of transplants unconditional on survival to identify the causal pre-transplant regime effect on survival. If Mechanism 2 is present without Mechanism 1, this measure ignores accumulated information about survival prior to τ , thereby conflating selection dynamics over time. As we demonstrate empirically, this can substantially exaggerate selection effects when they are present.

¹⁶This interpretation also requires standard SUTVA and support conditions.

3.2.3 Unconfoundedness and the Role of Covariates in Assessing Selection

The empirical analysis in this study rests on the assumption that $(\{T^{z,s}\}, \{S^z\}) \perp\!\!\!\perp Z \mid X = x$. With respect to its plausibility, it is known that ABO blood types are associated with certain disease processes, including vascular conditions and malignancies (Wu et al., 2008; Sun et al., 2015; Abegaz, 2021), as well as with a limited set of infectious diseases (Cooling, 2015). However, there is little evidence that genetic variation in ABO blood groups systematically predicts mortality. Nevertheless, because ABO distributions differ by race and may correlate with other demographic or medical characteristics, we empirically assess the robustness of this assumption.

The analysis proceeds in three stages. We first estimate specifications without any covariates, then add the official OPTN variables used in prioritizing waitlist candidates, and finally include the most comprehensive set of demographic, health, and geographic controls permitted by data availability. This sequential approach allows us to examine the sensitivity of the pre-transplant estimates of $\beta_{\tau,z',z,x}$ to different sets of observable variables. We also report results disaggregated by race, sex, and education. In a complementary step, we leverage all four ABO blood types to illustrate the ordinal empirical relationship between transplant rates and pre-transplant survival outcomes.

The procedure of presenting results with an increasing set of conditioning variables is also important when qualifying the analysis of selection. Given a sufficiently rich set of observed covariates, one might argue that selection can always be explained in terms of observables. Accordingly, all discussion of selection on unobservables in this paper should be understood as conditional on the covariates included in each specification.

3.3 Estimation Approach

We estimate $\beta_{\tau,z',z,x}$ using several approaches that involve trade-offs specific to our setting. The primary estimator is based on a semi-parametric continuous-time proportional hazards model with competing risks.¹⁷ Because survival and treatment processes are jointly determined, we model their hazards as:

$$\begin{aligned}\theta_t^T(x, z, s) &= \lambda_t^T(z, \mathbb{1}(s \leq t)) \exp(x' \beta^T) \\ \theta_t^S(x, z) &= \lambda_t^S(z) \exp(x' \beta^S)\end{aligned}$$

where θ_t^T and θ_t^S denote the instantaneous hazards of death and treatment, respectively. Duration dependence functions λ_t^T and λ_t^S are specified as piecewise constant baseline hazards, varying by regime z and whether treatment has occurred by time t .¹⁸

¹⁷A continuous-time specification is preferred primarily for computational reasons. When identifying assumptions are plausible and time intervals are short, continuous and discrete-time duration models typically yield similar estimates (Kastoryano and van der Klaauw, 2022). The latter also offer non-parametric discrete-time estimators adaptable to our setting and discuss relevant trade-offs.

¹⁸We use one-year intervals for the piecewise segments. This choice is guided by the patterns observed in Figure A1b (Appendix C) and ensures sufficient flexibility while maintaining computational tractabil-

Estimation proceeds by Maximum Likelihood, where all survival durations are right-censored at the time of transplant. The resulting estimator yields individual-level predicted hazards, which we use to compute the pre-transplant survival functions in $\beta_{\tau,z',z,x}$ of the form $\prod_{t=1}^{\tau} \Pr(T > t \mid S > t, T \geq t, Z = z)$:

$$\sum_{\{i\}} \hat{w}_i(s) \exp\left(-\int_1^{\tau} \hat{\theta}_t^T(x_i, z, S > \tau) dt\right) = \sum_{\{i\}} \hat{w}_i(s) \exp\left(-\int_1^{\tau} \hat{\lambda}_t^T(z_i, 0) \exp(x_i' \hat{\beta}^T) dt\right)$$

where the weights $\hat{w}_i(s)$ reflect the estimated distribution of treatment times:

$$\hat{w}_i(s) = \frac{\hat{\theta}_s^S(x_i, 1) \exp\left(-\int_0^s \hat{\theta}_{\tau}^S(x_i, 1) d\tau\right)}{\sum_{\{i\}} \hat{\theta}_s^S(x_i, 1) \exp\left(-\int_0^s \hat{\theta}_{\tau}^S(x_i, 1) d\tau\right)}$$

We compute standard errors for $\beta_{\tau,z',z,x}$ using the delta method.¹⁹

As a calibration check on the estimator, we show throughout the results that our estimates of $\beta_{\tau,z',z,x}$ without including any covariates are similar in magnitude to differences in Kaplan-Meier survivor functions after censoring survival at the moment of transplant (e.g. as in Figure 1d). As is well known, the drawback of this estimator is that it faces computational limitations when the covariate space is high dimensional.

To address this, we also consider alternative estimators better suited to non-linearities and high-dimensional covariate spaces. Specifically, we explore non-parametric (nearest-neighbor matching) and machine learning approaches, including double/debiased ML, Cox-Lasso, random survival forests, and deep learning models for survival analysis. The non-parametric estimator yields results similar to the baseline model. The machine learning approaches do not perform particularly well in our setting, with results mimicking the upward bias from a simple, and mis-specified, OLS estimation on pre-transplant survival. The main reason for the poor performance is that current ML models are not well-suited to accommodate competing risks and random right censoring, or not designed for causal inference.

4 Data

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

ity. We report results only up to five years, as estimation becomes less stable at longer durations due to the small number of AB candidates treated after year five.

¹⁹Replication materials and estimation code are available at www.skastoryano.com.

The dataset contains detailed information on candidate characteristics, waitlist entry dates, transplant dates, and dates of death. The baseline sample includes all candidates who entered the waitlist between June 1, 2002, and December 1, 2014 – the implementation month of the revised kidney allocation system. I exclude candidates listed for multiple-organ transplants and those under 18 years of age, both of whom follow distinct allocation rules. I also exclude candidates who received a living donor kidney, as they bypass the standard allocation process based on factors that are typically unobserved.²⁰ The analysis further restricts to first waitlist spells, excluding individuals who re-enter due to graft failure or renewed kidney failure, and removes rare blood type subgroups and immune-sensitized candidates with an initial cPRA score above zero, who face exceptional allocation rules. Appendix A documents the data cleaning process and the share of observations excluded at each stage.

The main decomposition analysis uses a time unit of two months. The outcome of interest, T , is the duration from waitlist entry to death, if observed. Death dates are obtained from Social Security records (Massie et al., 2014).

The assignment variable Z is the biological randomization to blood type AB or O. I focus on these two groups to identify pre-transplant survival differences. As shown in Figure A2a (Appendix C), AB candidates – universal recipients – have the highest transplant hazard in the first three years on the waitlist. In contrast, candidates with O and B blood types have the lowest transplant probabilities, while A types fall in between.

The main specification controls for all OPTN-relevant health variables used in allocation and additional health-related covariates with low rates of missingness. Prior to the 2014 reform, only age was relevant for deceased donor offers in the selected sample. Post-reform, only the Estimated Post-Transplant Survival (EPTS) score enters into offer prioritization.²¹ Covariates measured at $t = 0$ include age, diabetes status, dialysis status, prior non-kidney transplant, sex, race, and primary diagnosis.²² We also include, in some specifications, a set of additional health-related variables that require caution. These variables could, in principle, be imbalanced across blood groups, but they are also updated over time, which raises concerns. Including them could lead to bad control problems if the updating over time is not independent from the transplant and survival outcomes. These variables include indicators for whether a candidate would accept a kidney from a hepatitis B- or C-positive donor, BMI, and a history of malignant tumours.²³

²⁰This exclusion may be endogenous to blood type if the likelihood of securing a living donor varies by blood group. I return to this point in the results section.

²¹EPTS is computed as: $EPTS = 0.047 \cdot \max(\text{Age} - 25, 0) - 0.015 \cdot \text{Diabetes} \cdot \max(\text{Age} - 25, 0) + 0.398 \cdot \text{Prior Transplant} - 0.237 \cdot \text{Diabetes} \cdot \text{Prior Transplant} + 0.315 \cdot \log(\text{Years on Dialysis} + 1) - 0.099 \cdot \text{Diabetes} \cdot \log(\text{Years on Dialysis} + 1) + 0.130 \cdot \mathbb{1}\{\text{Years on Dialysis} = 0\} - 0.348 \cdot \text{Diabetes} \cdot \mathbb{1}\{\text{Years on Dialysis} = 0\} + 1.262 \cdot \text{Diabetes}$.

²²This mirrors the covariates in (Wolfe et al., 2008), excluding those with high missingness: Time on Dialysis (26.5%) and Albumin (13.2%). Matching and machine learning models also include transplant center location.

²³The model does not include other relevant variables which present a large fraction of missing values: hypertension (26% missing), creatinine measure of candidate (33% missing). We also exclude a variable

I report results separately for candidates entering before and after the December 2014 reform, since priority rankings were redefined at that time.²⁴ Most robustness checks focus on the pre-reform sample, which allows for longer-term outcome evaluation and a larger sample of AB candidates. Summary statistics by blood type are shown in Appendix B, Table A1. Pre-reform, the average candidate is 52 years old, 62% male, with 75% on dialysis and 35% diabetic at entry. While blood types are generally balanced on health indicators, there are marked racial differences: the B blood type group has a higher share of Black candidates, and AB/B groups include more Asian candidates. Some imbalance in education also exists and is addressed in robustness analyses.

5 Results

This section presents the main empirical findings. We begin by assessing whether pre-transplant survival varies systematically across blood types, and hence across transplant rates. In the process, we also discuss the plausibility of our regime unconfoundedness assumption. We then estimate the survival effect of receiving a transplant early and examine how the characteristics of transplanted patients evolve over time. Next, we consider overall differences in survival between blood types with different transplant rates. Finally, we discuss which agents may be driving the observed patterns, drawing on existing literature.

5.1 Pre-transplant effects and observed variables

We first examine how much of the pre-transplant survival differences shown in Figure 1d can be attributed to selection on observed characteristics. Figure 2a displays the pre-transplant effect $\beta_{\tau, AB, O, x}$ – the difference in survival at horizons 1 through 5 years – without covariates. Figure 2b adds allocation-relevant covariates (age brackets) to the model. In both specifications, candidates with AB blood type exhibit lower pre-transplant survival.

The estimates in Figure 2b suggest that, after five years, AB-type candidates have a survival probability 5 percentage points lower than O-type candidates. Given baseline survival rates (Figure A1b), this corresponds to a 25% higher risk of pre-transplant death among AB-type candidates, attributable to unobserved selection. Figure 2c includes additional health and demographic covariates from Appendix Table A1; the estimated effect remains sizable. Figure 2d shows that the effect persists post-2014 reform.²⁵ This result is consistent with the agent based structural model in Agarwal et al. (2021) showing

indicating a patient’s functional status since a patient’s latest measured activity level is unlikely to be independent of the kidney acceptance decision and death.

²⁴Pre-reform candidates are censored at the date of reform.

²⁵Figure A1 of Appendix C presents the transplant hazard and pre-transplant survival for the sample entering the waitlist before and after the December 2014 reform. Although the transplant hazard for AB types is slightly muted in initial months on the waitlist after 2015, the general patterns are similar.

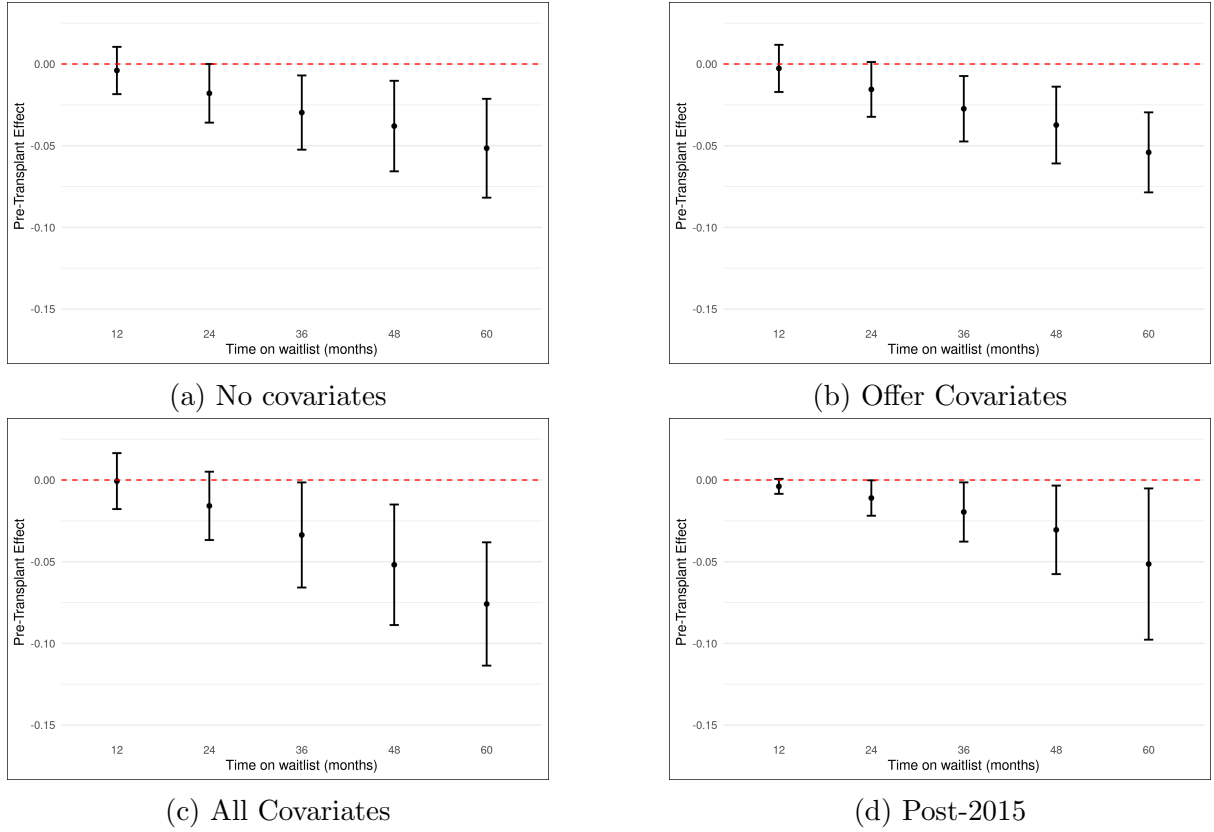


Figure 2: Pre-transplant difference in survival effect $\beta_{\tau, z', z, x}$. Includes candidates entering waitlist after 01 June 2002. Based on selected sample of Scientific Registry of Transplant Recipients data described in Appendix A.

(a) includes no covariates, (b) includes offer covariates: age bracket dummies, (c-d) includes all variables presented in Appendix Table A1. Observations (a-c): $N_{O:no-Tr} = 93,922$, $N_{O:Tr} = 35,989$, $N_{AB:no-Tr} = 5125$, $N_{AB:Tr} = 5032$. Observations (d): $N_{O:no-Tr} = 43,338$, $N_{O:Tr} = 11,698$, $N_{AB:no-Tr} = 2241$, $N_{AB:Tr} = 2176$.

the new system only modestly improves patient welfare and discard rates relative to the pre-2014 mechanism. Hereafter, all modelled pre-transplant effects include all covariates.

Appendix D considers additional estimation approaches. Figure A5a uses propensity score matching – with exact matching on race – and including transplant center fixed effects prior to hazard estimation, yielding similar results. Figures A5b and A5c report double/debiased ML estimates with and without inverse probability of censoring weights (IPCW).²⁶ These estimates are larger and more efficient than those from our main estimator, but they closely mirror results from a simple OLS model that does not account for censoring or competing risks. This suggests that they may fail to appropriately adjust for the structure of the data. For this reason, we focus the remainder of the analysis on the joint proportional hazards model which is better suited to the empirical setting.²⁷

²⁶As yet, to the author’s knowledge, there is no standard approach for double/debiased ML causal estimation which accommodates competing-risk survival settings with right censoring. Our adapted double/debiased ML model is described in the footnote to Figure 2 but not accompanied by theoretical results. It is a double/debiased ML model where each observation is weighted by an inverse probability of censoring weighting function estimated by neural nets to account for the random right censoring of the outcome and censored outcomes at the moment of transplant (competing-risk censoring).

²⁷Appendix D Table A2 also presents results from machine learning approaches designed for survival models - Cox-lasso, random survival forest, deep learning for survival models. The results from these

Taken together, Figures 2a–2d document substantial pre-transplant survival differences that cannot be explained by observed variables. Appendix Figure A4 shows these effects are primarily driven by White and male candidates, with weaker or null effects for minorities and women. The results also hold when including living donor transplants.

5.1.1 Transplant rates and pre-transplant survival across blood types

As discussed in previous sections. Three explanations are consistent with these results. The first is that blood-type groups are not random with respect to pre-transplant survival and that our main unconfoundedness assumption is violated. The second is that there is a regime effect on pre-transplant survival (Mechanism 1) and the third is that there is a regime effect on the timing of transplant (Mechanism 2), which we also refer to as the selection effect.

To offer further counter-evidence to the first explanation, that blood types are not random, Figure 3 examines transplant rates and pre-transplant survival across all ABO types for White candidates.²⁸ A clear ordinal pattern emerges: the higher the transplant probability, the lower the pre-transplant survival. Figures 3c and 3d confirm this with modelled pre-transplant effects.

These patterns are consistent with the interpretation that regime effects – either Mechanism 1 or Mechanism 2 – are driving the observed differences. As we discuss in the next sections, the most plausible explanation, supported by existing literature, is that transplant centers preferentially allocate kidneys to healthier candidates under high-transplant regimes. This leads to negative selection among the remaining candidates on the waitlist in those regimes. If this is indeed the dominant mechanism, we can also offer a setting-specific, quantitatively interpretable result. From Figure 1a, we estimate that AB blood type candidates face, on average, a transplant rate 2.5 times higher than O blood type candidates over the first five years on the waitlist. Combined with the observed 25 percent difference in pre-transplant survival, this suggests that a 1% increase in the transplant rate corresponds to a 0.1% increase in transplants among candidates who would have otherwise survived five years without receiving a kidney.

models do not perform well in the current setting and produce unrealistic effects. This is likely because they are not designed for causal evaluation in survival models with random and competing risk right censoring.

²⁸Appendix Figure A2 presents corresponding hazard and overall survival.

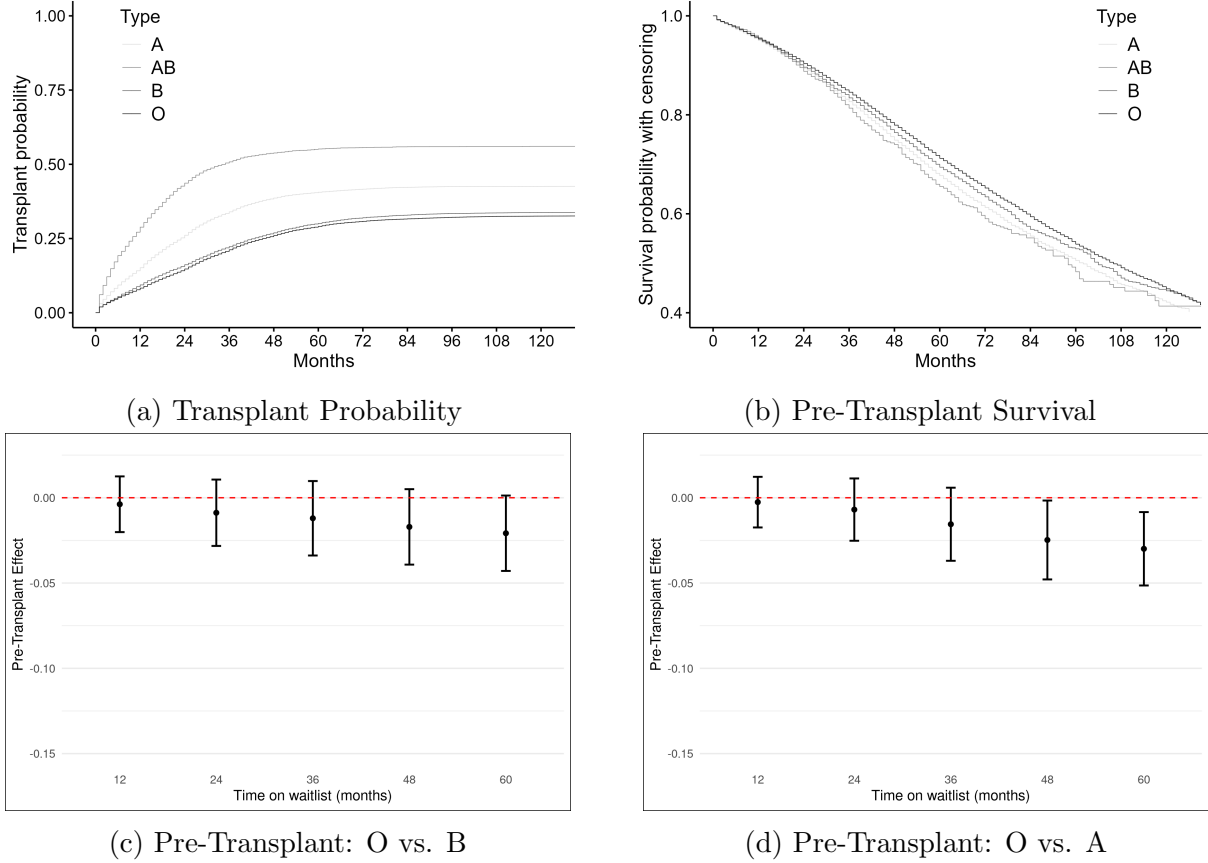


Figure 3: Treatment probability and Pre-transplant survival effects $\beta_{\tau, z', z, x}$ for A and B blood type candidates

Includes candidates entering waitlist after 01 June 2002, durations truncated at 01 December 2014. Based on selected sample of Scientific Registry of Transplant Recipients data described in Appendix A. Covariates include all variables presented in Appendix Table A1. Observations: $N_O = 71,075$, $N_{AB} = 4764$, $N_B = 14,887$, $N_A = 50,149$.

5.2 Selection and the Effect of a Transplant on Survival

The previous section provided evidence of differences in pre-transplant survival across blood types, which persist even after adjusting for variables related to waitlist prioritization, as well as baseline health, demographic, and geographic characteristics. To distinguish regime effects on pre-transplant survival from selection effects related to treatment timing, we now restrict attention to candidates who received a transplant within the first three months of waitlist entry.

In general, if transplant centers act as primary decision-makers and prioritize healthier candidates, then higher transplant rates could affect post-transplant survival in opposing ways. On one hand, a *timing effect* may dominate: at any given waitlist duration, higher transplant rates for AB candidates could lead to earlier transplantation of relatively healthier individuals, increasing survival in the AB group relative to the O group. On the other hand, a *composition effect* may also be present: if transplant centers adopt a top-down strategy by first selecting high-health candidates and then moving to lower-health ones, then the transplanted AB group may eventually include a larger share of

medium- or low-health individuals than the O group, potentially lowering AB survival.

To further confirm the timing effect is more important than the composition effect, we compare post-transplant survival for AB and O candidates who received a kidney within the first three months on the waitlist. Specifically, we examine various parametric and non-parametric causal specifications of the following,

$$Y_i = f(\mathbb{1}(\text{blood}_i = AB), X_i, u_i) \quad (4)$$

Figure 4a reports results for White candidates – the largest racially homogeneous group – transplanted within the first three months. We present (i) an unadjusted regression of survival on blood type, (ii) a linear regression controlling for OPTN baseline prioritization variables, (iii) a double/debiased ML specification using all candidate health variables, as well as education, functional ability, and location indicators, and (iv) an expanded double/debiased ML specification including donor and donor-recipient match variables from Table A1 of Appendix B.

All specifications yield similar results: higher transplant rates for early-transplanted AB candidates are associated with significantly greater survival over the first 4–5 years post-transplant.²⁹ Supporting evidence comes from Figures 4c and 4d. Figure 4c shows that AB candidates transplanted within the first three months have significantly higher functional ability – a proxy for health³⁰ – than O candidates. However, this difference largely disappears beyond three months.

Figure 4d plots the time trend in average functional ability for AB and O transplant recipients, and is also informative. If transplant centers had adopted a top-down strategy – prioritizing healthier candidates first but then moving on to transplant less healthy candidates – we would expect functional ability to decline over waitlist time, particularly for AB candidates. Yet, Figure 4c shows that, despite remaining longer on the waitlist, functional ability at the time of transplant remains roughly constant or even slightly increasing beyond three months.³¹ Figure 4e further shows that age among transplant recipients also declines with time on the waitlist, suggesting persistent emphasis on healthier candidates (younger age as a proxy).

From Figures 4a, 4c, 4d and 4e we conclude that a timing effect dominates, with healthier AB candidates more likely to receive transplants, particularly in early periods on the waitlist. From the perspective of transplant centers as the main decision-makers, the evidence suggests they do not follow a strict top-down allocation model and do not proportionally expand transplants across the full health distribution as transplant rates

²⁹The results are robust to narrowing the sample to transplants within the first month, as shown in Figure A6a in Appendix E. Similar patterns appear post-2015 (Figure A6c), though with smaller samples and slightly delayed effects due to a slightly offset transplant hazard rate for the post-2015 sample.

³⁰This constructed measure ranges from 1 to 3: 1. poor health and functional ability, 2: medium health and functional ability, 3: high health and functional ability.

³¹Fluctuations after 48 months stem from small sample sizes and are not significantly different by blood type.

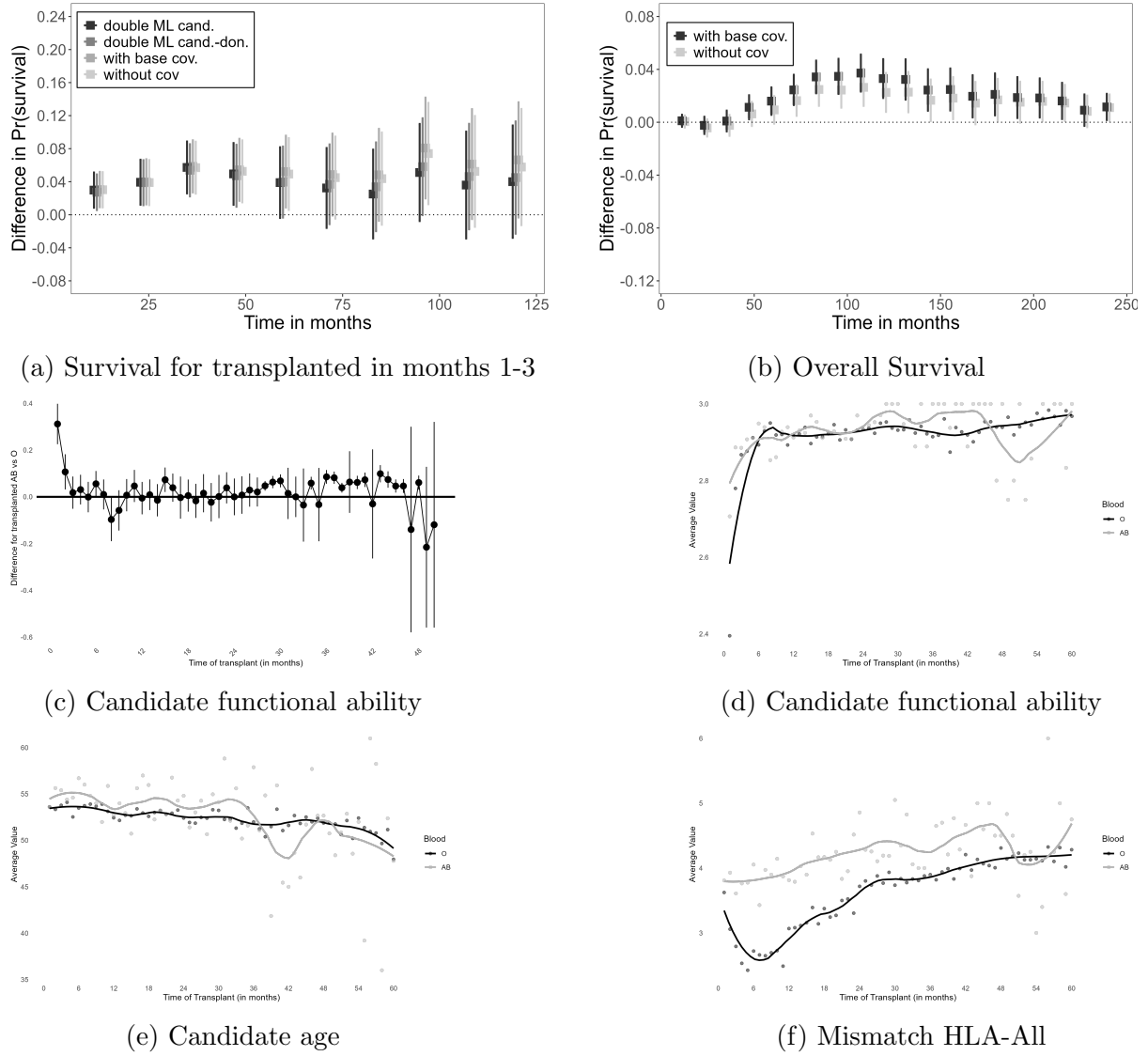


Figure 4: Difference in survival between AB and O blood types over time

Includes candidates entering waitlist after 01 June 2002, durations truncated at 01 December 2014. Based on selected sample of Scientific Registry of Transplant Recipients data described in Appendix A.

(without cov) includes no covariates, (with base cov) includes offer bracket dummies, (double/debiased ML cand) includes all candidate variables presented in Appendix Table A1, (double/debiased ML cand-don) includes all candidate, donor, and candidate-donor match variables presented in Appendix Table A1. Observations (left): $N_{O:treated} = 2406$, $N_{AB:treated} = 577$. Observations (right): $N_O = 71,075$, $N_{AB} = 4764$.

rise. Instead, there is a tendency to prioritize healthier, newly listed candidates. This pattern is harder to reconcile with patient-driven decision-making. For example, if low-health AB candidates disproportionately defer early offers, the AB group should show a sharper functional ability decline over time, which is not observed in Figure 4d. In the next section we further inspect the plausibility of candidate decisions driving the regime effect for Mechanism 1 or 2. In short, for candidate decisions to explain the patterns observed in this section, candidates would need to form precise beliefs about offer rates and survival risks and act on them immediately upon entering the waitlist – yet cease to do so after three months. This assumption appears less credible than arguments consistent with transplant-center selection.

Finally, we note that survival differences shown in Figure 4a are not materially affected by controlling for donor or donor-recipient match variables.³² This could imply that kidney quality differences do not play a major role, or that the AB transplant rate increase does not yield substantially better organ quality.³³ Figure A7 in Appendix E shows AB recipients have unexpectedly higher HLA mismatches than O recipients.³⁴ However, these mismatch differences are highly correlated with missingness.³⁵ Hence, we cannot conclusively assess the role of kidney quality. A more reliable insight comes from average mismatch trends (Figure 4f), which increase over time. Alongside stable functional ability and declining age, this suggests that later transplants favor healthier candidates with rarer tissue profiles.

5.3 Overall Survival

In the final part of our analysis, we examine overall differences in survival between AB and O blood group candidates. Figure 4b presents estimates under various covariate specifications. The survival trajectories of the two groups are similar for the first three years following waitlist entry. Thereafter, AB candidates exhibit higher survival, peaking at approximately 4 percentage points above the O group around year 7. This difference gradually declines, stabilizing at roughly 1 percentage points by year 12. In relative terms, AB candidates have about a 11% higher survival probability after 7 years, and 2% after 12 years.

To quantify these differences, we use the approximated difference in transplant rate for AB vs O candidates – approximately 2.5 times higher over the first five years on the waitlist (as shown in Figure 1a). These values imply that a 1% increase in the transplant rate over this period is associated with a 0.04% increase in survival at 7 years, and 0.008% at 12 years.

At first glance, these effects may appear small, particularly considering that roughly 50% of AB candidates receive a transplant within five years. However, this modest impact can be consistent with a prioritization of healthier candidates for transplantation, as discussed in earlier sections. For such individuals, a delay of a few months may

³²Figure A6b in Appendix E shows similar survival patterns when further restricting the analysis to candidates with the highest observed functional ability, underlying the selection on unobserved variables.

³³In principle, because there are more kidney offers in the AB-blood type regime, the expected value of the chosen maximum quality kidney may be higher for AB types than for O types with similar characteristics. A simple proof of this result is provided in Appendix H. Whether this is the case in practice is unclear. The argument relies on the assumption that candidates and/or clinicians occasionally make joint decisions across multiple kidney offers. According to Agarwal et al. (2021), candidates receive an average of 17–18 offers per month. If the transplant hazard trends in Figure 1a approximate offer rates, then offer volumes are slightly higher in the early months. Since offers often arrive a few days in advance, it is possible that decisions are occasionally made simultaneously over several options. All else equal, this could enhance survival in the AB group.

³⁴HLA mismatches at loci A, B, and DR are critical for graft compatibility and survival. Lower mismatches reduce the risk of immune rejection and improve graft survival.

³⁵Over 15% of mismatch values are missing among transplant recipients, with similar correlations across other blood types.

have minimal consequences for survival. Conversely, candidates in poorer health gain fewer life-years from transplantation, given both their higher risk of graft failure and the likelihood of receiving lower-quality kidneys through assortative matching. Consequently, the survival gains from earlier transplantation are likely concentrated among candidates in the middle of the health distribution. If relatively few of these intermediate candidates are induced to receive a transplant under the high-transplant regime, then overall survival differences between AB and O blood groups may remain modest.

5.4 Explaining Positive Selection of Transplanted Candidates

Drawing on existing literature and simulations from a choice-theoretic search model, we offer two explanations for the regime affecting the timing of transplant (Mechanism 2: selection) and one consistent with a regime effect on pre-transplant survival (Mechanism 1).

5.4.1 Transplant Center Incentives

A first explanation of selection effects is that transplant centers act as the primary decision-makers and selectively allocate kidneys to relatively healthier candidates. Several institutional features of the U.S. kidney waitlist system support the idea that transplant probability increases with unobserved candidate health.

One key disincentive is financial: transplants for lower-health candidates are more costly both in absolute terms and after Medicare compensation (Axelrod et al., 2017, 2018). These higher costs are not reflected in the performance evaluations of transplant centers.³⁶

Moreover, until July 2023, the Organ Procurement and Transplantation Network (OPTN) evaluated transplant centers based solely on 1-year post-transplant risk-adjusted survival of transplanted candidates. This narrow metric has faced strong criticism for encouraging centers to avoid high-risk transplants.³⁷

Institutional incentives may also reinforce each other. If transplant centers are reluctant to accept marginal kidneys, Organ Procurement Organizations (OPOs) – which retrieve and distribute organs – face lower incentives to recover these kidneys. As discussed by Chan and Roth (2024) and Kimberly et al. (2018), this feedback loop further reduces kidney availability for lower-health candidates.

Even beyond institutional constraints, transplant centers may be optimizing a utilitarian metric such as life-years-from-transplant (LYFT), which tends to favor healthier candidates (Agarwal et al., 2020).

³⁶See Appendix I for discussion of the risk-adjusted evaluation model and its potential limitations.

³⁷See Chan and Roth (2024) and Chandraker et al. (2019) for extensive critiques of the 1-year survival metric. Empirically, Stith and Hirth (2016) documents a decline in transplants to sicker patients following its implementation.

5.4.2 Candidate Behavior in Response to Higher Transplant Rates

A second explanation for selection effects relates to differential candidate behavior. As discussed in Appendix J, if candidates face a higher kidney offer rate, lower-health individuals may become more selective in accepting transplants, particularly if they are more likely to be offered sub-optimal kidneys.³⁸ In contrast, higher-health individuals may be more willing to accept early offers. This behavioral asymmetry results in relatively more high-health candidates being transplanted early in high-transplant-rate regimes (e.g., blood type AB), and relatively more low-health candidates remaining on the waitlist.

5.4.3 Negative Perceived Effect of Transplant

We can also formulate a behavioral, agent-based argument in support of regime effects on pre-transplant survival (Mechanism 1), which may operate alongside the selection mechanisms discussed above. Specifically, transplants may be perceived as high-risk by both patients and providers. Such perceptions could explain the pre-transplant survival patterns observed in Figure 1d.

To illustrate the logic, we simulate a standard dynamic discrete choice search model (Mortensen, 1986), augmented to include transplant effects. The model and results are discussed in Online Appendix G.³⁹ Figure 5b shows that the empirical pattern of pre-transplant survival cannot be replicated when transplants reduce the risk of life-threatening shocks. However, when transplants are modeled as increasing this risk, the simulated pre-transplant survival patterns match those observed in the data.

5.4.4 Weighing the Evidence Across Mechanisms

Ultimately, we cannot fully rule out any of the proposed mechanisms. However, the results in Section 5.2 provide strong evidence in favor of selection effects driven by transplant center incentives, while alternative explanations remain largely speculative. In particular, both the regime-based pre-transplant survival mechanism and the hypothesis that candidates endogenously select their treatment timing rely on tenuous assumptions about information acquisition and decision-making. To explain why differences in the

³⁸Candidates must be informed by law if they are being offered certain sub-optimal kidneys ([OPTN Final rule 121.11\(b\)\(iV\)](#)). Because the new kidney allocation system after 2014 prioritizes longer potential life candidates, unhealthier candidates are more likely to be offered, and therefore informed, of these sub-optimal kidneys. If we assume that being informed about an offer of a sub-optimal kidney leads to higher rejection independent of underlying health and offered kidney quality, then we would expect a higher rejection rate from low-health individuals. While this argument seems like a strong explanation for the results, it is worth recalling that pre-transplant differences in survival are already present prior to the implementation of the aforementioned policy in 2014.

³⁹Briefly, the agent (candidate or provider) maximizes expected survival and accepts a transplant if its quality exceeds a candidate-specific reservation threshold. Candidates exit the model upon receiving a severe health shock. We further assume the reservation threshold is invariant to the offer rate, ruling out Mechanism 2. Under these assumptions, survival patterns consistent with our data only emerge if the transplant is (perceived as) risky.

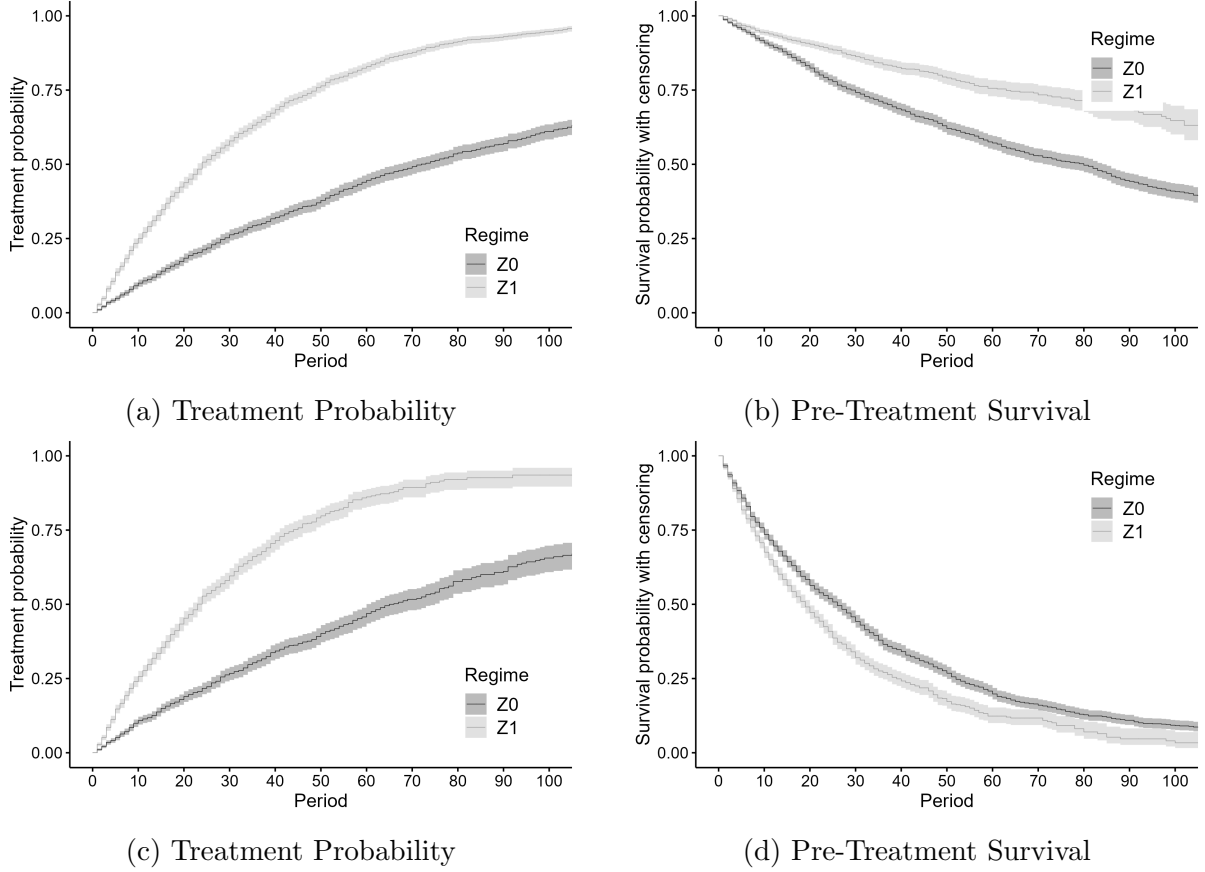


Figure 5: Simulations with (a-b) positive and (c-d) negative treatment effects
Data generating process and simulation details presented in Appendix G. Observations (a-b): $N_{Z=1:no-Tr} = 1657$, $N_{Z=1:Tr} = 822$, $N_{Z=0:no-Tr} = 738$, $N_{Z=0:Tr} = 1783$. Observations (c-d): $N_{Z=1:no-Tr} = 1919$, $N_{Z=1:Tr} = 560$, $N_{Z=0:no-Tr} = 1488$, $N_{Z=0:Tr} = 1033$.

health of transplanted candidates emerge within the first three months on the waitlist, but not thereafter, one must assume that candidates immediately perceive and respond to differences in transplant rates. This would require candidates to form and act on precise beliefs about offer rates and survival risks early and sharply in their waitlist experience, but not beyond three months.

6 Conclusion

This paper proposed a set of steps to evaluate pre-treatment regime and selection effects in a particular dynamic causal setting. While the application here focuses on the U.S. kidney transplant system, the proposed procedure is broadly applicable to waitlist environments in health, education, labor, and finance.

Leveraging variation in transplant rates induced by blood-type compatibility, we document substantial pre-transplant survival differences consistent with selection effects. These findings suggest that increases in transplant rates do not uniformly expand access across the health distribution of candidates. Instead, they disproportionately benefit higher-health individuals, leading to a positive selection of the transplanted pool. Despite

this, we find only modest differences in overall survival between groups with higher and lower transplant rates, a result likely driven by limited life-year gains from transplant among both the healthiest and least healthy candidates.

Our results have implications for recent policy reforms aimed at reducing organ discard rates and expanding access for lower-health candidates. Since July 2023, transplant center performance metrics have incorporated a risk-adjusted offer-to-acceptance score, and as of July 2024, an additional score based on pre-transplant mortality rates among waitlisted candidates. These changes aim to reduce waste and incentivize broader access to transplantation, especially for sicker patients.⁴⁰

If transplant centers are the principal agents behind the observed selection patterns, these policy changes may have unintended consequences. In particular, shifting evaluation criteria toward offer acceptance and waitlist mortality could induce centers to modify their selection at the point of waitlist entry – screening out higher-risk patients to maintain favorable metrics. The extent of such strategic behavior will depend on the balance of financial incentives tied to waitlist management, transplantation activity, and reputational performance. As such, future research and policy oversight should account for how performance metrics interact with institutional selection dynamics in multi-agent environments like transplantation.

⁴⁰See discussion by the Membership and Professional Standards Committee [here](#) and formal information [here](#).

References

- Abbring, J. H. and Van den Berg, G. J. (2005). Social experiments and instrumental variables with duration outcomes. *Tinbergen Institute Discussion paper no. TI 05-047/3*.
- Abegaz, S. B. (2021). Human abo blood groups and their associations with different diseases. *BioMed research international*, 2021:1–9.
- Agarwal, N., Ashlagi, I., Azevedo, E., Featherstone, C. R., and Karaduman, Ö. (2019). Market failure in kidney exchange. *American Economic Review*, 109(11):4026–4070.
- Agarwal, N., Ashlagi, I., Rees, M. A., Somaini, P., and Waldinger, D. (2021). Equilibrium allocations under alternative waitlist designs: Evidence from deceased donor kidneys. *Econometrica*, 89(1):37–76.
- Agarwal, N., Ashlagi, I., Somaini, P., and Waldinger, D. (2018). Dynamic incentives in wait list mechanisms. In *AEA: Papers and Proceedings*, volume 108, pages 341–347.
- Agarwal, N., Hodgson, C., and Somaini, P. (2020). Choices and outcomes in assignment mechanisms: The allocation of deceased donor kidneys. Technical report, National Bureau of Economic Research.
- Aubert, O., Reese, P. P., Audry, B., Bouatou, Y., Raynaud, M., Viglietti, D., Legendre, C., Glotz, D., Empana, J.-P., Jouven, X., et al. (2019). Disparities in acceptance of deceased donor kidneys between the united states and france and estimated effects of increased us acceptance. *JAMA Internal Medicine*, 179(10):1365–1374.
- Axelrod, D., Schnitzler, M. A., Xiao, H., Naik, A. S., Segev, D. L., Dharnidharka, V. R., Brennan, D. C., and Lentine, K. L. (2017). The changing financial landscape of renal transplant practice: a national cohort analysis. *American Journal of Transplantation*, 17(2):377–389.
- Axelrod, D. A., Schnitzler, M. A., Xiao, H., Irish, W., Tuttle-Newhall, E., Chang, S.-H., Kasiske, B. L., Alhamad, T., and Lentine, K. L. (2018). An economic assessment of contemporary kidney transplant practice. *American Journal of Transplantation*, 18(5):1168–1176.
- Bernstein, L. (2023). Troubled u.s. organ transplant system targeted for overhaul. *Washington Post*.
- Chan, A. and Roth, A. E. (2024). Regulation of organ transplantation and procurement: A market-design lab experiment. *Journal of Political Economy*, 132(11):3827–3866.

- Chandraker, A., Andreoni, K. A., Gaston, R. S., Gill, J., Locke, J. E., Mathur, A. K., Norman, D. J., Patzer, R. E., Rana, A., Ratner, L. E., et al. (2019). Time for reform in transplant program-specific reporting: Ast/asts transplant metrics taskforce. *American Journal of Transplantation*, 19(7):1888–1895.
- Chassang, S., Padró i Miquel, G., and Snowberg, E. (2012). Selective trials: A principal-agent approach to randomized controlled experiments. *American Economic Review*, 102(4):1279–1309.
- Cooling, L. (2015). Blood groups in infection and host susceptibility. *Clinical Microbiology Reviews*, 28(3):801–870.
- Danovitch, G. M. (2012). *Handbook of kidney transplantation*. Lippincott Williams & Wilkins.
- Dickert-Conlin, S., Elder, T., and Teltser, K. (2019). Allocating scarce organs: How a change in supply affects transplant waiting lists and transplant recipients. *American Economic Journal: Applied Economics*, 11(4):210–239.
- Gordon, E. J., Knopf, E., Phillips, C., Mussell, A., Lee, J., Veatch, R. M., Abt, P., Dunn, S., and Reese, P. P. (2020). Transplant candidates’ perceptions of informed consent for accepting deceased donor organs subjected to intervention research and for participating in posttransplant research. *American Journal of Transplantation*, 20(2):474–492.
- Israni, A. K., Salkowski, N., Gustafson, S., Snyder, J. J., Friedewald, J. J., Formica, R. N., Wang, X., Shteyn, E., Cherikh, W., Stewart, D., et al. (2014). New national allocation policy for deceased donor kidneys in the united states and possible effect on patient outcomes. *Journal of the American Society of Nephrology: JASN*, 25(8):1842.
- Kastoryano, S. and van der Klaauw, B. (2022). Dynamic evaluation of job search assistance. *Journal of Applied Econometrics*, 37(2):227–241.
- Kim, J. and Li, M. (2022). Optimal organ allocation policy under blood-type barriers with the donor-priority rule. *Theoretical Economics*, 17(1):331–369.
- Kimberly, K., Bernstein, L., and Keating, L. (2018). Lives lost, organs wasted. *Washington Post*.
- Lentine, K. L., Smith, J. M., Miller, J. M., Bradbrook, K., Larkin, L., Weiss, S., Handarova, D. K., Temple, K., Israni, A. K., and Snyder, J. J. (2023). Optn/srtr 2021 annual data report: kidney. *American Journal of Transplantation*, 23(2):S21–S120.
- Massie, A. B., Kuricka, L., and Segev, D. L. (2014). Big data in organ transplantation: registries and administrative claims. *American Journal of Transplantation*, 14(8):1723–1730.

- Mortensen, D. T. (1986). Job search and labor market analysis. *Handbook of Labor Economics*, 2:849–919.
- Potluri, V. S. and Bloom, R. D. (2022). Effect of policy on geographic inequities in kidney transplantation. *American Journal of Kidney Diseases*, 79(6):897–900.
- Rasmussen, S., Thomas, A., Garonzik-Wang, J., Henderson, M., Stith, S., Segev, D., and Nicholas, L. H. (2018). Reported effects of the srtr 5-tier rating system on us transplant centers. In *American Journal of Transplantation*, volume 18, pages 749–750.
- Robins, J. (1986). A new approach to causal inference in mortality studies with a sustained exposure period—application to control of the healthy worker survivor effect. *Mathematical Modelling*, 7(9-12):1393–1512.
- Rosenzweig, M. R. and Wolpin, K. I. (2000). Natural “natural experiments” in economics. *Journal of Economic Literature*, 38(4):827–874.
- Roth, A. E., Sönmez, T., and Ünver, M. U. (2004). Kidney exchange. *The Quarterly Journal of Economics*, 119(2):457–488.
- Smith, J., Biggins, S., Haselby, D., Kim, W., Wedd, J., Lamb, K., Thompson, B., Segev, D., Gustafson, S., Kandaswamy, R., et al. (2012). Kidney, pancreas and liver allocation and distribution in the united states. *American Journal of Transplantation*, 12(12):3191–3212.
- Sönmez, T., Ünver, M. U., and Yenmez, M. B. (2020). Incentivized kidney exchange. *American Economic Review*, 110(7):2198–2224.
- Stith, S. S. and Hirth, R. A. (2016). The effect of performance standards on health care provider behavior: evidence from kidney transplantation. *Journal of Economics & Management Strategy*, 25(4):789–825.
- Sun, W., Wen, C.-P., Lin, J., Wen, C., Pu, X., Huang, M., Tsai, M. K., Tsao, C. K., Wu, X., and Chow, W.-H. (2015). Abo blood types and cancer risk—a cohort study of 339,432 subjects in taiwan. *Cancer Epidemiology*, 39(2):150–156.
- Teltser, K. F. (2019). Do kidney exchanges improve patient outcomes? *American Economic Journal: Economic Policy*, 11(3):427–453.
- Wey, A., Salkowski, N., Kasiske, B., Israni, A., and Snyder, J. (2017). Program-specific offer acceptance behavior for kidney programs across the spectrum of kdpi. In *American Journal of Transplantation*, volume 17, pages 440–440.
- Wolfe, R. A., McCullough, K. P., Schaubel, D. E., Kalbfleisch, J. D., Murray, S., Stegall, M. D., and Leichtman, A. B. (2008). Calculating life years from transplant (lyft): methods for kidney and kidney-pancreas candidates. *American Journal of Transplantation*, 8(4):997–1011.

- Wu, O., Bayoumi, N., Vickers, M., and Clark, P. (2008). Abo (h) blood groups and vascular disease: a systematic review and meta-analysis. *Journal of Thrombosis and Haemostasis*, 6(1):62–69.
- Zhang, J. (2010). The sound of silence: Observational learning in the us kidney market. *Marketing Science*, 29(2):315–335.

APPENDIX

A Data selection and description

Main analysis data selection:

The initial data contains 1,010,051 observations. I remove candidates with no activation date (954,406), and select only people set to receive a kidney transplant (894,372) removing people who are not receiving their first kidney transplant (663,826). I then select for the initial analysis only candidates entering the waitlist between June 1st 2002 and December 1st 2014 (462,107). Among these, I only keep candidates who are over 18 (447,783). I further remove candidates with unusual A1, A2, A1B, A2B blood types (443,047). In the AB vs. O blood types analysis, I also remove the A and B blood types (162,500). I drop candidates with a positive cPRA (153,290) and drop candidates who received a transplant from a living donor (129,722).

Variable definition in descriptive statistics Table [A1](#):

All candidate characteristics *White*: Race-White, *Asian*: Race-Asian, *Black*: Race-Black, *Other*: Race-Other Non-White, *Age*: Candidate age at listing, *Prev_{trans}*: Received previous transplant (other than kidney transplant), *Diabetes*: Had Diabetes upon entering the waitlist, *Dialysis*: On Dialysis upon entering the waitlist, *Dialysis_{time}*: Time on Dialysis upon entering the waitlist, *Diag_{poly}*: primary diagnoses: polycystic, *Diag_{hype}*: primary diagnoses: hypertension, *Diag_{glom}*: primary diagnoses: glomerular, *Diag_{other}*: primary diagnoses: other *Female*: Female, *BMI*: BMI, , *Prev_{malig}*: Any previous Malignancy, *Acpt_{HepB}*: Will accept an Hepatitis B Core Antibody Positive Donor?, *Acpt_{HCV+}*: Will accept an HCV Positive donor?, *Functional*: three point constructed functional ability scale with 1 lowest and three highest, *Educ_{HSless}*: Attained high school or less, *Educ_{COLsome}*: obtained some college education, *Educ_{COLdeg}*: obtained a college degree. Donor and match characteristics: *Mismatch_A*: HLA-A mismatches, *Mismatch_B*: HLA-B mismatches, *Mismatch_{DR}*: HLA-DR mismatches, *donor_{share}*: donor part of shared organ scheme, *donor_{age}*: donor age, *donor_{weight}*: donor weight, *donor_{anox}*: cause of death: anoxia, *donor_{cere}*: cause of death: cerebrovascular accident, *donor_{head}*: cause of death: head trauma, *donor_{tumo}*: cause of death: central nervous system tumor, *donor_{other}*: cause of death: other/unknown.

B Descriptive Statistics

Table A1: Descriptive Statistics

	Pre-reform				Post-reform		White cand.		Wh. tr. mths 1-3	
	<i>O</i>	<i>AB</i>	<i>B</i>	<i>A</i>	<i>O</i>	<i>AB</i>	<i>O</i>	<i>AB</i>	<i>O</i>	<i>AB</i>
<i>White</i>	0.62 (0.49)	0.54 (0.5)	0.44 (0.5)	0.7 (0.46)	0.66 (0.47)	0.58 (0.49)				
<i>Asian</i>	0.05 (0.22)	0.12 (0.32)	0.13 (0.33)	0.05 (0.22)	0.07 (0.25)	0.14 (0.35)				
<i>Black</i>	0.3 (0.46)	0.33 (0.47)	0.41 (0.49)	0.23 (0.42)	0.25 (0.43)	0.26 (0.44)				
<i>Other</i>	0.02 (0.15)	0.01 (0.12)	0.02 (0.12)	0.02 (0.14)	0.02 (0.16)	0.02 (0.15)				
<i>Age</i>	51.93 (12.99)	52.49 (12.97)	52.09 (12.77)	52.58 (12.96)	53.08 (13.28)	53.9 (12.91)	52.62 (13.18)	53.48 (13.11)	49.77 (14.2)	51.96 (13.85)
<i>Prevtrans</i>	0.02 (0.13)	0.02 (0.14)	0.02 (0.12)	0.02 (0.15)	0.02 (0.15)	0.03 (0.18)	0.02 (0.15)	0.03 (0.17)	0.03 (0.18)	0.04 (0.19)
<i>Diabetes</i>	0.37 (0.48)	0.39 (0.49)	0.38 (0.49)	0.37 (0.48)	0.41 (0.49)	0.42 (0.49)	0.37 (0.48)	0.36 (0.48)	0.25 (0.43)	0.31 (0.46)
<i>Dialysis</i>	0.75 (0.43)	0.72 (0.45)	0.76 (0.43)	0.72 (0.45)	0.57 (0.5)	0.54 (0.5)	0.71 (0.46)	0.65 (0.48)	0.62 (0.48)	0.63 (0.48)
<i>Dialysis_{time}</i>	290.42 (649.8)	317.55 (608.62)	295.97 (647.79)	285.49 (610.38)	0* (0)	0* (0)	250.09 (587.17)	254.53 (493.71)	286.5 (664.07)	311.03 (549.49)
<i>Diag_{poly}</i>	0.07 (0.25)	0.07 (0.26)	0.06 (0.23)	0.08 (0.27)	0.08 (0.27)	0.08 (0.26)	0.09 (0.29)	0.1 (0.31)	0.09 (0.29)	0.11 (0.31)
<i>Diag_{hype}</i>	0.23 (0.42)	0.22 (0.42)	0.25 (0.43)	0.2 (0.4)	0.19 (0.39)	0.18 (0.39)	0.16 (0.37)	0.15 (0.36)	0.13 (0.33)	0.14 (0.34)
<i>Diag_{glom}</i>	0.1 (0.29)	0.1 (0.3)	0.1 (0.3)	0.1 (0.3)	0.11 (0.32)	0.13 (0.33)	0.1 (0.3)	0.1 (0.31)	0.15 (0.36)	0.13 (0.34)
<i>Diag_{other}</i>	0.61 (0.49)	0.61 (0.49)	0.6 (0.49)	0.62 (0.49)	0.62 (0.49)	0.62 (0.49)	0.64 (0.48)	0.64 (0.48)	0.63 (0.48)	0.62 (0.48)
<i>Female</i>	0.38 (0.49)	0.37 (0.48)	0.38 (0.48)	0.37 (0.48)	0.36 (0.48)	0.35 (0.48)	0.36 (0.48)	0.35 (0.48)	0.37 (0.48)	0.35 (0.48)
<i>BMI</i>	28.64 (5.71)	28.61 (5.8)	28.57 (5.78)	28.7 (5.72)	29.26 (5.55)	29.06 (5.59)	28.5 (5.56)	28.64 (5.68)	27.62 (5.43)	28.05 (5.56)
<i>Prev_{malig}</i>	0.06 (0.23)	0.06 (0.24)	0.05 (0.22)	0.07 (0.25)	0.09 (0.28)	0.09 (0.29)	0.07 (0.25)	0.08 (0.27)	0.07 (0.25)	0.07 (0.25)
<i>Acpt_{HepB}</i>	0.56 (0.5)	0.55 (0.5)	0.58 (0.49)	0.54 (0.5)	0.66 (0.47)	0.64 (0.48)	0.55 (0.5)	0.52 (0.5)	0.46 (0.5)	0.45 (0.5)
<i>Acpt_{HCV+}</i>	0.06 (0.24)	0.05 (0.21)	0.06 (0.25)	0.05 (0.22)	0.29 (0.45)	0.25 (0.43)	0.05 (0.22)	0.04 (0.19)	0.07 (0.26)	0.05 (0.22)
<i>Functional</i>	2.89 (0.33)	2.9 (0.32)	2.9 (0.32)	2.89 (0.33)	2.83 (0.41)	2.84 (0.4)	2.89 (0.34)	2.89 (0.33)	2.81 (0.51)	2.85 (0.43)
<i>Educ_{HSless}</i>	0.55 (0.5)	0.49 (0.5)	0.51 (0.5)	0.52 (0.5)	0.44 (0.5)	0.39 (0.49)	0.56 (0.5)	0.49 (0.5)	0.47 (0.5)	0.47 (0.5)
<i>Educ_{COLsome}</i>	0.24 (0.43)	0.26 (0.44)	0.25 (0.44)	0.25 (0.43)	0.26 (0.44)	0.26 (0.44)	0.22 (0.42)	0.24 (0.43)	0.25 (0.43)	0.24 (0.43)
<i>Educ_{COLdeg}</i>	0.21 (0.41)	0.25 (0.43)	0.23 (0.42)	0.23 (0.42)	0.3 (0.46)	0.35 (0.48)	0.22 (0.41)	0.27 (0.44)	0.28 (0.45)	0.29 (0.46)
							if ever treated			
<i>Mismatch_A</i>							1.18 (0.73)	1.24 (0.71)	0.99 (0.73)	1.14 (0.72)
<i>Mismatch_B</i>							1.38 (0.72)	1.47 (0.68)	1.17 (0.74)	1.34 (0.71)
<i>Mismatch_{DR}</i>							1.08 (0.72)	1.18 (0.7)	1 (0.73)	1.11 (0.73)
<i>donor_{share}</i>							0.54 (0.5)	0.5 (0.5)	0.77 (0.42)	0.6 (0.49)
<i>donor_{age}</i>							40.39 (14.95)	39.12 (14.98)	39.61 (13.53)	39.21 (14.31)
<i>donor_{weight}</i>							78.75 (21.64)	78.49 (21.87)	77.45 (18.05)	78.27 (20.63)
<i>donor_{anox}</i>							0.29 (0.45)	0.27 (0.44)	0.19 (0.39)	0.21 (0.41)
<i>donor_{cere}</i>							0.33 (0.47)	0.31 (0.46)	0.34 (0.47)	0.33 (0.47)
<i>donor_{head}</i>							0.34 (0.48)	0.39 (0.49)	0.44 (0.5)	0.42 (0.49)
<i>donor_{tumo}</i>							0 (0.07)	0 (0.06)	0.01 (0.07)	0.01 (0.07)
<i>donor_{other}</i>							0.03 (0.16)	0.03 (0.16)	0.02 (0.14)	0.03 (0.17)
N	142026	11264	42672	93347	58500	4750	88220	6099	6193	937

Final two columns are White candidates and White candidates transplanted in first three months on the waitlist. Means for each group presented with standard deviations in parenthesis. Normalized differences relative to the O blood-type group greater or equal to 0.1 are marked in bold. Variable abbreviations described in Appendix section A. *Post-reform data on *Dialysis_{time}* only lists the calendar time a person went on dialysis after entering the waitlist.

C Additional results: dynamic treatment effect analyses

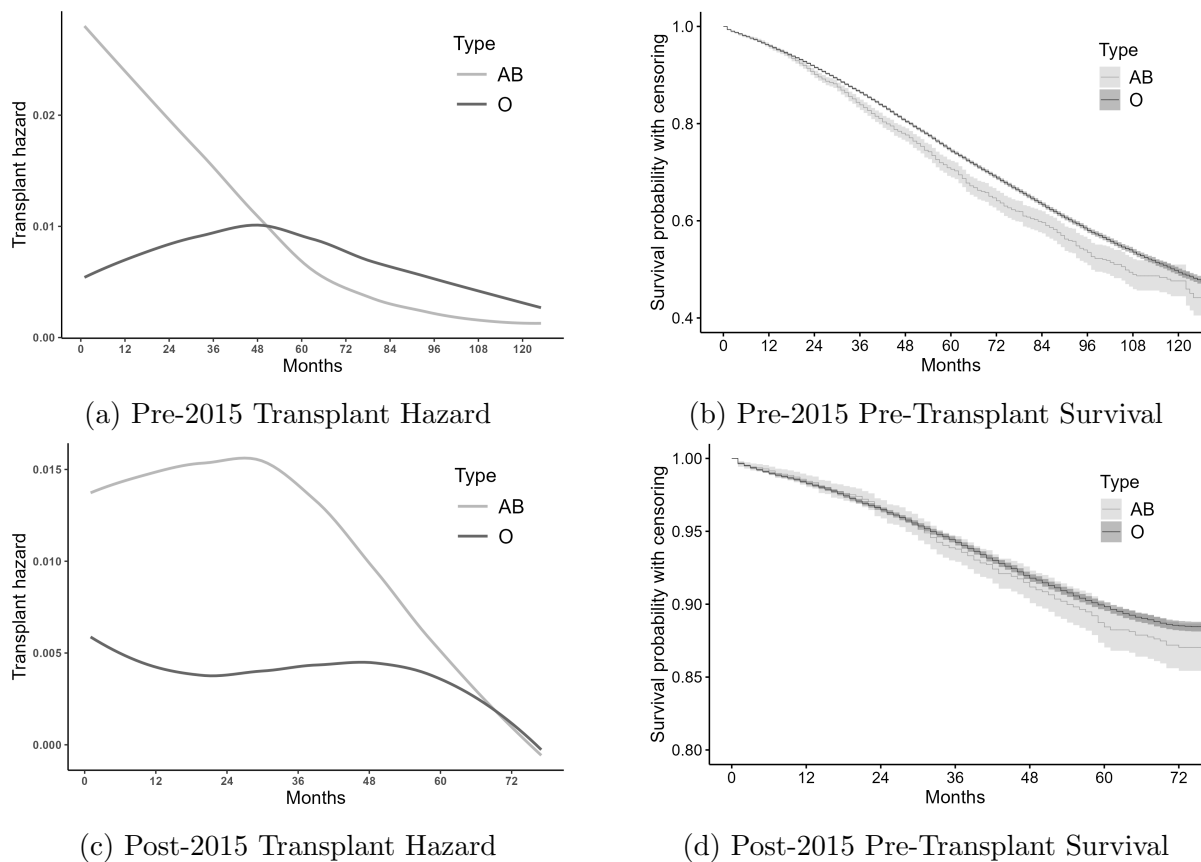
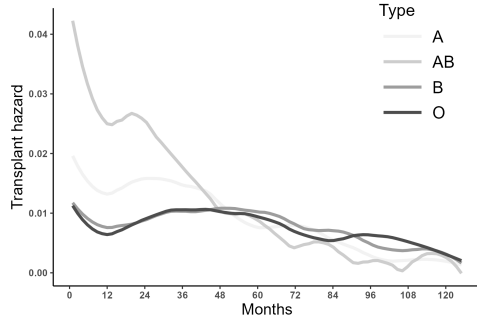


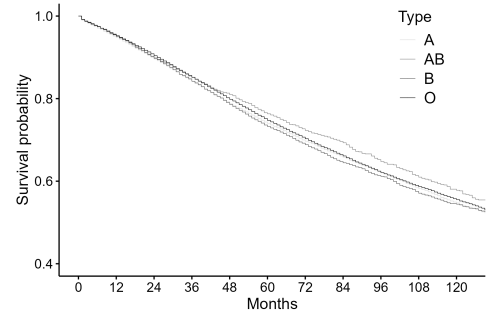
Figure A1: Survival of candidates on the kidney transplant waitlist

Includes candidates entering waitlist after 01 June 2002. Based on selected sample of Scientific Registry of Transplant Recipients data described in Appendix A.

Observations in pre-reform sample (top frames): $N_O = 120,302$, $N_{AB} = 9420$. Observations in post-reform sample (bottom frames): $N_O = 50,126$, $N_{AB} = 3970$.



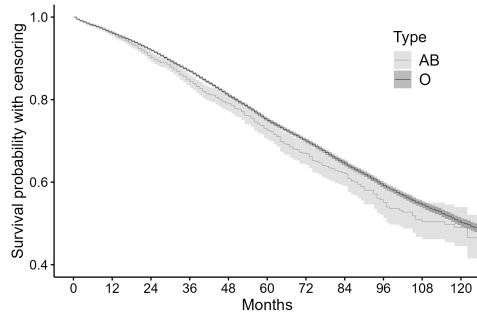
(a) Hazard rate for all blood types



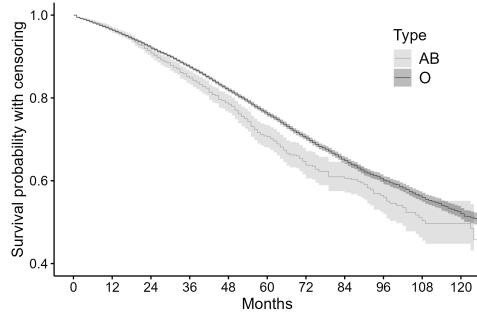
(b) Survival for all blood types

Figure A2: Hazard and overall survival for different blood types

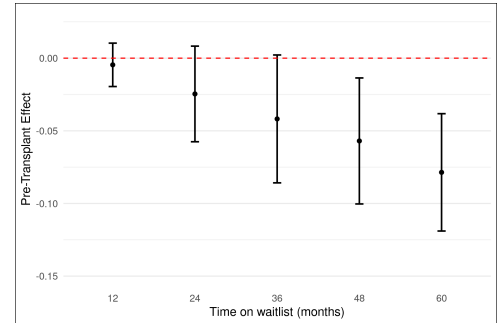
Includes candidates entering waitlist after 01 June 2002, durations truncated at 01 December 2014. Based on selected sample of Scientific Registry of Transplant Recipients data described in Appendix A. Observations: $N_O = 71,075$, $N_{AB} = 4764$, $N_B = 14,887$, $N_A = 50,149$.



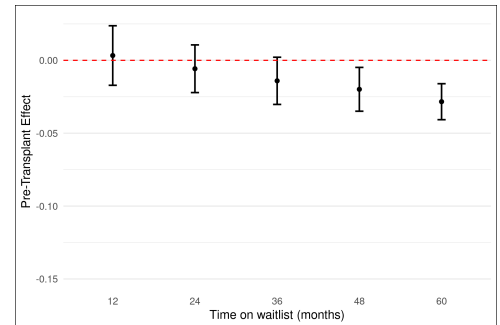
(a) Survival for HS degree or less



(c) Survival for at least some college

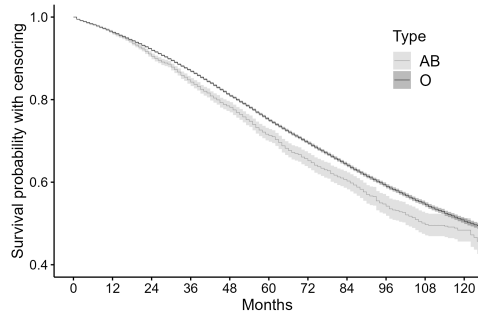


(b) Effects for HS degree or less

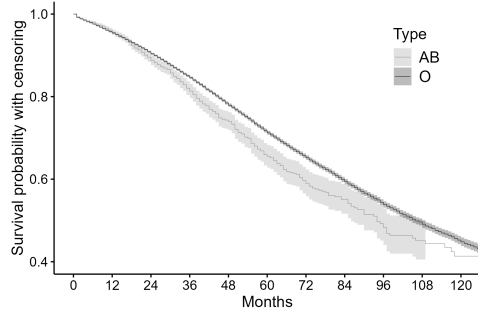


(d) Effect for at least some college

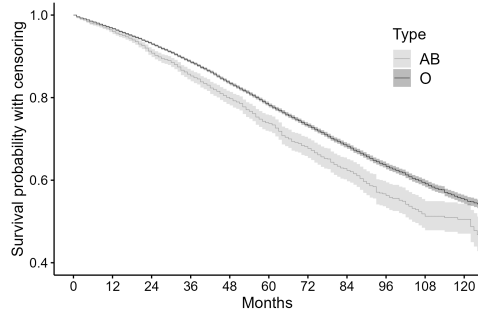
Figure A3: Pre-transplant survival and effect $\beta_{\tau, z', z, x}$ for different subsamples. Includes candidates entering waitlist after 01 June 2002, durations truncated at 01 December 2014. Based on selected sample of Scientific Registry of Transplant Recipients data described in Appendix A.



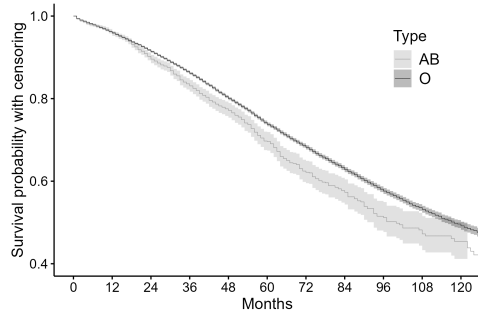
(a) Survival including living donors



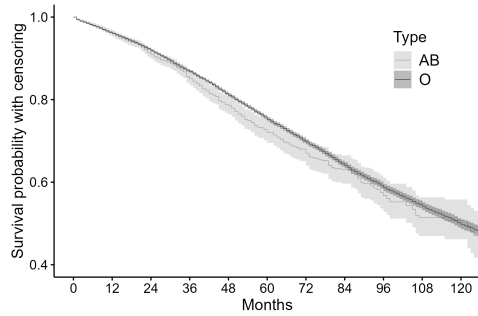
(c) Survival for white people



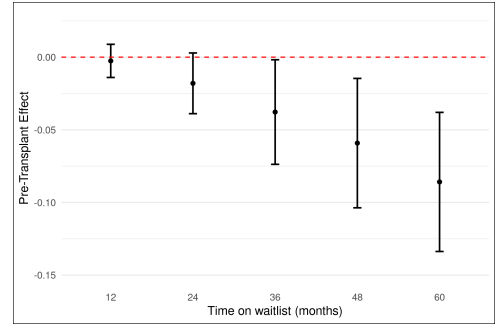
(e) Effects for non-white people



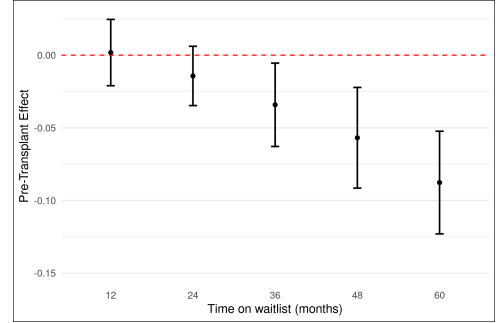
(g) Survival for males



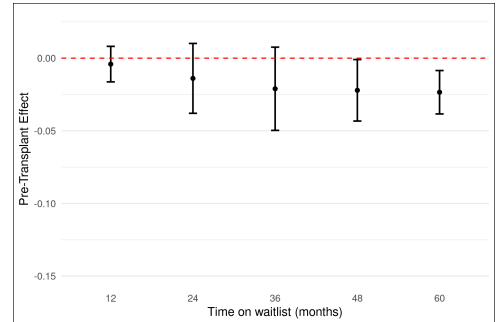
(i) Survival for females



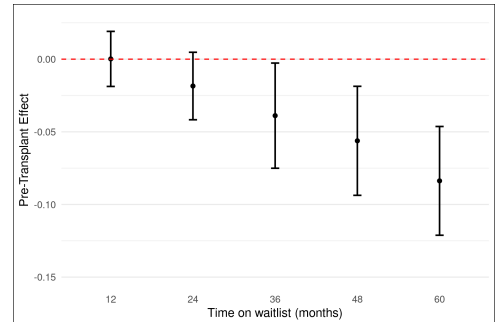
(b) Effects including living donors



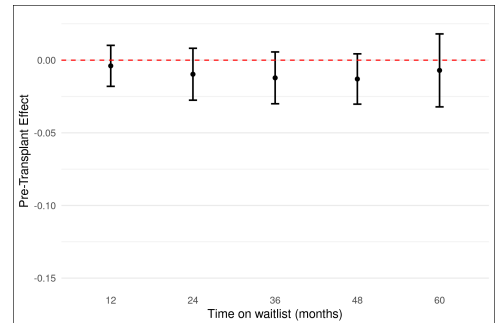
(d) Effects for white people



(f) Survival for non-white people



(h) Effects for males



(j) Effects for females

Figure A4: Pre-transplant survival and effect $\beta_{\tau, z', z, x}$ for different subsamples. Includes candidates entering waitlist after 01 June 2002, durations truncated at 01 December 2014. Based on selected sample of Scientific Registry of Transplant Recipients data described in Appendix A.

D Dynamic treatment effect non-parametric and machine learning robustness checks

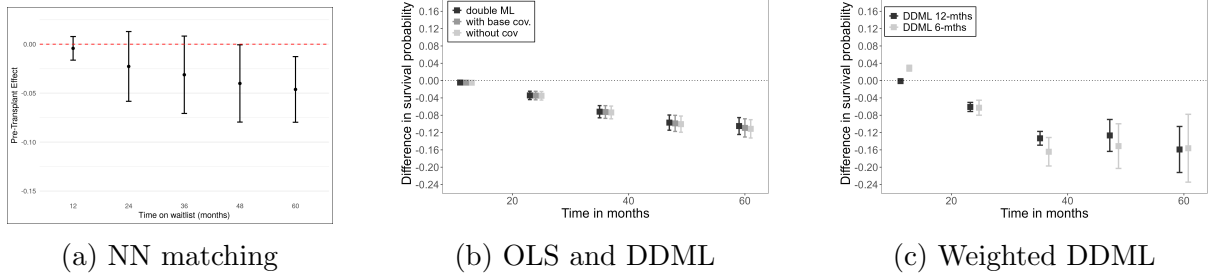


Figure A5: NN matching and Double/debiased ML (without and with IPCW).

Includes candidates entering waitlist between 01 June 2002 and 01 December 2014. Based on selected sample of Scientific Registry of Transplant Recipients data described in Appendix A. NN matching applies an exact match on race, sex, diabetes at entry, and dialysis at entry. It then applies a propensity score match on all other candidate variables listed in A1. OLS and Double/debiased ML estimators in middle figure are unweighted and censor all observations treated before τ . Double/debiased ML estimators censor all observations treated before τ but are weighted by an inverse probability of censoring weighting function generated by neural nets to account for the random right censoring of the outcome and censored outcomes at the moment of transplant (competing risk censoring). Double/debiased ML estimates are sensitive to the time intervals over which these weights are constructed – 3 month intervals produced erratic results. Standard errors are not adjusted for the estimation of the weights.

Matching observations: $N_O = 9420$, $N_{AB} = 9420$; Double/debiased ML of survival on $\mathbf{1}(\text{blood} = AB)$ depend on the number of censored observations at the moment of transplant, observations: $N_O < 120,302$, $N_{AB} < 9420$.

Table A2: Estimates by Specification

Time (mths)	No Covariates	Cox-Lasso	Deep Survival	Rdm. Forest Survival
12	-0.005 (0.003)	-0.854 (3.248)	-0.743 (0.000)	-46.096 (0.007)
24	-0.035 (0.005)	-0.795 (3.233)	-0.734 (0.001)	-51.586 (0.005)
36	-0.074 (0.008)	-0.744 (3.203)	-0.715 (0.001)	-56.599 (0.004)
48	-0.101 (0.010)	-0.720 (3.158)	-0.684 (0.001)	-60.211 (0.005)
60	-0.111 (0.011)	-0.726 (3.100)	-0.635 (0.001)	-61.798 (0.007)
72	-0.104 (0.011)	-0.754 (3.035)	-0.580 (0.001)	-61.173 (0.009)
84	-0.091 (0.011)	-0.760 (2.975)	-0.528 (0.001)	-58.865 (0.010)
96	-0.069 (0.010)	-0.773 (2.913)	-0.559 (0.001)	-55.140 (0.011)
108	-0.042 (0.009)	-0.800 (2.853)	-0.423 (0.001)	-50.807 (0.010)
120	-0.021 (0.007)	-0.810 (2.797)	-0.382 (0.002)	-46.351 (0.009)

Includes candidates entering waitlist between 01 June 2002 and 01 December 2014. Estimations of survival on $\mathbf{1}(\text{blood} = AB)$ after censoring survival at the moment of transplant. All candidate health, demographic and location variables included for ML estimators. Based on selected sample of Scientific Registry of Transplant Recipients data described in Appendix A. ML of survival on $\mathbf{1}(\text{blood} = AB)$ depend on the number of censored observations at the moment of transplant observations: $N_O < 120,302$, $N_{AB} < 9420$.

E Additional Results: effects on transplanted and overall

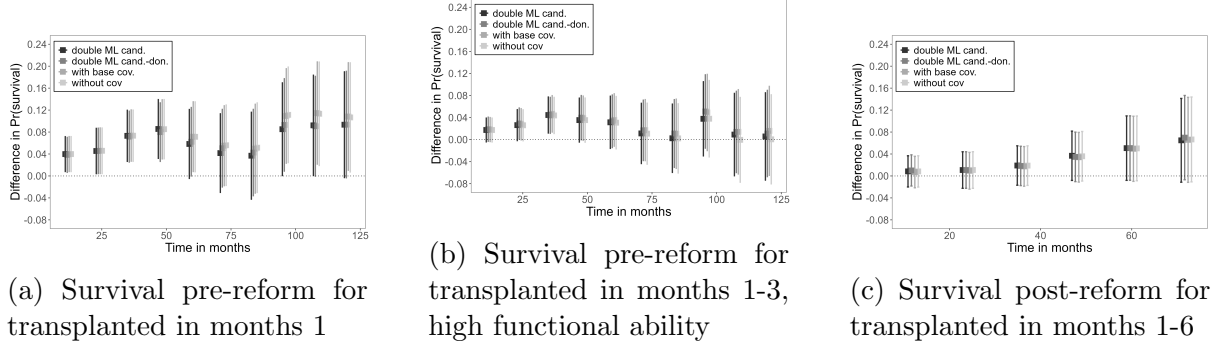
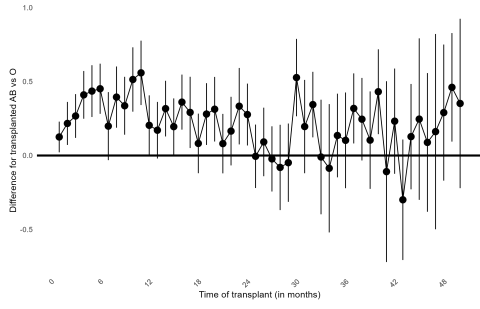


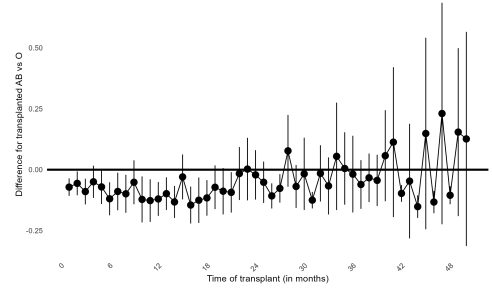
Figure A6: Difference in survival between AB and O blood types over time

Includes candidates entering waitlist after 01 June 2002, durations truncated at 01 December 2014. Based on selected sample of Scientific Registry of Transplant Recipients data described in Appendix A. Window of 6 months used in post-reform analysis to increase sample size.

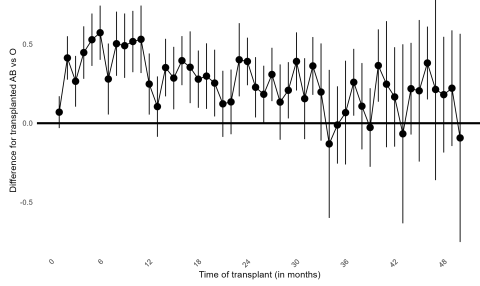
(*without cov*) includes no covariates, (*with base cov*) includes offer covariates: age bracket dummies, (*double/debiased ML cand*) includes all candidate variables presented in Appendix Table A1, (*double/debiased ML cand-don*) includes all candidate, donor, and candidate-donor match variables presented in Appendix Table A1. Observations (left): $N_{O:treated} = 1402$, $N_{AB:treated} = 289$. Observations (middle): $N_{O:treated} = 1560$, $N_{AB:treated} = 454$. Observations (right): $N_{O:treated} = 1283$, $N_{AB:treated} = 236$.



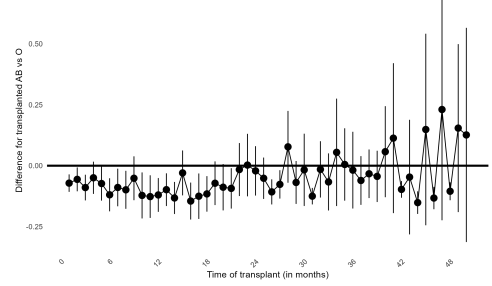
(a) Mismatch HLA-A



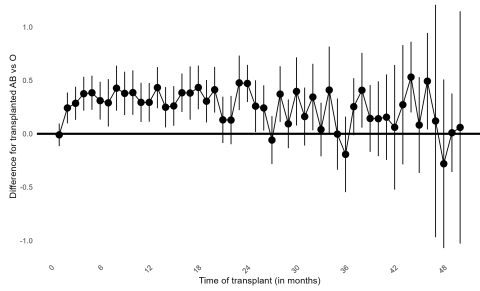
(b) Mismatch HLA-A missing



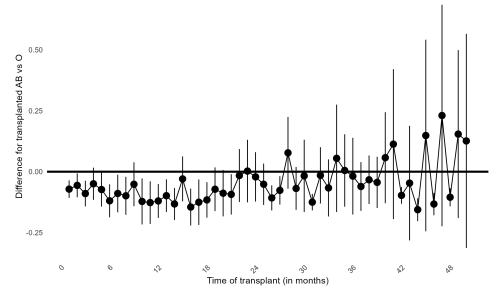
(c) Mismatch HLA-B



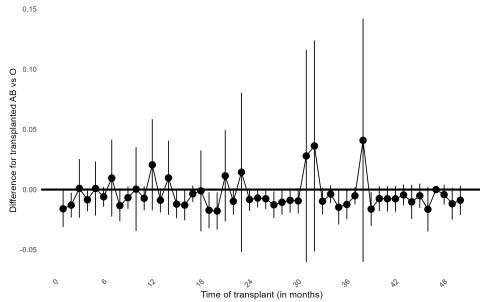
(d) Mismatch HLA-B missing



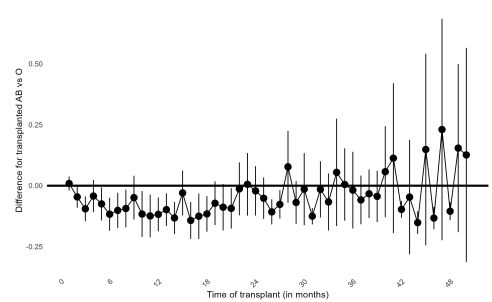
(e) Mismatch HLA-DR



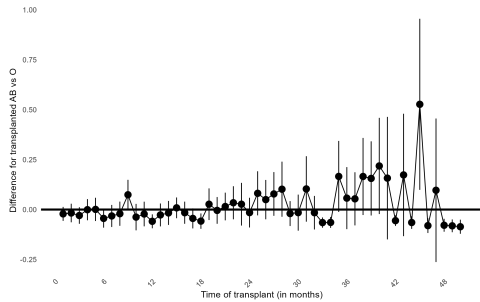
(f) Mismatch HLA-DR missing



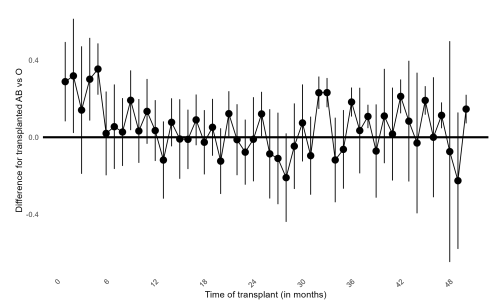
(g) Graft failure



(h) Graft failure missing



(i) Candidate functional ability pre-reform missing



(j) Candidate functional ability post-reform

Figure A7: Difference in observed characteristics of transplanted. Results from a linear regression of characteristic on the blood the indicator, with the indicator equal to one for AB blood types.

F Regime effects on the treatment timing: simple example

Here we consider in a simple example how the patterns of Figure 1d can arise when the regime affects the treatment timing in a one period model. For each blood type, there are 5 high unobserved health (h) and 5 low unobserved health (l) candidates. In all scenarios we assume survival is increasing in health by imposing that h-types who receive a transplant always survive while l-types only survive if they receive a transplant. In the upper panel, we consider the scenario in which the probability of accepting a kidney is higher for h-types than for l-types by assuming h-types always accept an offered kidney while l-types only accept an offered kidney with a 1/3 probability. Further assume that 6 kidneys are randomly offered in the AB-blood type regime but only 2 are offered in the O-blood type regime. In this setting, for the AB-blood type regime there will be 2 h-types and, in expectation, 4 l-types who do not receive a transplant. Of these only the h-types survive. As such, the pre-transplant survival rate will be 2/6 in the AB-blood regime. A similar calculation shows that the pre-transplant survival rate in the O-blood regime is 4/9. As such, the difference in pre-transplant survival $\beta_{\tau,z',z,x} = -1/9$ is lower in the AB-blood regime as in the empirical results.

A similar exercise is presented in the second panel in which we assume h-types and l-types have the same 1/3 probability to accept an offered kidney. In this scenario, and holding all other previous assumptions, the pre-transplant survival remains the same in the O-type and AB-type regime. In the last panel we consider the case in which h-types are assumed to have a lower probability of accepting an offered kidney than l-types. This scenario results in higher pre-transplant survival in the AB-blood regime than in the O-blood regime. We see that only the first case are compatible with the observed empirical patterns in the kidney setting and the main results.

Table A3: Survival Dynamics in Different Acceptance/Offer Scenarios

	cand.	offered kidneys	accepted kidneys	pre-transpl. survival	pre-transpl. survival diff.	$\beta_{\tau,z',z,x}$
$\partial \Pr(\text{accept kidney} U = u)/\partial u > 0$						
AB	5h	3	3	2h/2h	2/6	-1/9
	5l	3	1	0l/4l		
O	5h	1	1	4h/4h	4/9	
	5l	1	0	0l/5l		
$\partial \Pr(\text{accept kidney} U = u)/\partial u = 0$						
AB	5h	3	1	4h/4h	4/8	0
	5l	3	1	0l/4l		
O	5h	1	0	5h/5h	5/10	
	5l	1	0	0l/5l		
$\partial \Pr(\text{accept kidney} U = u)/\partial u < 0$						
AB	5h	3	1	4h/4h	4/6	1/9
	5l	3	3	0l/2l		
O	5h	1	0	5h/5h	5/9	
	5l	1	1	0l/4l		

G Dynamic discrete choice model of candidate behavior

To relate the dynamic treatment effect model to the literature on dynamic discrete choice models we consider in this appendix a standard job search model. The model is then extended to include a treatment. I loosely adapt the discussion of the search model to this paper's kidney transplant application from the point of view of a candidate, although the explanations can be slightly adapted to consider choices from the perspective of transplant centers. For expositional purposes, the model is solved under simplistic assumptions. The discussion then explains how the dynamic discrete choice model relates to the parameters in the proportional hazard model in Section 3.3. Thereafter I describe the data generating process and present simulation results for the estimator.

G.1 Standard dynamic discrete choice model

Consider an agent who enters an initial state at time $t = 0$ and assume that time is discrete. In each subsequent period the agent faces the choice of staying within this state or leaving. Whenever he is in the initial state, the agent derives utility w_0 . In the application, $t = 0$ is the moment a candidate enters the kidney transplant waitlist and each period the candidate, which we consider as a unit combined of psychological and biological factors, puts in a certain amount of effort to remain alive. w_0 represents the combined psychological and biological utility of staying alive.

Next, with probability λ the agent receives an offer to leave the state. An offer can be interpreted as a negative health shock on the body due to kidney failure. The agent also faces a cost c corresponding to the necessary biological effort to prevent a health shock, with lower costs corresponding to higher effort. Once the agent receives an offer, he has to decide immediately whether or not to accept it. An offer is characterized by its instantaneous utility w drawn from the distribution $G(w)$. Upon accepting an offer, the agent derives the same instantaneous utility w for each subsequent period. So once a candidate's biological constitution receives a health shock beyond what it can fight, they die and receive the (perceived) utility of death thereafter.

We assume the agent optimization behavior follows a dynamic discrete choice model which nests search models and optimal stopping models. The agent forms expectations over future instantaneous utilities. Denoting by ρ the discount rate, the present discounted combined psychological and biological value of remaining alive at the start of period t , $V_{0,t}$, can be described by the Bellman equation,

$$V_{0,t} = w_0 - c + \rho\lambda\mathbb{E}[\max\{V_1(w), V_{0,t+1}\}] + \rho(1 - \lambda)V_{0,t+1}$$

$V_1(w)$ is the discounted value that the agent would acquire by failing to fight off a health shock, dying as a consequence, and reaping instantaneous biological utility w . The agent

follows a stationary reservation utility strategy where w^* is the minimum offer of w required to induce the agent to exit in the following period. In terms of this study, w^* represents the utility associated to the limit at which someone's biological constitution prefers to stop functioning, rather than exert the continued effort to keep a person alive, despite the psychological desire to remain alive. For a healthy person with a well functioning immune system, we would expect w^* to be very high, since only an extreme negative health shock (large w) would induce someone's biological functions to give in. A lower w^* implies, all else equal, that a person's health is worse, making lower utilities from death w more appealing. Under a reservation utility strategy, we can reformulate the Bellman equation as

$$V_{0,t} = w_0 - c + \rho \lambda \int_{w^*}^{\infty} (V_1(w) - V_{0,t+1}) dG(w) + \rho V_{0,t+1}$$

We can augment this model to include a treatment prescribed by a regime. Let us assume that the agent knows that he has been assigned to a certain regime z which allocates future treatment. For simplicity we consider in this section a stochastic assignment regime where the agent faces the same probability π to receive treatment at each period. So each regime z is fully characterized by its value of π . This type of randomization can correspond as in the empirical setting to a randomization set by nature but may also be a rule imposed by a policymaker. In the empirical setting, it can best be interpreted as a situation in which agents receive different signals of how likely they are to receive treatment, and interpret this signal as a constant hazard π to treatment each period.

In principle, we could model π from an additional search process by specifying a search model of offered kidneys for the accepted kidney. I do not add this layer of complexity in this model.

In the application, candidates on the waitlist were randomized at birth to have an AB blood type or a B/O blood type. This blood type, while inconsequential during most peoples' lives, is an important determinant to the time a candidate must wait until receiving a kidney transplant, which is the treatment in this setting. From the point of view of a candidate, their blood type may be the most salient feature determining the duration until they receive a transplant, but is not the only factor influencing the timing of a kidney transplant. Given the many factors determining the waiting time until a match, the blood type randomization can be seen as a stochastic treatment assignment mechanism since it influences the chances of finding a kidney match but does not determine the exact date a candidate will receive the transplant. Receiving a kidney transplant would likely reduce the arrival of health shocks (λ), change the effort a candidate needs to put into their general health upkeep (search costs c), or change the distribution of the (perceived) utility from death ($G(w)$).

To simplify the exposition, assume treatment only affects the distribution of the (perceived) utility from death by prescribing a higher mean to $G^{tr}(w)$ relative to $G(w)$ for an

agent upon receiving the treatment. Allowing agents to form expectations over treatment outcomes, the alive value functions before receiving a kidney transplant, $V_{0,t}$, and after receiving a kidney transplant, $V_{0,t}^{tr}$, are given by,

$$\begin{aligned} V_{0,t} &= w_0 - c + \rho\lambda\mathbb{E}[\max\{V_1(w), (1 - \pi)V_{0,t+1} + \pi V_{0,t+1}^{tr}\}] + \rho(1 - \lambda)[(1 - \pi)V_{0,t+1} + \pi V_{0,t+1}^{tr}] \\ V_{0,t}^{tr} &= w_0 - c + \rho\lambda\mathbb{E}[\max\{V_1^{tr}(w), V_{0,t+1}^{tr}\}] + \rho(1 - \lambda)V_{0,t+1}^{tr} \end{aligned}$$

We can solve this model for the reservation utilities after and before treatment,⁴¹

$$\begin{aligned} w^*(\pi) &= \frac{(1 - \rho)(1 - \pi)}{1 - \rho + \rho\pi}(w_0 - c) + \frac{\rho\lambda(1 - \pi)}{(1 - \rho)(1 - \rho + \rho\pi)} \int_{w^*}^{\infty} (1 - G(w)) dw + \frac{\pi}{1 - \rho + \rho\pi} w^{tr*} \\ w^{tr*} &= w_0 - c + \frac{\rho\lambda}{1 - \rho} \int_{w^{tr*}}^{+\infty} (1 - G^{tr}(w)) dw \end{aligned}$$

Under the assumption that $V_{0,t}^{tr} > V_{0,t}$ one can demonstrate that the pre-treatment reservation utility $w^*(\pi)$ is increasing in π . This means that if agents foresee positive effects of receiving treatment on the w offer distribution, and are in a regime with higher chances to receive treatment, then they will remain for longer in the initial state if they are not yet treated. In terms of the empirical application, this implies that if candidates on the waitlist foresee a kidney transplant to improve their biological utility of life, and they have a high probability of rapidly receiving a kidney transplant (AB blood type), then they will exert more effort each period to stay alive before receiving the transplant. The empirical findings are not consistent with this prediction. They show that candidates with a higher probability of receiving a kidney transplant display a higher pre-transplant mortality. One way to consolidate the empirical results with the biological predictions of the model is to allow a candidate's behavior to asymmetrically influence their biological functions in response to their probability of receiving a future kidney transplant. In particular, we must assume candidates with a high probability of treatment pay less attention to their general health, leading to lower survival.

It is worth considering the interpretation of parameters in the model of subsection 3.3 under different treatment assignment mechanisms prescribed by the regime. If the regime enforces a constant treatment hazard each period and there is full compliance, then $\theta_t^S(z) = \pi^S(z)$. If in addition agents know the regime, do not vary their search strategy over time, and the expected value of future variables over intermediate shocks is constant over time, then $\lambda_t^T(z)$ will be constant. This constant is then interpreted as the effect on the exit hazard of a constant treatment hazard regime. This is the setting considered in the dynamic discrete choice search model above.

⁴¹Full derivation of solutions are presented in subsection G.4

G.2 Data Generating Process

The data generating process for the simulation data follows the dynamic discrete choice model presented in the above section with the following specifications,

$$\begin{aligned}
U_{it}^{no-exit} &= w_{0it} - c_{it} \\
U_{it}^{exit} &= w_{it} \\
c_{it} &= \beta_a^c \cdot a_i + \beta_e^c \cdot e_i \\
w_{it} &= \beta_a^w \cdot a_i + \beta_s^w \cdot I(s < t) + \xi_{it} \quad \xi_{it} \sim \mathcal{N}(0, \sigma_\xi^2) \\
w_{0it} &= 0.75 \cdot \beta_a^w \cdot a_i
\end{aligned}$$

where $U_{it}^{no-exit}$ and U_{it}^{exit} are the instantaneous utilities when the agent chooses to remain in the initial state or exit. λ_{it} follows a Poisson distribution with mean $\beta_a^\lambda \cdot a_i + \beta_e^\lambda \cdot e_i$. The treatment outcome for the group under regime $Z = 1$ is drawn each period from a binary distribution with probability $\pi_{Z=1} = Pr(S = s | S \geq s, Z = 1) = 0.03$. The regime assignment for the $Z = 0$ group is $\pi_{Z=0} = Pr(S = s | S \geq s, Z = 0) = 0.01$. I impose that the treatment takes place before the exit decision in period t .

Using this model I generate the treatment durations, exit durations, and accepted offers for a population of 5000 agents over 5000 periods. Within this population, a_i and e_i assume discrete values in the intervals $[1, 6]$ and $[1, 3]$ respectively. The initial randomization assigns half of the population to each regime $Z = 0$ or $Z = 1$. As in the discussion above, I choose to focus on a situation where the treatment affects only the offer distribution $G(w; a)$. The treatment effect is negative and is calibrated to equal one standard deviation of the offer distribution, σ_w , with $\beta_s^w = 0$. The full choice of parameters for each policy setting is presented below. To estimate the stationary solution for the accepted offer I simulate expectations of w_{it} over 1000 draws and iterate over the value function until convergence.

Table A4: Parameter choices in simulations

μ_w	13.762,	μ_c	0.893,	μ_λ	0.092,	ρ	0.995
σ_w	5.497,	σ_c	0.257,	σ_λ	0.031,	σ_ξ	3
β_a^w	4,	β_a^c	0.2,	β_a^λ	0.5/21,	$\pi_{Z=0}$	0.01
β_s^w	5.497,	β_e^c	0.1,	β_e^λ	0.1/21,	$\pi_{Z=1}$	0.03

G.3 Simulation Results and Discussion

To apply the continuous time methods under study in this paper, it is preferable to have a large dataset where the unit of time represents a relatively short period. In practice, if the unit of time is too large it may be challenging to account for dynamic selection and

for the simultaneity of treatment and exit outcomes within a period. In the estimation, I treat effort e_i as an unobserved characteristic for the researcher, and fully stratify a_i . The researcher observes individual treatment and exit duration outcomes, ability, w_{0it} , an indicator Z for the regime assignment, and an indicator if the observation is right censored. After generating the dynamic discrete choice data I censor all observations greater than $t = 60$ ($\sim 43.7\%$) and apply random right censoring to $\sim 6.3\%$ of the remaining observations. Descriptive survival curves and hazards are presented in Figure A8 in the case of a positive treatment effect and in Figure A9 in the case of a negative treatment effect.

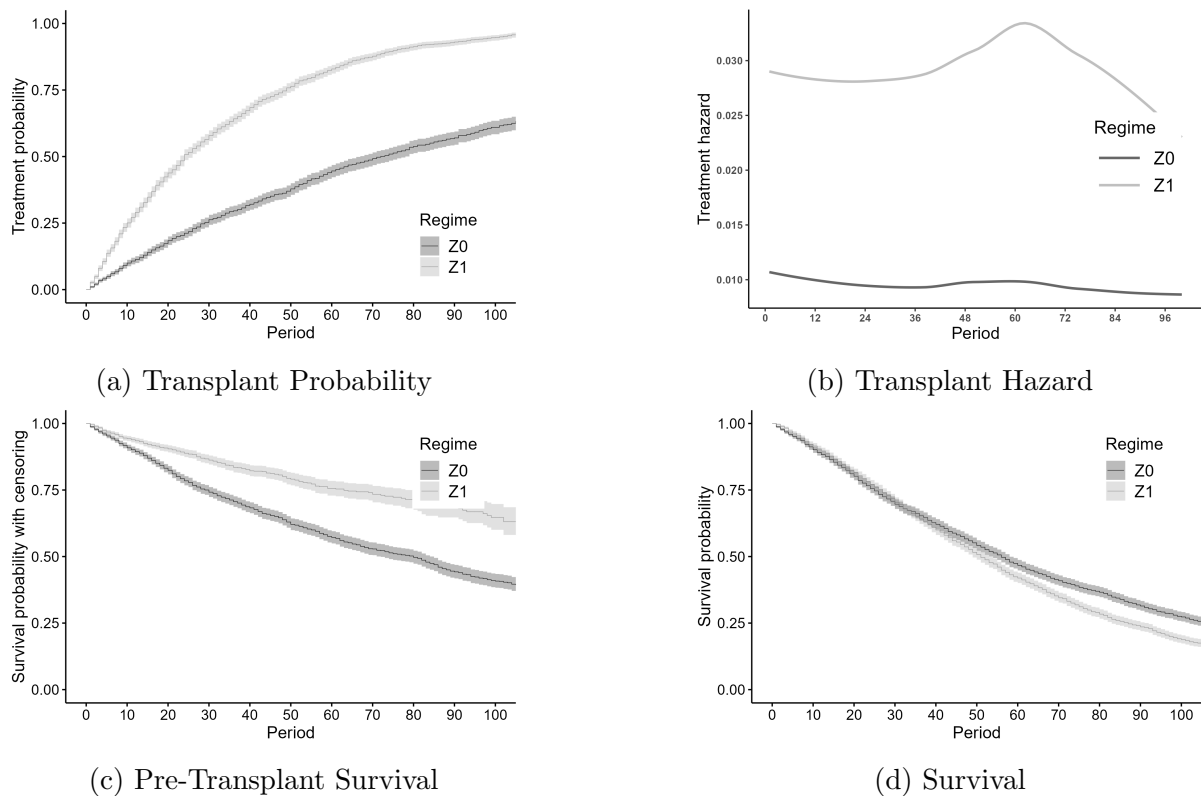


Figure A8: Results from simulation with positive treatment effects

Based on selected sample of Scientific Registry of Transplant Recipients data. Selected sample is described in section 4. $N_{Z=0: no-Tr} = 1013$, $N_{Z=0: Tr} = 453$, $N_{Z=1: no-Tr} = 441$, $N_{Z=1: Tr} = 1093$.

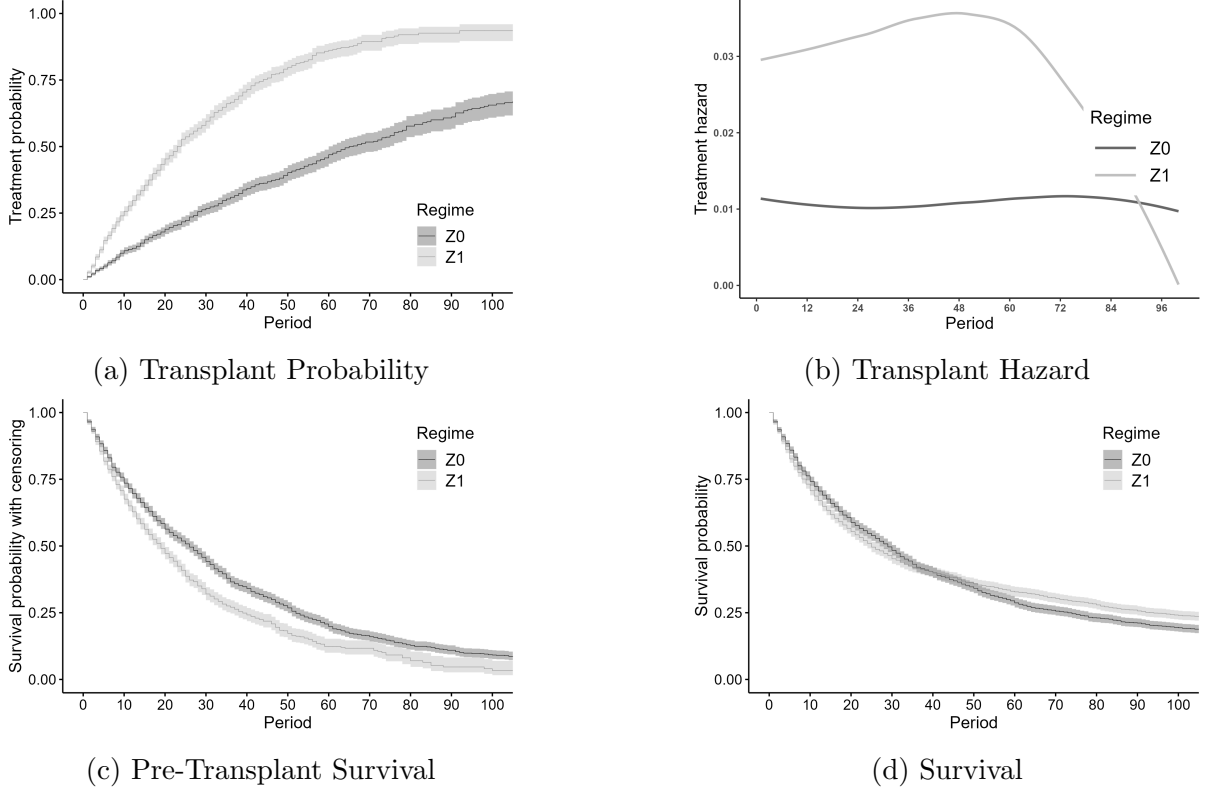


Figure A9: Results from simulation with negative treatment effects
Based on selected sample of Scientific Registry of Transplant Recipients data. Selected sample is described in section 4.
 $N_{Z=0: no-Tr} = 1013$, $N_{Z=0: Tr} = 453$, $N_{Z=1: no-Tr} = 441$, $N_{Z=1: Tr} = 1093$.

G.4 Reservation utilities in dynamic discrete choice model

Consider the post-treatment Bellman equations from the setting described in section G.

$$V_{0,t} = w_0 - c + \rho \lambda \mathbb{E}_w [\max\{V_1(w), (1 - \pi)V_{0,t+1} + \pi V_{0,t+1}^{tr}\}] + \rho(1 - \lambda)[(1 - \pi)V_{0,t+1} + \pi V_{0,t+1}^{tr}]$$

$$V_{0,t}^{tr} = w_0 - c + \rho \lambda \mathbb{E}_{w^{tr}} [\max\{V_1^{tr}(w), V_{0,t+1}^{tr}\}] + \rho(1 - \lambda)V_{0,t+1}^{tr}$$

This second equation can be written as,

$$V_{0,t}^{tr} = w_0 - c + \rho \lambda \int_{w^{tr*}}^{\infty} (V_1(w^{tr}) - V_{0,t+1}^{tr}) dG^{tr}(w) + \rho V_{0,t+1}^{tr}$$

Since all parameters and distributions in the model are time independent, we have a stationary reservation utility strategy. In a stationary strategy $V_{0,t}^{tr} = V_{0,t+1}^{tr} = V_0^{tr}$ for all $t > 0$. Furthermore, the reservation utility w^{tr*} is such that the agent would refuse any offer below it and accept any offer above it so $V_1(w) = \frac{w}{1-\rho}$ if $w \geq w^{tr*}$, V_0^{tr} if $w^{tr} < w^{tr*}$, and $V_1(w^{tr*}) = \frac{w^{tr*}}{1-\rho} = V_0^{tr}$ if $w = w^{tr*}$. It follows that,

$$\begin{aligned} V_0^{tr} &= w_0 - c + \rho \lambda \int_{w^{tr*}}^{\infty} (V_1(w^{tr}) - V_0^{tr}) dG^{tr}(w) + \rho V_0^{tr} \\ &= w_0 - c + \frac{\rho \lambda}{1 - \rho} \int_{w^{tr*}}^{\infty} (w - w^{tr*}) dG^{tr}(w) + \frac{\rho w^{tr*}}{1 - \rho} \end{aligned}$$

Replacing again $V_0^{tr} = \frac{w^{tr*}}{1-\rho}$, rearranging this equation and using integration by parts we obtain the post-treatment reservation utility,

$$w^{tr*} = w_0 - c + \frac{\rho\lambda}{1-\rho} \int_{w^{tr*}}^{\infty} (1 - G^{tr}(w)) dw$$

Note that this reservation utility does not depend on the regime π .

Now consider the reservation utility before treatment with a treatment assignment policy π . Since the problem is still stationary, the agent will again accept any value of w higher than his reservation w^* . We can therefore rewrite

$$\mathbb{E}_w[\max\{V_1(w) - (1-\pi)V_{0,t+1} - \pi V_{0,t+1}^{tr}, 0\}] = \int_{w^*}^{\infty} w - w^* dG(w) + (1-\pi)V_{0,t+1} - \pi V_{0,t+1}^{tr}$$

which results in the same V_0 value function,

$$V_0 = w_0 - c + \frac{\rho\lambda}{1-\rho} \int_{w^*}^{\infty} w - w^* dG(w) + \rho[(1-\pi)V_0 + \pi V_0^{tr}]$$

Since the agent will accept any value of w higher than his reservation w^* we know that $V_1(w^*) = V_0 = \frac{w^*}{1-\rho}$ which we replace in the above equation and rearrange to get,

$$w^* = \frac{1-\rho}{1-\rho+\rho\pi}(w_0 - c) + \frac{\rho\lambda}{1-\rho+\rho\pi} \left(\int_{w^*(\pi)}^{+\infty} w - w^* dG(w) \right) + \frac{\rho\pi}{1-\rho+\rho\pi} w^{tr*}$$

We can further show prove that w^* is increasing in π if $V_0^{tr} > V_0$:

First we rewrite the previous equation to isolate $\int_{w^*}^{+\infty} w - w^* dG(w)$,

$$\int_{w^*}^{+\infty} w - w^* dG(w) = \frac{1-\rho}{\rho\lambda} \left[c - w_0 + \frac{1-\rho(1-\pi)}{1-\rho} w^* - \frac{\rho\lambda\pi + \rho(1-\lambda)\pi}{1-\rho} w^{tr*} \right]$$

Let us hold w^* constant in the previous equation. w^{tr*} is also constant because it does not depend on π . If we increase π , the derivative of the right-hand side with respect to π is

$$\frac{1-\rho}{\rho\lambda} \left[\frac{\rho}{1-\rho} w^* - \frac{\rho}{1-\rho} w^{tr*} \right] \quad \leftrightarrow \quad \frac{1-\rho}{\lambda} [(V_0 - V_0^{tr})]$$

The last equation is negative when $V_0 - V_0^{tr} < 0$, so when the value of treatment is higher than that of no-treatment. Therefore, $\int_{w^*}^{+\infty} w - w^* dG(w)$ is decreasing in π . Furthermore, written as a function of π , with $w^* = w^*(\pi)$, it is also decreasing in w^* which implies that w^* increases in π . The agent is more willing to wait for treatment.

H Accepted kidney quality is increasing with non-sequential kidney offers

The goal is to prove that the expected accepted kidney quality will be increasing in the number of kidneys simultaneously offered to candidates (in consultation with clinicians). We can translate this problem into the following. Say we have an urn with balls labelled $i \in 1, \dots, 100$. There are an infinite amount of balls for each label i . We want to prove that the expected value of the maximum labelled ball is increasing in the number of draws.

Let X_n be a random variable representing the maximum labelled ball after n draws. We can express $\Pr(X_n \leq k)$ as the product of the probabilities of each individual draw being less than or equal to k :

$$\Pr(X_n \leq k) = \prod_{i=1}^n \Pr(\text{Draw}_i \leq k)$$

Since there are an infinite number of balls for each label, the probability of drawing a ball with label i or less is $i/100$ for each individual draw so $\Pr(\text{Draw}_i \leq k) = \frac{k}{100}$. Substituting this into the expression for $\Pr(X_n \leq k)$, we get

$$\Pr(X_n \leq k) = \left(\frac{k}{100}\right)^n$$

It follows that we can express the probability mass function of X_n as:

$$\Pr(X_n = k) = \Pr(X_n \leq k) - \Pr(X_n \leq k-1) = \left(\frac{k}{100}\right)^n - \left(\frac{k-1}{100}\right)^n$$

The expected value of X_n is then given by:

$$\mathbb{E}[X_n] = \sum_{k=1}^{100} k \cdot P(X_n = k) = \mathbb{E}[X_n] = \sum_{k=1}^{100} k \left[\left(\frac{k}{100}\right)^n - \left(\frac{k-1}{100}\right)^n \right]$$

From which it follows straightforwardly that $\mathbb{E}[X_n]$ is increasing in n since the n th power terms for larger k dominate those for smaller k . As a result, the expected value of the maximum labelled ball is increasing in the number of draws. The result can even more simply be shown in the non-infinite case.

I SRTR risk adjusted score model

The SRTR risk adjustment models are based on Bayesian methods (Bayesian Methods for Assessing Transplant Program Performance). In the case of the 1-year post-transplant survival, the model produces survival predictions using the previous 2.5 years of data and adjusting for a set of donor and candidate characteristics, as well as their interactions (Developing Statistical Models to Assess Transplant Outcomes Using National Registries: The Process in the United States). Given the amount of included variables and the amount of missing variables, some adjustments are necessary. First, some lower threshold of national events are imposed (over 25 events), then a chained equation mean matching algorithm is used to impute values when missing, and last LASSO is used for variable selection. For the offer-acceptance risk adjustment score, any kidney which was never accepted does not enter the calculation.

Some questions have been posed concerning these predictive scores. As remarked in the following official [Q&A:How can my program have 0 graft failures, but still be ranked as a tier 4 program?](#), some transplant centers with 0 graft failures receive scores of 4 out of 5 in their performance evaluation. SRTR’s explanation for this seeming anomaly is that it results from transplant centers offering very healthy kidneys to very healthy candidates. Since the expected graft failure is very low for this combination, transplant centers receive reduced scores. While the final outcome may be desirable, the fact that the predictive statistical model itself, which is purely a function of candidate and donor characteristics, down-ranks transplant centers with perfect 1-year graft records is strange.

J Can candidate selection on kidney quality explain observed empirical patterns?

In this section we build on the incentive-based explanation set out in [G](#). We consider again a forward looking agent model as described in the previous section, but assume reservation survival thresholds are invariant to differences in the kidney offer rate. Instead, the setup assumes only that candidates’ kidney quality acceptance thresholds change, and allow this change to be heterogeneous depending on underlying unobserved health. Because there is uncertainty around what size a health shock a candidate will receive each period, agents’ (candidate and/or transplant centers) decisions must balance a trade-off. At time t , a candidate would like to select the kidney at time t or later which ensures the longest post-transplant longevity while acknowledging that their probability of succumbing to a negative health shock increases the longer they remain on the waitlist. This optimal stopping problem is the object of study in [Agarwal et al. \(2021\)](#).

In general, facing a higher kidney offer rate, rational candidates will become more selective about the quality of the kidney they are willing to accept. This change can be modelled as a rise in the kidney quality threshold in response to an increase in the kidney

offer rate. It will also lead to an average decrease in the acceptance to offer ratio. A priori, however, it is unclear whether the acceptance to offer ratio will decrease more for higher or lower health candidates. Facing a higher offer rate, higher health candidates can reliably wait longer for a high quality kidney than lower health candidates. However, if the marginal returns to a higher quality kidney are larger for lower health candidates, meaning they will in expectation benefit more from an equal time wait than higher health candidates, then lower health candidates may have a larger decrease in their acceptance to offer ratio.

For the empirical patterns in Figure 1 of the introduction to arise in the forward looking agent model described in this section, the second stated dynamic must be more prevalent. Facing a higher offer rate, lower health candidates become relatively more selective about which kidneys to accept compared to higher health candidates.

At first glance, the results in Figure 4a appear to corroborate with the kidney quality selection hypothesis. More specifically, if the relative acceptance to offer ratio is lower for poor health candidates in the AB blood group than in the O blood group, then the distribution of health among candidates accepting a transplant in the first months on the waitlist should be more skewed towards healthier candidates in the AB-blood type group. Relatively more healthy candidates receiving a transplant in the AB-blood regime should in turn lead to higher survival among those transplanted early in the AB-blood type regime.