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DISCUSSION PAPER SERIES

IZA DP No. 16995

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ABSTRACT

Biological, Behavioural and Spurious Selection on the Kidney Transplant Waitlist*

The kidney allocation system aims to distribute kidneys from deceased donors in an equitable and potential-life optimising manner. This is a difficult task, not least because intrinsic biological differences, such as a person's ABO blood type, influence the allocation. This paper begins by presenting a curious and undocumented empirical fact: candidates on the kidney transplant waitlist with blood types implying they will more rapidly be offered a kidney display lower pre-transplant survival. The paper investigates whether this difference in pre-transplant survival is due to biological, behavioural, or spurious selection. To that end, we promote a two-in-one randomization design which allows us to credibly fit our empirical setting within a dynamic potential outcomes framework. Using this framework, drawing from economic theory, and noting problematic financial and legal market incentives, the paper systematically evaluates different explanations for pre-transplant survival patterns. Our analysis establishes a small set of behavioural explanations which directly inform debates about how to reduce the excessive discard of viable kidneys in the US transplant market.

JEL Classification: I12, I18, C22, C41

Keywords: kidney transplant, expectation effects, dynamic treatment effects, survival models

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1 Introduction

Healthy donated organs for transplantation are under-supplied. In the United States, there are around 140,000 candidates on the kidney transplant waitlist, with around 40,000 added each year but only 25,000 removed after a successful transplantation (Lentine et al., 2023). Because of this under-supply, the existing allocation system goes to great lengths in trying to ensure an optimal and equitable distribution of available kidneys. One of the difficulties in devising an optimal distribution strategy is navigating the problem of intrinsic biological inequalities.

Underlying the feasibility of a kidney transplant is the need for compatibility between the donor and the recipient's ABO-blood type and human leukocyte antigen (HLA) tissue type. Without sufficiently high compatibility on both, a donated organ will generate an immune response and result in graft failure.[1] As a result, candidates with different blood types and HLA-tissue types can face stark differences in their likelihood of rapidly finding a suitable donated kidney.[2]

The differences are particularly pronounced in the case of blood types. AB-blood candidates, who are universal recipients, have the highest hazard of receiving a transplant at any time in the first four years on the kidney transplant waitlist and, as a result, have a higher overall probability of receiving a transplant than any other blood type. This difference is clear in Figures 1a-1b for kidney transplants from deceased donors,[3] when comparing AB-blood type to O-blood type candidates who can only receive a transplant from other O blood type donors.

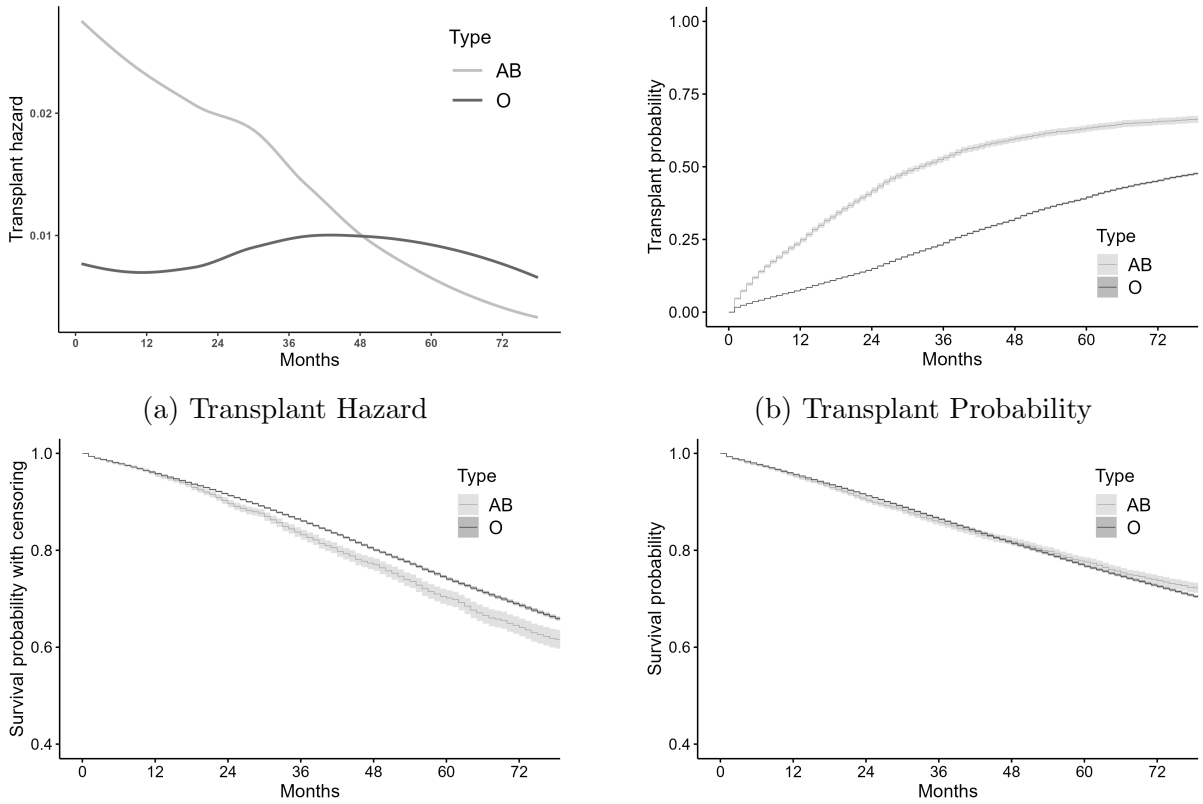
Differences in transplant hazard rates are paired with curious pre-transplant trends in survival. Figure 1c plots the probability of survival for AB-types and O-types when censoring the duration to death at the moment a candidate receives a kidney transplant. We see that over the first 4-5 years on the waitlist, the AB-type candidates have a significantly lower survival rate than O-type candidates prior to receiving a kidney transplant.

Several explanations are consistent with this empirical pattern. First, there may simply be differences in survival for candidates of different blood-types, possibly due to correlated socio-economic or biological factors. Second, differences in pre-transplant survival may be directly related to biological or behavioural selection of the individuals receiving a transplant. For example, it may be that candidates with better health are more likely to be offered and/or accept a kidney. If this were the case, a larger share of high potential survival patients would be censored for the AB blood group in Figure 1c. As a result, differences in pre-treatment mortality would be largely due to selection on

[1] ABO incompatible transplants are possible but remain rare (de Weerd and Betjes, 2018).

[2] Another important determinant is a candidate's immune sensitisation (cPRA), which is a measure of how likely, based on existing antigens, a candidate's body is to reject a transplant.

[3] Much of the early interest in allocation systems from economists focused on kidney transplant exchange programs among living donors (Roth et al., 2004, 2007; Agarwal et al., 2019). These exchange programs, along with the larger market for living donor kidneys, represent a minority of recorded transplantations. The majority, over 70%, of kidneys available for transplant are from deceased donors.



(a) Transplant Hazard

(b) Transplant Probability

(c) Pre-Transplant Survival

(d) Survival

Figure 1: Survival of candidates on the kidney transplant waitlist
Based on selected sample of Scientific Registry of Transplant Recipients data described in Appendix B.
Observations: $N_O = 134,730$, $N_{AB} = 10,544$.

observed or unobserved biological factors. A relevant, and related, question is whether the bias towards healthy candidates receiving transplants is mainly driven by candidate or transplant center decisions. Selection could also take other behavioural forms. In line with search models, candidates, in consultation with clinicians, may integrate the kidney offer rate in their decision making process. In particular, it may be that candidates with worse health characteristics respond differently to a higher kidney offer rate than candidates with better health. The observed pre-transplant patterns can arise if candidates with worse health are relatively more selective about which kidneys to accept when facing a high kidney offer rate. We also cannot exclude that candidates waiting for a kidney become less diligent about their personal health in response to a higher kidney offer rate, moral hazard which may increase the possibility of a negative health shock and lead to the observed empirical pre-transplant patterns. Without a systematic empirical assessment of assumptions pertaining to each theory, it is not possible to appraise the plausibility of each explanation.

This paper investigates the source of pre-transplant differences in survival by combining a dynamic potential outcomes framework with a detailed discussion of unobserved confounders and selection drawing from economic theory. More specifically, as our baseline framework, we use a dynamic treatment effect model which combines the G-computation

identifying assumptions of Robins (1986) with the decomposition of causal effects proposed in Abbring and Van den Berg (2005) for settings with an endogenous intermediate treatment and final outcome which are both duration variables. In addition, to separate competing hypotheses, we call on economic insights including those from theoretical search models in economics. We further relate our discussion to the complex financial and legal incentives influencing kidney allocations and acceptance.

Understanding processes of selection on transplant waitlists is central to current reforms in the US transplant system. These reforms are attempting to address the long-standing and increasing discard of suitable donated kidneys (Senate hearing: UNOS).[4] The central market failure driving this loss is that poorer health individuals who could benefit from suboptimal kidneys, both in quality and quantity of life, fail to receive them. Despite the persistence and severity of this issue, it is unclear whether this market failure is mainly due to candidates refusing to accept suboptimal kidneys, or whether misaligned financial incentives bias transplant centers to refuse suboptimal kidneys. With a view to future policy changes, the analysis allows us to rule out certain hypotheses for the source of this selection problem. It further outlines which assumptions are required for the remaining hypotheses to explain the selection problem, relates these assumptions to agents' financial and legal incentives, and underlines the role of unobserved factors, including biases in expectations, in decision making processes.[5]

The paper begins by presenting a two-in-one randomization design with a wider appeal for experimental and non-experimental evaluations of information effects with duration variables. It is inspired, among others, by the literature on threat effects in active labour market settings (Black et al., 2003; Arni et al., 2022).[6] We consider a situation in which individuals are randomized to a specific type of *regime* upon entering a state at time 0. This regime is a special type of randomization which conveys information and dictates a stochastic propensity for the timing of a future *treatment* among agents, as opposed to a specific treatment time. Thereafter, at different moments in time and depending upon their regime, surviving agents are randomized to actually receive treatment. In general, the design circumvents a common difficulty in empirical applications of dynamic treatment effect models by ensuring that the sequential unconfoundedness assumption holds by construction.[7]

In our case, the regime randomization is a candidate's blood type which prescribes a blood-type specific hazard for kidney offers, but does not determine exactly when a

[4] In 2021, the percentage of deceased donor kidneys that were retrieved but not utilized for transplantation increased to 24.6%, a rise from the 17.9% in 2011 (Lentine et al., 2023).

[5] Our discussion also relates to endogenous attrition in longitudinal panel studies and the use of instruments in settings with dynamic treatment assignment.

[6] The literature on pre-treatment effects has also touched on several fields of public policy. Early areas of focus include anticipation effects of tax reforms (Mertens and Ravn, 2012), and sorting in the housing market to evaluate the value of school facility investments (Cellini et al., 2010).

[7] This design can readily be extended to experimental setups in which the researcher is interested in how expectation or other placebo-type effects alter the effect of a treatment.

suitable kidney will be offered. In addition, we observe all health related variables used in the allocation of kidney offers. Within our dynamic treatment effect framework, we explain that if the variables determining kidney offers and accepted kidneys were to coincide perfectly, we would be able to identify pre-transplant regime effects and ex-post transplant effects depending on candidates' blood type. Because we cannot guarantee this assumption holds, and we only observe accepted kidneys, not offered kidneys, we do not focus on obtaining causal ex-post transplant effects.[8] Instead, we focus on pre-transplant effects and leverage the dynamic decomposition framework to separate our discussion of selection into two parts. A first part relates to selection on the health-related variables determining kidney offers which are used by OPTN/SRTR and the prominent ones determining survival on the waitlist, the US transplant network organisation, and which we observe in our data. The second part relates to selection on other unobserved health-related variables. Separating these parts speaks directly to policy questions concerning variables, in particular behavioural ones, outside the current scope of OPTN/SRTR.

Using a semi-parametric proportional hazard model,[9] we show that over the first 5 years on the kidney transplant waitlist, AB blood type candidates have a 6-7 percentage point higher pre-transplant probability of death relative to the O blood types baseline of about 24 percent probability of death. These pre-transplant effects appear across various model specifications and in various subsamples, including males and females, white and non-white people, a sample which includes candidates who receive kidney transplants from living donors and candidates entering the waitlist before and after a reform in the allocation mechanism in December 2014. The results suggest that pre-transplant differences in survival are due to selection or confounding on variables outside the kidney offer mechanism set, and outside additional prominent ones held by the Scientific Registry of Transplant Recipients (SRTR).

Our analysis thereafter assesses the various forms of selection and confounding on unobserved variables which can plausibly give rise to the pre-transplant empirical patterns. First, we assess whether the pre-transplant effect is due to unobserved confounding variables correlated to ABO blood type groups. We argue this hypothesis to be unlikely due to the one-to-one ordinal relation in the data between the kidney transplant rates by blood type and pre-transplant survival by blood type.

In a second part we turn to incentive-based explanations for the observed pre-transplant patterns. To start, we present a theoretical search model with forward looking optimising agents who can only adjust their pre-transplant reservation survival threshold (ie. physical health) in response to changes in the kidney offer rate. We show both theoretically and in simulations that the empirical patterns under a standard search model can only be reproduced if we assume transplants produce a negative health shock. Thus, if lifestyle

[8] Agarwal et al. (2020) offer a comprehensive ex-post analysis focusing on patient life-years from kidney transplants where they exploit instrumental variation in offers and a continuous shifter of choices to achieve identification.

[9] We present simulation results to assess the performance of our estimator.

and health adjustments are to explain the pre-transplant survival patterns, then they are to be explained by important moral hazard. Although this hypothesis cannot be entirely excluded, there is no existing evidence for strong moral hazard responses to differences in kidney offer rates.

A third hypothesis is that agents with differing unobserved health change their kidney acceptance threshold asymmetrically in response to an increase in the kidney offer rate. More specifically, we explain that the empirical patterns in the data are consistent with a model in which, when facing a higher kidney offer rate, candidates with worse health are relatively more selective about which kidneys they accept than candidates with better health. This selection theory offers a testable prediction for the transplant decision in the first period on the waitlist. It also offers a testable prediction for the relative effect of a transplant across blood type regimes for those who accept a kidney in the first period. We show that, on the face of it, both of these period one results suggest that differences in pre-transplant survival can be attributed to candidate-clinicians's behavioural changes resulting from differences in the kidney offer rate.

In a last part, we question this behavioural response to differences in the kidney offer rate. We first show that the observed pre-transplant patterns in Figure 1c will arise mechanically if kidney acceptance and pre-transplant survival are both increasing in unobserved health, and kidney offers are higher in the AB-blood type group. However, these assumptions alone do not predict the previously mentioned first period results. We explain that the first period results can only arise in a setting without behavioural responses to changes in the kidney offer rate under certain conditions. If the main decision-maker is the clinician/transplant center, then a sufficient set of additional assumptions is that the quality of offered kidneys is independent of blood type, that transplant centers offer more kidneys to higher health candidates, and that transplant centers make simultaneous decisions over several viable kidney offers in a non-null amount of cases. If the main decision-maker is the candidate, then the observed patterns can arise if two conditions replace the assumption that transplant centers offer more kidneys to higher health candidates. The first is that the distribution of offered kidneys in terms of quality is right skewed, with more low-quality than high quality offers. The second is that lower health candidates are more likely to reject a kidney than higher health candidates.

Existing studies and recent policy changes support both sides of these different assumptions. In terms of transplant center incentives, OPTN performance evaluations of transplant centers are currently expanding to include additional measures. Prior to July 2023, performance evaluations were based on a 1-year post transplant risk-adjusted candidate survival score. Post-2023, assessment scores for transplant centers have expanded to include an offer-to-acceptance ratio score as well as a survival score for candidates on a transplant center's waitlist. These adjustments are explicitly aimed at reducing the waste of viable kidneys. What is less clear is whether theses performance evaluation

adjustments are meant to achieve this goal by offsetting other financial incentives[10] or re-center clinicians' misunderstanding of risk adjustment in the 1-year post-transplant survival model. Anecdotal reports suggest the latter is important, leading clinicians to base acceptance decisions on the simpler heuristic of 1-year post-transplant survival rather than the risk-adjusted measure (Mohan et al., 2018; Kimberly et al., 2018).[11]

On the side of candidates, they must be informed by law when offered certain sub-optimal kidneys (OPTN Final rule 121.11(b)(iV)). This signal is susceptible to trigger excessive rejections among less healthy candidates who, because they have a lower potential-life expectancy, are more likely to receive such offers. In addition, existing literature (Zhang, 2010) suggests that lower quality kidneys rejected by many candidates and offered to lower health candidates are more likely to be rejected based on the bad signal rather than the objective kidney quality.

Our main policy suggestion following the analysis of this paper is to develop a statistical system which, for each offered kidney, predicts the expected amount of time any given candidate within a transplant center can expect to wait until receiving a kidney at least as good as the proposed one. This type of system can easily be built adjacent to the current score models and conveyed to transplant centers and candidates alike with a view to adjusting misaligned expectations.

The discussion and results in this paper contribute to several fields. The methodological setup in this paper builds on a rich history of dynamic treatment effects work in economics (Ham and LaLonde, 1996; Abbring and Van den Berg, 2003; Heckman and Navarro, 2007; Heckman et al., 2016; Han, 2021). The paper also adds to a growing field addressing expectation effects in economics (see Haaland et al. (2023) for a survey). Our results also contribute to the literature on transplants in general and kidney transplants in particular, with notable recent work focused on optimal allocation mechanisms for the deceased donor waitlist given candidate-clinician discretion to reject offers (Zhang, 2010; Agarwal et al., 2018, 2021, 2020). Finally, the results also relate to research underlining the relevance of market dynamics in the supply of kidneys (Dickert-Conlin et al., 2019; Teltser, 2019).

The remainder of the paper proceeds as follows. We begin in the next section by briefly introducing the kidney transplant allocation system. Section 3 introduces the potential outcomes framework, describes causal effects of interest, discusses the identifying assumptions in relation to our kidney transplant setting and describes our estimation approach. Section 4 describes our data and Section 5 presents our empirical results.

[10]Some studies suggest the financial cost of transplants for lower health candidates are higher in absolute and marginal terms after medicare compensation (Axelrod et al., 2017, 2018).

[11]If well understood, the risk adjustment model, which accounts for donor, candidate, and donor-candidate interaction variables, should not have incentivized transplant centers to ignore lower quality kidneys or unhealthy candidates.

2 Kidney Transplant Allocation System

Transplantation is the most effective method to treat end-stage renal disease. Dialysis, the main alternative, is associated with higher mortality and higher overall costs (Axelrod et al., 2018). In the United States, the allocation of deceased kidneys to candidates on the waitlist is designed, coordinated, and administered by the Organ Procurement and Transplantation Network (OPTN). A person is allowed to register on the kidney transplant waitlist and start accruing time when their kidney function falls below 20%.[12] Upon entering the waitlist, OPTN collects in-depth information about the candidate's health conditions, immunological profile, and any other characteristic needed to ascertain kidney compatibility.

When a potential kidney becomes available, OPTN gather extensive biological information about the kidney and the donor's medical history. Through its automated UNet system, OPTN then calculate a priority order for any candidate who is compatible with the potential kidney. The kidney offer process balances equity and efficiency, prioritising candidates on the waitlist following strict criteria. Unlike some other organs, such as livers and hearts, priority is also not given to candidates who need a transplant most urgently.

Prior to December 2014, the main prioritisation criteria were the compatibility of blood and tissue types, the age of the candidate, the time a candidate had been on the waitlist, and their geographic proximity to the center holding the deceased donated kidney (Smith et al., 2012).

This allocation system was deemed to result in excessive amounts of unutilised kidneys, lost graft years,[13] high re-transplant rates, and inequity in distribution for minorities. In an effort to address these issues, and update a system which had not undergone significant change in 20 years, a new allocation system was introduced in December 2014. Some of the changes included adding points for patients with high levels of antibodies, and giving priority to healthier candidates for the highest potential longevity kidneys. This prioritization is computed using an Estimated Post-Transplant Survival (EPTS) score which is a function of age, diabetes status, whether the candidate is on dialysis, the number of years they have been on dialysis, and whether they previously received a transplant. Under the new allocation rules, less emphasis is placed on waiting time and geographical location.[14] In terms of ABO blood type prioritisation, B and O blood type candidates are prioritised for identical blood type kidneys unless perfect tissue compatibility (zero-antigen HLA) exists with AB blood types (Israni et al., 2014). This prioritisation, which existed both before and after the reform, attempts to adjust for the

[12] More precisely when the Glomerular Filtration Rate (GFR) is 20 or below.

[13] Candidates with shorter life expectancy may receive kidneys with the potential to function longer.

[14] In 2021, the geographic criteria was further adjusted to attribute points, on a linearly decreasing scale with notches, based on the distance between a candidate's listed transplant center and the donor hospital (Potluri and Bloom, 2022).

lower supply of B and O blood type kidneys. Key to our future assumptions, the UNet mechanism determining a kidney offer is a function of observed variables of which all health related ones are available to us in our data.

There also exists some discretion on the part of candidates and their clinicians concerning which kidney they are willing to accept. Most kidneys will be rejected automatically based on age, health, and kidney function criteria set by candidates and their physicians upon entering the waitlist. These criteria choices are available in our data. Other kidneys will still be rejected after passing the initial screenings (Gordon et al., 2020). Looking at data offers between January 1, 2010 and December 31, 2013, Agarwal et al. (2018) show that, among kidneys passing screening criteria, only 0.9% of kidney offers are accepted and only 70% of those are actually transplanted.[15] This low acceptance rate is largely due to relatively undesirable kidneys being offered to several thousand patients.[16] Decisions to reject offers have no bearing on future offers. As a result of rejected offers, close to 25% of kidneys are never transplanted.[17] It is an open question, and one we return to in our discussion, whether the central decision maker for these rejections is more often the candidate or the clinician and transplant center.

3 Evaluation Framework

In this section we first introduce the potential outcomes notation and present our evaluation framework, underlining which assumptions identify causal effects and what these assumptions imply in our kidney transplant waitlist setting.

3.1 Potential Outcomes and Treatment Effects

The decomposition framework presented in this section combines the G-computation identifying assumptions of Robins (1986) with the decomposition of causal effects proposed in Abbring and Van den Berg (2005) and also draws on work from Lechner (2009) and Heckman et al. (2016). Our potential outcomes notation combines the dynamic presentation of Abbring and Heckman (2007) with the more familiar notation in static treatment and mediation analyses (Angrist et al., 1996; Imai et al., 2010).

[15] This last discrepancy is due to final crossmatch blood screenings which predict high chances of kidney graft failure. Throughout the text, we abstract from this discrepancy and often refer to transplanted kidneys as accepted kidneys, since the last crossmatch stage occurs after the main behavioural decisions focused on in the paper.

[16] Several factors determine a kidney's quality in general. These include the donor's general health prior to death, their gender and age, as well as their kidney function and anatomy. Some donors die with infectious diseases which the candidate may contract if they proceed with a transplant. See Danovitch (2012) for a comprehensive review of kidney biology.

[17] There is also a substantial amount of variation in kidney-acceptance rates among the 250 transplant centers (Wey et al., 2017).

3.1.1 Regime and Potential Treatment Duration

We follow each agent from the moment of entering an initial state which is set as $t = 0$. In our empirical setting this will be the moment an individual enters the kidney transplant waitlist, but 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 randomized to one of two regimes denoted by the random variable Z (we suppress the subscript i for convenience). Z is observed by the econometrician and may be partially or fully known by the agents. We focus on the most simple setting in which agents can either be assigned to a baseline regime $Z = 0$ or to an alternative regime $Z = 1$.

The regime is a special type of two-in-one randomization which has received little attention in the literature. It is a variable which introduces administrative or other constraints which changes the hazard rate to treatment, but does not determine a specific time for the future treatment. Although the framework allows a general interpretation of the regime randomization, it can find particularly interesting applications when being considered as an information randomization on expectations or beliefs. In our kidney transplant setting, $Z = 0$ for AB blood type individuals who face a high probability of rapidly finding a suitable kidney. $Z = 1$ for O blood type individuals who have a lower probability of rapidly finding a suitable kidney.

To define durations to treatment we use a potential outcomes notation. We allow time to be discrete or continuous when defining potential outcomes and causal effects of interest. Let the random variable S^z be the potential duration to treatment, or equivalently the treatment time, had an agent been subject to regime $Z = z$. The treatment time can take on any value $s > 0$.

We consider the simplified setting in which there is only a single binary treatment which, once allocated, remains permanently thereafter. In our kidney transplant setting, s is the duration of time on the waitlist until an individual receives a kidney transplant. One important peculiarity about the timing of treatment is that it is stochastic from the point of view of the agent. A person may know that they have a low probability of finding a suitable kidney, but will not know the exact time s at which they will receive a kidney transplant.

3.1.2 Potential Exit Duration

We further define the potential outcome $T^{z,s}$ as the duration to exit had the agent been subject to regime $Z = z$, and been treated at $S^z = s$. In our example, $T^{1,6 \text{ months}}$ would be the potential time to death for a person on the kidney transplant waiting list with O blood type ($Z = 1$), and who (would have) received a kidney transplant after 6 months on the waitlist ($S^1 = 6$ months). Several comparisons can be made based on this notation. In this paper, our object of interest is the probability of survival past a period τ given treatment at s. Any comparison therefore requires formulating counterfactual potential

outcomes of the form $\Pr(T^{z,s} > \tau)$.[18]

The usual problem with formulating causal comparisons with this potential outcome is that counterfactuals are never observed. On top of the usual static selection problems, there is dynamic selection in the sense that unobserved variables will influence the timing of exit, and some agents will exit before ever receiving treatment. Within this context, we use the shorthand notation $T^{z,\infty}$ to define the potential non-treated outcome. It represents the duration to exit had the agent been exposed to regime $Z = z$ and had not received treatment in finite time ($s \to \infty$), or, more concretely, by the time τ at which the outcome is evaluated.

3.1.3 Regime and Treatment Effect Decomposition

Using this notation, we can already define our relevant decomposition for a given treatment time s:

$$
\begin{aligned}
\Pr(T^{z,s} > \tau) &= \beta_0 + \beta_z z + \beta_s \mathbb{1}(S = s) + \beta_{zs} z \mathbb{1}(S = s) \\
&\text{with } \beta_0 = \Pr(T^{0,\infty} > \tau) \\
&\quad \beta_z = [\Pr(T^{1,\infty} > \tau) - \Pr(T^{0,\infty} > \tau)] \\
&\quad \beta_s = [\Pr(T^{0,s} > \tau) - \Pr(T^{0,\infty} > \tau)] \\
&\quad \beta_{zs} = [(\Pr(T^{1,s} > \tau) - \Pr(T^{1,\infty} > \tau)) - (\Pr(T^{0,s} > \tau) - \Pr(T^{0,\infty} > \tau))]
\end{aligned} \tag{1}
$$

This decomposition is a reformulation of one first proposed in Abbring and Van den Berg (2005). It parallels the well known decomposition when evaluating heterogenous treatment effects.

As in a heterogeneity decomposition, β_0 represents the average probability of survival past τ had all candidates been of AB blood type and had none of them received a kidney transplant. β_z represents the pre-treatment difference in survival and is the central parameter of interest in our study. It represents the difference in the probability of survival past τ were a candidate to be of O blood type instead of AB blood type, withholding any effects of actually receiving a kidney transplant. β_s represents the difference in the probability of survival past τ were candidates to be of blood type AB and receive a kidney transplant at s compared to their probability of survival were they not to receive a kidney transplant by τ. β_{zs} represents the additional difference in the probability of survival past τ were candidates to be of O blood type and receive a kidney transplant at s relative to β_s.[19]

[18] In this paper our outcome of interest is the probability of survival instead of the expected exit duration $\mathbb{E}[T^{z,s}]$. Focusing on the expected duration to exit may be hampered by the fact that in many duration settings, as is the case in our application, a large fraction of exit outcomes are censored so the right tail of the exit distribution will be poorly approximated. Also, the expected potential exit duration outcomes can all be expressed as functions of the probability to exit, $\Pr(T^{z,s} \leq \tau) = 1 - \Pr(T^{z,s} > \tau)$. Alternatively, some studies focus on the relative effects of the potential hazard as causal effects. The drawback of focusing directly on the hazard to infer about causal effects is that its magnitude is difficult to interpret for cost-benefit analyses without transforming it into a survivor function.

[19] In some cases, the above formulation may not be the most policy relevant decomposition. A more

3.2 Assumptions and Identification of Causal Effects

In this section, we discuss the nonparametric identification of dynamic treatment effects originally formulated in Robins (1986). The evaluation framework will be presented in discrete time with exits and treatment times $t \in \mathbb{N}$ and $s \in \mathbb{N}$ to intuitively parallel assumptions from the static causal inference setting. To reduce notational burden, we assume the observed period interval is the smallest discrete unit of time.

For each agent, when $T \geq S$, we observe the joint distribution (Z, S, T, X) where Z is the regime assignment, S the time to treatment, T the time to exit, and X is a set of baseline covariates at $t = 0$. Since identification is non-parametric, we leave the conditioning on observed baseline ($t = 0$) covariates X implicit for notational convenience. However, we do refer to covariates when discussing the problems of intermediate or time-varying covariates.

3.2.1 Dynamic Unconfoundedness Assumption

The first identification requirement is that the regime is randomized and that treatments are randomized on survivors. For this we invoke the following dynamic unconfoundedness assumptions first proposed by Robins (1986) and introduced into the economics literature by Lechner (2009),

Assumption A.I: Dynamic Unconfoundedness: *For $z = 0, 1$, $s \in \mathbb{N}$, $t \in \mathbb{N}$, $s \geq t$, denote by $\{T^{z,s}\}$, $\{S^z\}$ the sets of all potential variables, then,*

$$\begin{aligned} (i) \quad & (\{T^{z,s}\}, \{S^z\}) \perp\!\!\!\perp Z \\ (ii) \quad & \{T^{z,s}\} \perp\!\!\!\perp \mathbb{1}(S = s) \mid S \geq t, T \geq t, Z = z \end{aligned}$$

In the context of our study, the first assumption states that the blood type is randomized at $t = 0$ with respect to the potential time to death and potential time until receiving a kidney transplant. The second assumption says that receiving a kidney transplant is randomized for the survivors on the waitlist who did not yet receive a kidney transplant. Assumption $A.Ii$ implies and is implied by the 'no-anticipation' assumption of Abbring and Van den Berg (2003), $\Pr(T^{z,s} > \tau) = \Pr(T^{z,s'} > \tau)$ for all $s, s' > \tau$.[20]

In an experimental setup involving a regime changing expectations, the most obvious direct strategy to ensure these assumptions hold would be to randomize individuals at

relevant decomposition may require averaging effects over a treatment time interval, such as treatment before period s. When producing the decomposition relative to treatment in regime $Z = 1$, the average effects over the treatment interval $(0, s]$ are given by $\sum_{t=0}^{s} \cdot \Pr(S^1 = s) \Pr(T^{z,s} > \tau)$. Our average policy parameters of interest would then be β_0, β_z, $\beta_{(0,s]} = \sum_{t=0}^{s} \Pr(S^1 = s) \cdot \beta_s$ and $\beta_{z(0,s]} = \sum_{t=0}^{s} \Pr(S^1 = s) \cdot \beta_{zs}$.

[20] Assumption A.I implicitly contains structural assumptions concerning the timing of causes and effects within a period. In our formulation of the conditioning set, $S \geq t$, we allow treatment at period t to influence exit in the same period. So, receiving a kidney transplant can influence survival immediately. If there is censoring in the sample, one can add a similar dynamic unconfoundedness condition under which right censored observations are dynamically missing (completely) at random.

baseline to two groups: one which is told they have a low chance to receive a future treatment, another which is told that they have a high chance to receive a future treatment. Both groups could also be told their chances (or hazards) between $(0, 1)$ of receiving treatment to enquire into (mis-)perceptions of probabilities. The advantage of this type of randomization strategy in dynamic settings is twofold. First, it can be easily implemented. Second, it satisfies both unconfoundedness assumptions of Assumption $A.I$ by wrapping two randomizations into one. This randomization design would find particular use for researchers interested in how expectation, information or other placebo-type effects alter the effect of a treatment in a context with duration variables.

When it comes to kidney transplants, ethical concerns preclude manipulating people's expectations or randomising the allocation of kidneys. Also, because the central focus of this paper is explaining differences in pre-transplant survival between blood types, we will not appeal to arguments conditional on covariates alone when it comes to ascertaining Assumption $A.I$. For $A.Ii$ in our study, a conditional on covariates assumption would impose that a person's blood type is independent of their potential duration to death and transplant. ABO blood types have been identified as risk factors in specific disease processes such as vascular disease and malignancy (Wu et al., 2008; Sun et al., 2015; Abegaz, 2021) as well as for a small number of infectious diseases (Cooling, 2015). However, on the whole, there is no clear evidence that some ABO blood types consistently lead to higher overall mortality in combination with diseases. That being said, certain ABO blood types are known to be correlated with particular races. These associations are related to other drivers of mortality. As such, our empirical examination of assumption $A.Ii$ proceeds in two parts. We first produce additional specifications with added baseline covariates, including race, biological sex and previous malignancies, among others. These results ascertain the robustness of the estimated pre-transplant effect β_z to some leading choices of confounders and known drivers of mortality. Thereafter, we exploit the four variations of blood type and present subsample results which, taken together, render it difficult to argue that differences in pre-transplant survival operate through pre-existing differences in candidates blood type, rather than different processes of selection over the waitlist.

Assumption $A.Iii$ should be questioned in observational dynamic settings. OPTN, through the UNet system, allocate kidney *offers* to candidates on the waitlist purely based on candidate and donor characteristics, and registered restrictions. As discussed in section 2, although we observe all health related variables held by OPTN, candidates in consultation with clinicians, or clinicians and transplant centers independently, are likely selecting which kidney offers to *accept*. These decisions may be based on variables outside the set determining the allocation of offers, and possibly outside the additional ones available in our data. This selection problem is our main reason for not focusing on post transplant effects β_s and β_{zs} in the paper.[21] However, an important contribution of our

[21] See Agarwal et al. (2020) for a dedicated discussion to ex-post effect in the context of kidney trans-

paper is explaining how a violation of assumption Iii, while biasing the ex-post transplant effects, does not prevent us from developing behavioural and policy relevant insights concerning the pre-transplant effect β_z. Under certain conditions, we can leverage the structure of assumption $A.Iii$ to separate out pre-transplant selection due to prominent variables held and currently used by OPTN, as opposed to other variables.

One condition is that there is no predetermined time from the point of view of candidates or surgeons at which candidates will be offered a suitable kidney. This likely holds as OPOs gather consent from people or relatives of people who experienced severe brain bleeding, have been declared brain dead, or for whom cardiac death is imminent. Retrieved kidneys are then transplanted within 24-48 hours. These time constraints imply that it is difficult to predict well in advance when a new and compatible donated kidney will be brought forward.

In addition, when it comes to kidney offers, we need to justify a source of exogenous randomization in the allocation system. Two such sources are assumed: regional differences in kidney availability and differences in donor-recipient tissue-type compatibility criteria (HLA-type). In terms of tissue compatibility, for two candidates similar on all measures, the one with a higher HLA tissue type compatibility is more likely to receive a kidney transplant.[22] Geographic location also introduces several layers of random variation in kidney offers. Prior to December 2014, large differences in allocation stemmed from regional differences in kidney availability (Ata et al., 2020; Massie et al., 2011). Due to the limited time between a kidney being made available and the time of transplant, few candidates are listed in multiple Donation Service Areas (Dickert-Conlin et al., 2019). As a result, among surviving agents who are similar on all measures, the one living within the same donation service area as that which procured the kidney would be prioritized. Prior to the December 2014 reform, one relevant condition under which some regional prioritization criteria could be ignored is if there was a perfect compatibility on blood and tissue type (zero-antigen mismatch) for a candidate in another donation area. When this occurred, and a kidney was transferred to another donation service area, the service area obtaining the kidney incurred a payback debt which required relinquishing the next available donated kidney with a similar ABO blood type. This debt payback system adds additional exogenous variation to the geographic and tissue compatibility dimension. Because this additional layer of variation was dropped in the new kidney allocation implemented in December 2014, our initial analysis focuses on pre-2015 data. As we will show, the results and insights from the pre-2015 analysis carry over to the new kidney allocation mechanism, which continues to display large geographic variation in deceased donor kidney availability (King et al., 2020).

plants

[22]HLA tissue types are not known to be correlated to blood types (Erikouglu et al., 2011).

3.2.2 Overlapping Support Assumption

In addition, we impose that regimes, treatments, and the decomposition can be evaluated,

Assumption A.II: Overlapping Support: For $z = 0, 1$, $t \in (0, s]$, $s \in \mathbb{N}$, $t \in \mathbb{N}$,

$$\begin{aligned} (i) \quad & 0 < \Pr(Z = z) < 1 \\ (ii) \quad & 0 < \Pr(S = t | S \geq t, T \geq t, Z = z) < 1 \end{aligned}$$

This overlapping support assumption guarantees first, $A.II(i)$, that we observe agents under both regimes. $A.II(ii)$ provides that there is no time before period s at which the treatment is allocated to all, or none, of the untreated survivors.

3.2.3 SUTVA Assumption

We add to these two assumptions Rubin's Stable Unit Treatment Value Assumption (Rubin, 1980) which we rejoin with Robins' consistency assumption (Robins, 1997; Murphy, 2003) in the following,

Assumption A.III: SUTVA: *For* $z = 0, 1$, $s \in \mathbb{N}$,

$$\begin{aligned} S^z &= S \qquad if\ Z = z \\ T^{z,s} &= T \qquad if\ S = s, Z = z \end{aligned}$$

In our empirical setting, this assumption states that the potential duration until an individual receives a kidney transplant and the potential time to death equal their corresponding observed duration to transplant and duration to death in our sample. The usual interpretation of the above is that potential outcomes for an agent do not depend on the observed or counterfactual outcomes of any other agent. However, since our data includes the universe of candidates on the waitlist in the US, and our main goal is to diagnose which forms of pre-transplant selection may be at play for that population, the questions of spillovers and extrapolation when rolling-out a treatment are of second order importance since the data were collected from the hypothetical causal setting of interest.

3.2.4 Identification of Regime and Treatment Effects

Under $A.I - A.III$, we can present the well-known identification result for the separate components of the causal decomposition in equation 1.

$$
\begin{aligned}
\beta_0 &= \prod_{t=0}^{\tau} \Pr(T > t | S > t, T \geq t, Z = 0) \\
\beta_z &= \prod_{t=0}^{\tau} \Pr(T > t | S > t, T \geq t, Z = 1) - \prod_{t=0}^{\tau} \Pr(T > t | S > t, T \geq t, Z = 0) \\
\beta_s &= \prod_{t=0}^{\tau} \Pr(T > t | S = s, T \geq t, Z = 0) - \prod_{t=0}^{\tau} \Pr(T > t | S > t, T \geq t, Z = 0) \\
\beta_{zs} &= \prod_{t=0}^{\tau} \Pr(T > t | S = s, T \geq t, Z = 1) - \prod_{t=0}^{\tau} \Pr(T > t | S > t, T \geq t, Z = 1) - \beta_s
\end{aligned} \tag{2}
$$

where we use a shorthand notation,

$$
\prod_{t=0}^{\tau} \Pr(T > t | S = s, T \geq t, Z = z) \equiv
$$

$$
\prod_{t=s}^{\tau} \Pr(T > t | S = s, T \geq t, Z = z) \cdot \prod_{t=0}^{s-1} \Pr(T > t | S > t, T \geq t, Z = z)
$$

We can also identify the probability of treatment at period s under regime z,

$$
\alpha_z = \Pr(S^z = s) = \Pr(S = s | S \geq s, T \geq s, Z = z) \cdot \prod_{t=0}^{s-1} \Pr(S > t | S \geq t, T \geq t, Z = z)
$$

The above equations are adapted versions of Robins (1986) and Gill and Robins (2001) "g-computation formula" which also admits non-binary and non-permanent treatments as well as non-duration outcomes. As in the general formula, our version allows for causal dependence of treatment histories on outcomes but also of outcome histories on the treatment. Our main parameter of interest throughout the results is β_z representing pre-transplant effects.

3.3 Estimation Approach

We use a simple estimation method which builds on a semi-parametric proportional hazard model.[23] We write the joint exit and treatment hazards in the proportional hazard specification,

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

For the estimation, we first parameterize the duration dependence functions $\lambda_t^T(z, \mathbb{1}(s \leq t))$ and $\lambda_t^S(z)$ as separate piecewise constant baseline hazards depending on Z and whether

[23] We choose a continuous time estimator rather than a discrete time one mainly for computational reasons. Also, for settings in which identifying assumptions are likely to hold and time intervals are small, continuous and discrete time estimators produce similar results in treatment effect duration models (Kastoryano and van der Klaauw, 2022). The latter also provide non-parametric discrete time duration estimators adaptable to our setting and discuss the tradeoffs between discrete and continuous time estimation approaches.

treatment occurred $\mathbb{1}(s \leq t)$. We then estimate the parameters of the joint density of the above by Maximum Likelihood.[24]

Each treated causal effect taking the form $\prod_{t=\tau_1}^{\tau_2} \Pr(T > t | S = s, T \geq t, Z = z)$ is then computed as,

$$\sum_{\{i\}} \hat{w}_i(s) \exp(-\int_{\tau_1}^{\tau_2} \hat{\theta}_t^T(x_i, z, s)dt) = \sum_{\{i\}} \hat{w}_i(s) \exp(-\int_{\tau_1}^{\tau_2} \hat{\lambda}_t^T(z_i, \mathbb{1}(s_i \leq t)) \exp(x_i' \hat{\beta}^T)dt)$$

While non-treated causal effects taking the form $\prod_{t=\tau_1}^{\tau_2} \Pr(T > t | S > t, T \geq t, Z = z)$ are computed as,

$$\sum_{\{i\}} \hat{w}_i(s) \exp(-\int_{\tau_1}^{\tau_2} \hat{\theta}_t^T(x_i, z, S > \tau_2)dt) = \sum_{\{i\}} \hat{w}_i(s) \exp(-\int_{\tau_1}^{\tau_2} \hat{\lambda}_t^T(z_i, 0) \exp(x_i' \hat{\beta}^T)dt)$$

with weights given by,

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

Finally, we use the delta method to compute standard errors around the causal effects of interest.[25]

In appendix E we provide some simulation results assessing the robustness of our estimation method on simulated data generated from a dynamic discrete choice search model. We also briefly discuss how the parameters of the above estimation relate to our identifying assumptions and to the search model. Our estimator performs well when the underlying data generating process is not excessively non-linear.

4 Data

This study used 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 US, 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.

The dataset we use contains detailed information on candidate characteristics, as well as information on the date a candidate enters the waitlist, when they receive a kidney transplant, and the date of death. In our initial analysis we include all candidates who entered the waitlist between June 1st 2002 and December 1st 2014, the month in which

[24] R programming code is available at www.skastoryano.com.

[25] To obtain the expected value of the effects over an interval of treatment times $[1, s]$, one should replace the denominators in the weights with a sum over $t = 1, \ldots, s$, $\sum_{t=1}^{s} \sum_{\{i\}} \hat{\theta}_t^S(x_i, 1) \exp(-\int_0^t \hat{\theta}_\tau^S(x_i, 1)d\tau)$.

the new kidney allocation system was implemented. We exclude any candidate who was scheduled to receive multiple transplants and candidates who were under 18 years of age, as both of these groups follow special allocation rules. In our baseline analysis, we also exclude candidates who receive a transplant from a living donor since those candidates effectively jump the line based on unobservables.[26] Finally, we only include first spells on the waitlist thereby excluding repeated observations for candidates who experience graft failure or renewed kidney failure. We exclude from the analysis a limited group of candidates with rare subgroup blood types. In the analysis, we also exclude the small fraction of immune sensitised candidates with a first cPRA score above 0 given that these candidates face special allocation rules. Appendix B provides a full description of our data selection and the fraction of dropped observations at each step.

In our main decomposition estimation, we take the unit of time to be two months. Our outcome duration T of interest is the time from the moment a candidate with kidney failure enters the waitlist until the time of death, if observed. In the SRTR data, information on the date of death is obtained from social security records (Massie et al., 2014)

The duration to treatment S is the time until a candidate receives a kidney transplant. The regime randomization Z in our baseline analysis is the biological randomization to a blood type AB or O. We purposively choose these two groups to draw inference from pre-transplant differences in survival. As illustrated in A1a of Appendix C, AB blood type candidates, who are universal recipients, have the highest hazard to transplant over the first 3 years on the waitlist among all ABO blood types. O blood type candidates and B blood types, as we will discuss later, have the lowest propensity to receive a transplant, while A blood type candidates have a propensity lying between that of AB types and the others.

In our main specification, we control for every health related variable relevant to the offer allocation mechanism and other prominent variables related to a candidate's health which do not contain excessive amounts of missing values. Given our sample selection, only the candidate age variable is relevant to offer allocations prior to the 2014 reform. In the post-2015 analysis, the only offer relevant variable in our selected sample is the Estimated Post Transplant Survival (EPTS) score, which is the candidate health score used to match donors to candidates.[27] In addition, we include time $t = 0$ variables for: age, diabetes status, whether the candidate is on dialysis, whether they previously received a transplant, BMI, biological sex, race, whether they previously had a malignant tumour, whether they would accept a kidney from a hepatitis B positive donor, and

[26]Note that this data selection may be endogenous to the blood type if candidates with a different propensity to finding a suitable kidney have different propensities to seeking out living donors. We return to this point in our results.

[27]EPTS scores are calculated as follows: $EPTS = 0.047 \cdot \max(Age - 25, 0) - 0.015 \cdot Diabetes \cdot \max(Age - 25, 0) + 0.398 \cdot PriorOrganTransplant - 0.237 \cdot Diabetes \cdot PriorOrganTransplant + 0.315 \cdot \log(YearsonDialysis + 1) - 0.099 \cdot Diabetes \cdot \log(YearsonDialysis + 1) + 0.130 \cdot (YearsonDialysis = 0) - 0.348 \cdot Diabetes \cdot (YearsonDialysis = 0) + 1.262 \cdot Diabetes$.

whether they would accept a kidney from an HCV positive donor.[28] At no point do we control for intermediate variables which require stringent assumptions to be considered exogenous.

The main set of variables for each blood type are described with summary statistics in appendix B columns 1-4. The average age of candidates on the waitlist is just under 52 years of age 40% of which are women. Most candidates are on dialysis upon entering the waitlist ($> 90\%$) and slightly over a third of them have diabetes upon entering the waitlist. In general, different blood types are comparable along all available health measures, but are not balanced on race. The O and A blood type groups have lower shares of minorities, with a relatively larger share of Black people in the B blood type group, and higher share of Asian people in the AB and B blood type groups.

5 Empirical assessment of pre-transplant selection

Our results discuss the specific hypotheses consistent with the patterns observed in Figures 1a and 1c of the introduction, as well as additional tests on the data. We then relate these hypotheses and their necessary assumptions to the current discussions about reforms to the organ transplantation system in the US.

5.1 Selection on kidney offer variables

A first point we need to inspect is how much of the difference in pre-transplant survival displayed in Figure 1c can be explained by selection on variables used in the allocation of kidney offers or other prominent health variables. Figure 2a presents the pre-transplant effect, β_z in our decomposition, on survival past one to five years when excluding any covariates. Figure 2b presents these same effects when adding covariates which are relevant in the allocation of kidney offers.[29] Prior to 2015, and given our data selection, this includes only age which we introduce into the model as a series of age bracket indicators. Our results indicate that O-blood types have a higher survival in all three specifications, but the pre-transplant effect is larger and significant at traditional levels only when including covariates.[30] In contrast to static settings, this difference in effects depending on whether covariates are included does not necessarily suggest that assumption $A.Ii$ is violated and blood-types are non-random at $t = 0$. This is because in our dynamic

[28] We do not include other relevant variables which present a large fraction of missing values: hypertension (26% missing), creatinine measure of candidate (33% missing). We also drop in our main analysis the limited (6.7%) of candidates who state they would accept a kidney from Hepatitis C donor.

[29] We present in Table A2 of Appendix D the full set of parameter effects from our decomposition for each column.

[30] In the estimation, we specify the segments of the duration dependence terms to be one year each. This choice is guided by the desire to calibrate our model on the observed patterns of Figure 1c in our estimation without covariates. The estimate of β_z in 2a approximates well the observed patterns in Figure 1c.

setting, covariates also serve the purpose of adjusting for selection on observed variables relevant to being offered, and accepting, a kidney transplant at any time $t > 0$.

Our point estimates in Figures 2b indicate that the difference in pre-transplant survival of candidates with O blood type relative to AB blood types increases to 5.0 percentage points over the first 5 years on the waitlist. Given the decreasing baseline survival of the AB blood group observable in Figure 1c, the difference in survival in percentage terms between both blood groups increases from 2 percent to 7 percent over the first five years on the waitlist. Figure 2c adds all additional $t = 1$ baseline covariates related to candidate's health in order to inspect how much remaining variation can be captured by major health risk factors. We do not find that the additional set of covariates substantially affect our pre-transplant estimates. Hereafter, all modelled pre-transplant effects include all covariates.

As previously noted, the kidney allocation system was adjusted in December 2014. The main change relevant to our analysis sample included giving priority to healthier candidates for the highest potential longevity kidneys through the EPTS measure.[31] Figure 2d considers whether pre-transplant effects changed for candidates who entered the waitlist under the new system. We find the pre-transplant effects follow the same patterns pre- and post-2015.

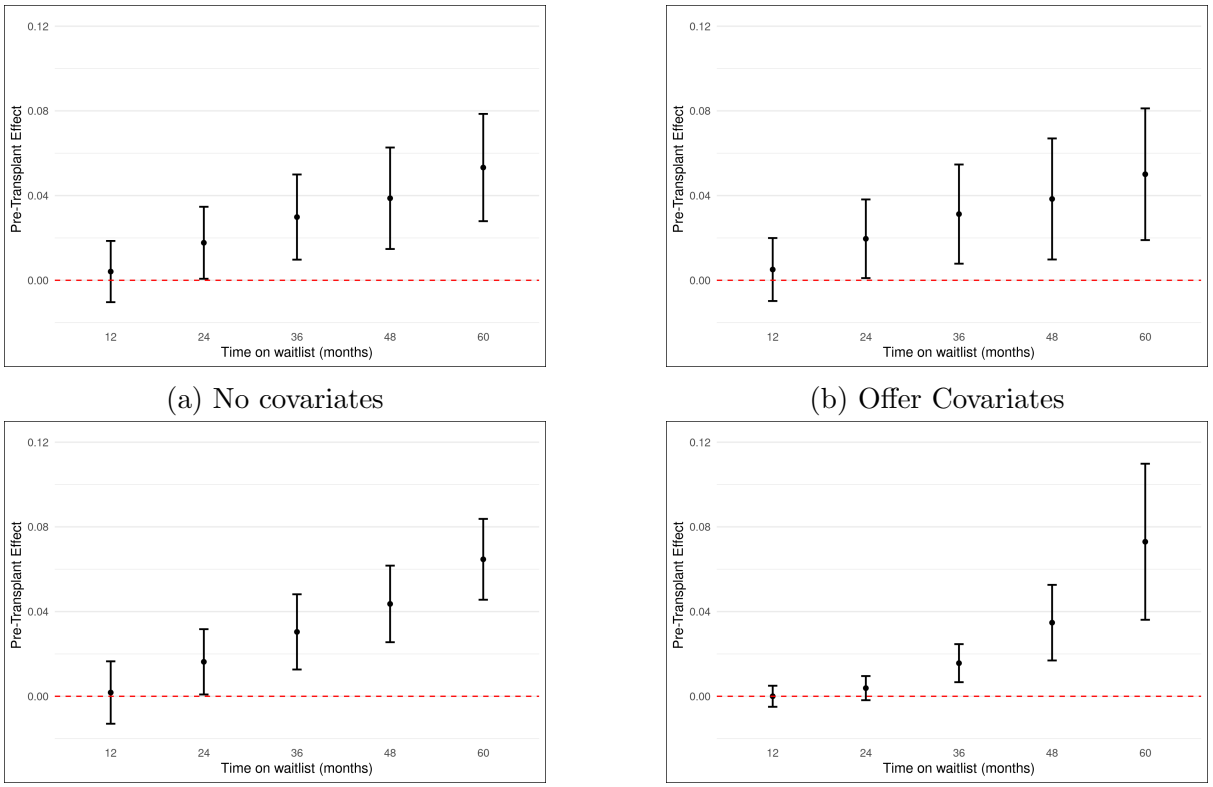
Taken together, the results from Figures 2a-2d show a pre-transplant effect which cannot be readily explained by selection into treatment on the basis of variables used when allocating kidney offers nor on the basis of other major risk factor variables influencing survival. Figure A3 of Appendix C shows that these effects appear for White people and minorities, and on both sexes, albeit noticeably lower for females. The results also remain robust to including the potentially non-random candidates who receive a living donor kidney transplant. We therefore proceed to test several hypothesis for selection on unobserved variables outside the set included in our specifications.

5.2 Unobserved blood-type correlated variables hypothesis

When it comes to selection on unobserved variables, several options present themselves. A first possibility is that there are unobserved baseline variables correlated to people's blood types which affect survival. As discussed previously, evidence for overall differences in mortality across blood types is lacking, but may be different for the specific populations on the transplant waitlist.

We do not find the argument of correlated baseline confounding to be convincing on the basis of pre-transplant survival patterns in relation to transplant rates across blood-types. To illustrate this argument, Figures 3a-3b present differences in transplant probabilities alongside differences in pre-transplant survival. Figure 3c further shows the modelled pre-transplant effect of O vs B blood types and 3d shows that same effect for

[31] More specifically, there was an emphasis on matching kidneys from the top 20% healthiest donors (KDPI< 20%) to the candidates in the top 20% of estimated post-transplant survival potential.



(a) No covariates

(b) Offer Covariates

(c) All Covariates

(d) Post-2015

Figure 2: Pre-transplant difference in survival effect β_z

Based on selected sample of Scientific Registry of Transplant Recipients data described in Appendix B.
(a) includes no covariates, (b) includes offer covariates: age bracket dummies, (c-d) include all variables presented in Appendix Table A1. Observations (c): $N_{O:no-Tr} = 93,922$, $N_{O:Tr} = 35,989$, $N_{AB:no-Tr} = 5125$, $N_{AB:Tr} = 5032$. Observations (d): $N_{O:no-Tr} = 43,338$, $N_{O:Tr} = 11,698$, $N_{AB:no-Tr} = 2241$, $N_{AB:Tr} = 2176$.

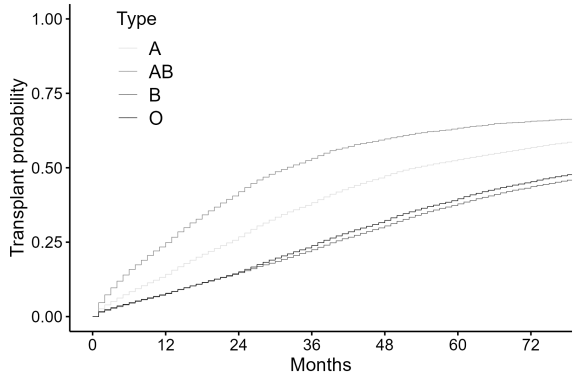
O vs A blood types. Taken together, we notice there is a one-to-one ordinal relation between the transplant probabilities, pre-transplant survival rates, and modelled effects. The higher the transplant probability, the lower the pre-transplant survival probability. These differences cannot be attributed to differences in racial compositions between blood types since, as presented in Figure A2 of Appendix C, the results on the sample of White people follow the same patterns.

For this relation over the duration on the waitlist to be causally determined by intrinsic characteristics of candidates with different blood types, we would need to make a strong assumption. We would need to assume that these period $t = 0$ baseline unobserved variables simultaneously affect variation in transplant rates and covariation in pre-transplant survival rates. This seems unlikely due to the source of variation in transplant rates.

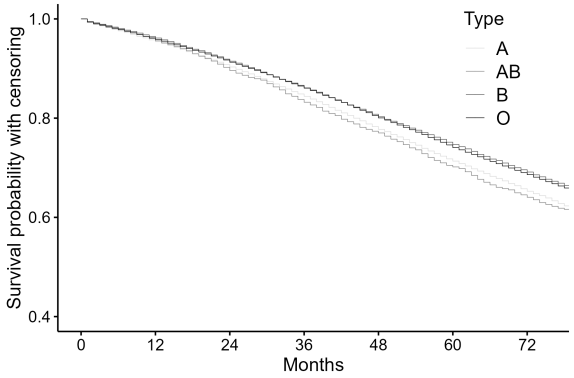
In most cases, kidneys from deceased donors are gathered, after family consent, from people who experienced severe brain bleeding, have been declared brain dead, or for whom cardiac death is imminent. While not impossible, it seems difficult to find convincing arguments why, even within racial groups, certain blood types are more prone to specific accidents making them eligible for kidney donation, more prone to die of natural deaths while waiting for a kidney, and that these distinct forms of death form a one-to-one ordinal relation with respect to blood type in the data. A more likely explanation is that differences in transplant rates across blood types are influencing the type and share of candidates receiving a transplant. This selection in turn determines which candidates remain on the waitlist which influences the measured pre-transplant survival. We explore different hypotheses along these lines in the remaining results.

5.3 Lifestyle change behavioural hypothesis

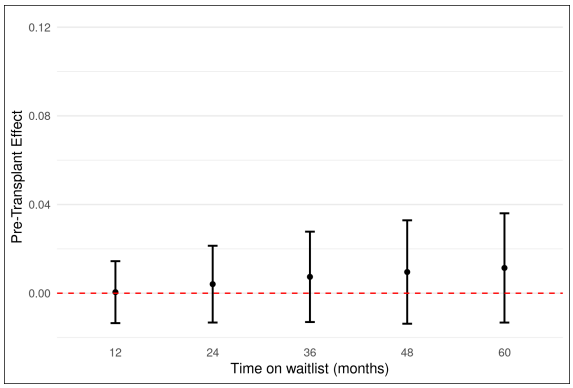
In the remaining sections we turn to incentive-based explanations for the observed pre-transplant patterns. In this section, we assume optimising forward looking agents within a standard dynamic discrete choice search model (Mortensen, 1986). In this first model, we assume candidates will accept a kidney if its quality is above an individual specific threshold, and also assume this threshold is invariant to the kidney offer rate. We extensively discuss features of the model in appendix E. In short, candidates wish to survive each period but face biological pressures. These pressures can be influenced to some degree by adopting a more or less healthy lifestyle. Each rational candidate forms a reservation survival value before receiving a transplant which is a function of their observed and unobserved (health) types, the mental and physical costs of remaining alive, the arrival rate of health shocks, the probability of receiving a transplant and the rational expected benefit of receiving a transplant. Also, while candidates know the distribution of health shocks they receive each period, they do not perfectly foresee or control these health shocks. A candidate's life ends upon receiving a negative health shock above their reservation survival threshold. From the setup of the model, it follows that poor health



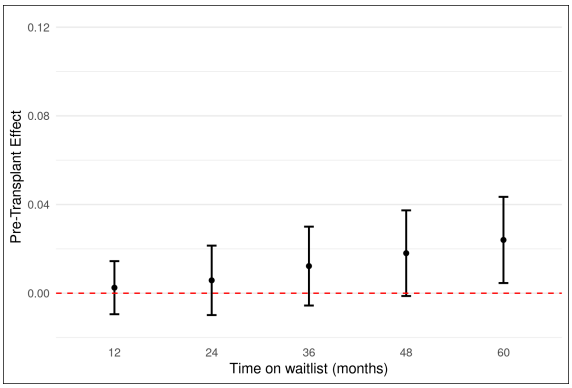
(a) Transplant Probability



(b) Pre-Transplant Survival



(c) Pre-Transplant: O vs. B



(d) Pre-Transplant: O vs. A

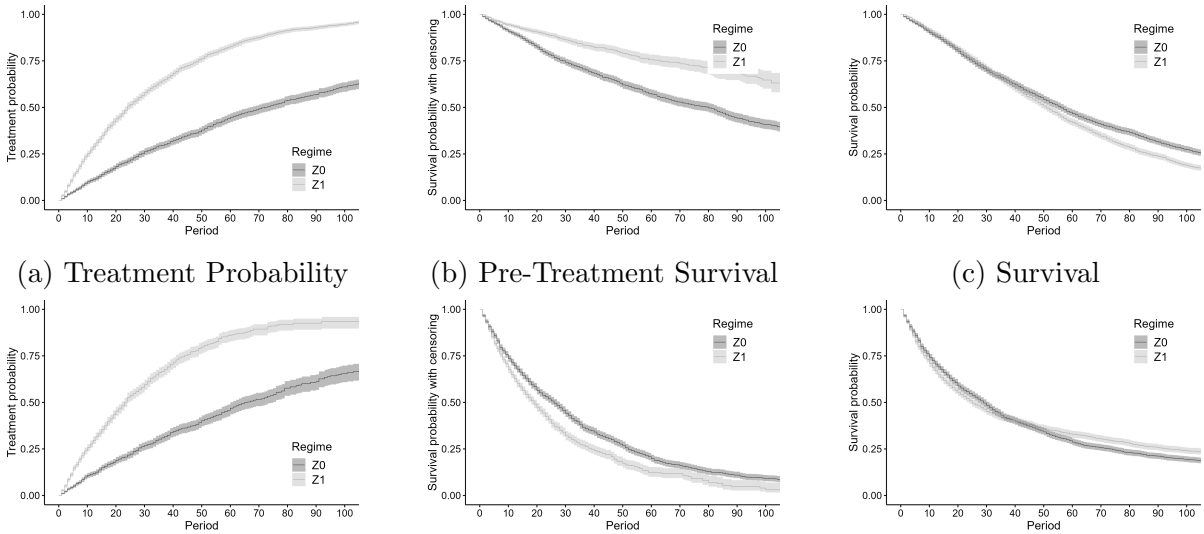
Figure 3: Treatment probability and Pre-transplant survival effects β_z for A and B blood type candidates

Based on selected sample of Scientific Registry of Transplant Recipients data described in Appendix B.
(c-d) include all variables presented in Appendix Table A1. Observations (c): $N_{O:no-Tr} = 93,922$, $N_{O:Tr} = 35,989$, $N_{B:no-Tr} = 28,432$, $N_{B:Tr} = 10,303$. Observations (d): $N_{O:no-Tr} = 93,922$, $N_{O:Tr} = 35,989$, $N_{A:no-Tr} = 50,005$, $N_{A:Tr} = 31,637$.

candidates are more likely to succumb to a strong negative health shock.

In the model discussed in this section, we assume differences in the kidney offer rate across blood types and changes in the offer rate over the duration on the waitlist only affect candidate's survival by changing their reservation survival threshold. This can be understood as agents adapting their health lifestyle in response to the signalled availability of kidneys. We show in appendix E that if candidates on the waitlist foresee a kidney transplant to improve their utility of surviving, and they have a high probability of rapidly receiving a kidney transplant (AB blood type), then they will take on a more healthy lifestyle in order to stay alive before receiving the transplant.

As can be observed in the simulated results of Figures 4a-4c, the model predicts a higher pre-transplant survival rate for AB blood-types (Z1), which is contrary to the observed patterns in the data. The pre-transplant survival and overall survival patterns in the data can, in fact, only be reproduced in the model if we assume a negative shock from the transplant which is foreseen by candidates, as shown in Figures 4a-4c. Thus, neo-classical assumptions must be replaced by behavioural ones if lifestyle and health adjustments are to explain the pre-transplant survival patterns. For instance, moral hazard in response to higher kidney offer rates could explain the lower pre-transplant survival among AB-blood types. While we cannot exclude some lifestyle adjustments to differences in offer rates, we show in the following section that the assumptions presented in this section alone cannot explain certain survival patterns for candidates who receive a transplant.



(a) Treatment Probability (b) Pre-Treatment Survival (c) Survival

(d) Treatment Probability (e) Pre-Treatment Survival (f) Survival

Figure 4: Simulations with (a-c) positive and (d-f) negative treatment effects
Data generating process and simulation details presented in Appendix E. Observations (a-c): $N_{Z=1:no-Tr} = 1657$, $N_{Z=1:Tr} = 822$, $N_{Z=0:no-Tr} = 738$, $N_{Z=0:Tr} = 1783$. Observations (d-f): $N_{Z=1:no-Tr} = 1919$, $N_{Z=1:Tr} = 560$, $N_{Z=0:no-Tr} = 1488$, $N_{Z=0:Tr} = 1033$.

5.4 Kidney quality threshold change behavioural hypothesis

In this section we turn to an alternative incentive-based explanation based on differential changes in kidney quality thresholds. This explanation can operate alongside the lifestyle adjustment hypothesis but can also explain the pre-transplant results in the absence of any moral hazard. We can show that the pre-transplant differences displayed in Figure 1 will arise if candidates with lower health are relatively more selective about which kidneys to accept than candidates with higher health when facing a higher kidney offer rate.

To illustrate this mechanism, consider a forward looking agent model as described in the previous section, but assume reservation survival thresholds do not change in response to differences in the kidney offer rate. Instead, we assume only that candidates' kidney quality acceptance thresholds change, and allow this change to be different depending on underlying unobserved health. Because there is uncertainty around what size a health shock a candidate will receive each period, their decisions must balance a tradeoff. 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 low health candidates, meaning they will in expectation benefit more from an equal time wait than good 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 good health candidates. If we assume expectations and choices are rational, this results, on average, in higher overall (transplanted + non-transplanted) survival among low health candidates in the AB-blood type regime than in the O-blood type regime. This average increase in survival is driven by kidney transplant recipients who delayed their decision to accept a kidney but eventually found a higher quality kidney than they would have had they been in the lower kidney offer rate regime. This delayed decision, however, also carries the risk of mortality which translates into a lower pre-transplant survival in the AB regime than in the O-regime

despite a higher overall survival.[32]

5.4.1 Testing differential survival effects for transplanted at $t = 1$

The above theory leads to some testable implications in our setting. Recall that, conditional on kidney allocation mechanism variables, assumption $A.Ii$ implies that kidney offers are distributed equally among higher and lower health candidates. As such, if the acceptance to offer ratio is lower for poor health candidates, 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 than in the O-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. This conclusion rests on one of two assumptions concerning information acquisition. If we assume candidates are the main decision makers of which kidney to accept, then we must assume candidates rapidly integrate the offer rate into their decision making process. If clinicians and transplant centers are the central decision makers on which kidney to accept, then we must assume they are aware and integrate differences in offer rates depending on a candidate's blood type when accepting kidneys.

We test the theory of non-uniform selection into receiving a transplant using a simple linear probability model on the sample of candidates who receive a transplant within the first month(s) on the waitlist.

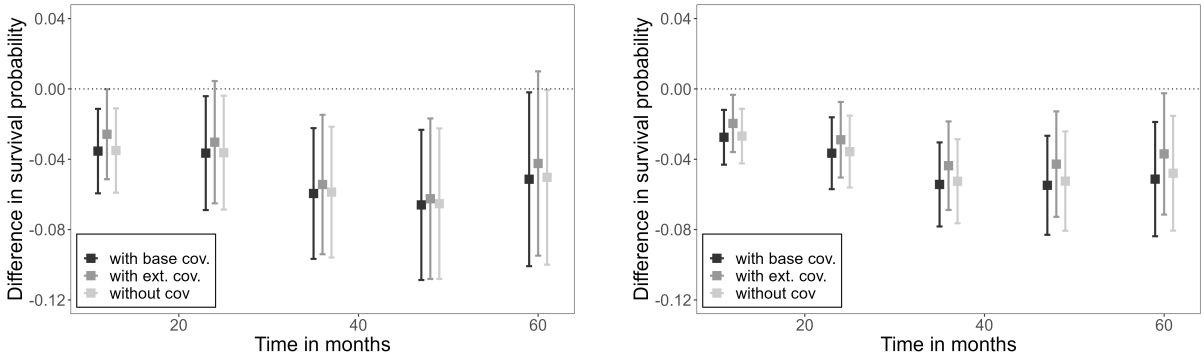
$$\mathrm{Y}_i = \alpha + \delta\, \mathrm{Z}_i + \beta\, \mathrm{X} + u_i \tag{3}$$

In this model, Y_i is an indicator for survival past a certain duration τ. Z_i is the blood type indicator with $\mathrm{Z}_i = 1$ for a O-type candidates and $\mathrm{Z}_i = 0$ for AB-type candidates. X, depending on the specification, includes all variables relevant to the transplant allocation mechanism, as well as other prominent mortality related variables. The parameter of interest, δ represents the difference in the probability of survival past τ for candidates receiving a transplant within the first month(s) on the waitlist. Note that we cannot compare treated across regimes at later time periods since, due to the previously described dynamic selection, there will be relatively fewer surviving positively selected AB blood type candidates than O blood type candidates remaining on the waitlist. Appendix B columns 5-6 present descriptive statistics for the O and AB blood type candidates receiving a transplant. Based on the age and diabetes covariates, we already note selection on observed variables in line with the hypothesis that unhealthier candidates in the AB

[32] It is worth noting that if the probability of accepting a kidney is decreasing in unobserved health, then the observed pre-transplant patterns of Figure 1c can only arise if the decrease in the acceptance to offer ratio for lower health candidates relative to that of higher health candidates is sufficiently large to offset the scale effect of more lower health candidates receiving a transplant in the higher offer rate regime. As we discuss in our results later in this section, this issue is irrelevant in our setting since the probability of accepting a kidney appears to be increasing in unobserved health (see results section 5.4.2).

group are more likely to wait for a kidney.

Figures 5a-5b show results from this model using different time intervals for defining an early transplant. Figure 5a presents results when defining the treatment window as all people who receive a transplant within the first month on the waitlist. Figure 5b defines it as those receiving a transplant within the first three months. As theoretically predicted, the results show that AB-blood types receiving a transplant in the first month(s) on the waitlist have a higher probability of surviving than O-blood types over a 4-5 year time horizon. These results therefore support the theory of that kidney acceptance thresholds for low and high health candidates change do not change uniformly when facing differences in the kidney offer rate.



(a) Treatment in month 1 on waitlist

(b) Treatment in month 1-3 on waitlist

Figure 5: Difference in survival between AB and O blood types at different time intervals for candidates treated in first 1 to 3 months on the waitlist

Based on selected sample of Scientific Registry of Transplant Recipients data described in Appendix B. (*without cov*) includes no covariates, (*with base cov*) includes offer covariates: age bracket dummies, (*with ext. cov*) includes all variables presented in Appendix Table A1. Observations (a): $N_O = 2089$, $N_{AB} = 463$. Observations (b): $N_O = 3757$, $N_{AB} = 955$.

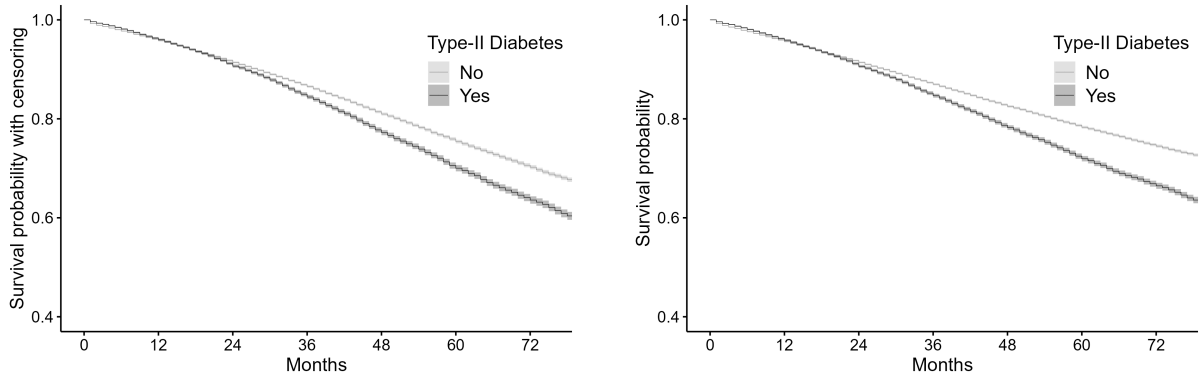
5.4.2 Testing differential probability of transplant at $t = 1$

In this section we further test the theory of more selective kidney acceptance among poor health candidates. To do so, we make use of a proxy for poor unobserved health which is not included in the set used to determine kidney allocations. This chosen proxy is whether a candidate's primary reason for needing a kidney transplant is due to Type-II diabetes. As shown in Figure 6a-6b, candidates with Type-II diabetes have a lower pre-treatment survival and a lower overall survival than other candidates on the waitlist.

With this proxy, we can estimate the probability of receiving a transplant in the first three months on the waitlist using the following linear probability heterogenous effect model,

$$\mathrm{D}_i = \beta_0 + \beta_Z \, \mathrm{Z}_i + \beta_W \, \mathrm{W}_i + \beta_{ZW} \, \mathrm{Z}_i \cdot \mathrm{W}_i + \beta_X \, \mathrm{X} + u_i \tag{4}$$

where D_i is a transplant indicator, with $\mathrm{D}_i = 1$ for a candidate receiving a transplant in the first month(s) and $\mathrm{D}_i = 0$ otherwise. W_i is a Type-II diabetes indicator with $\mathrm{W}_i = 1$ for candiates with Type-II diabetes listed as the main reason for a transplant upon entering

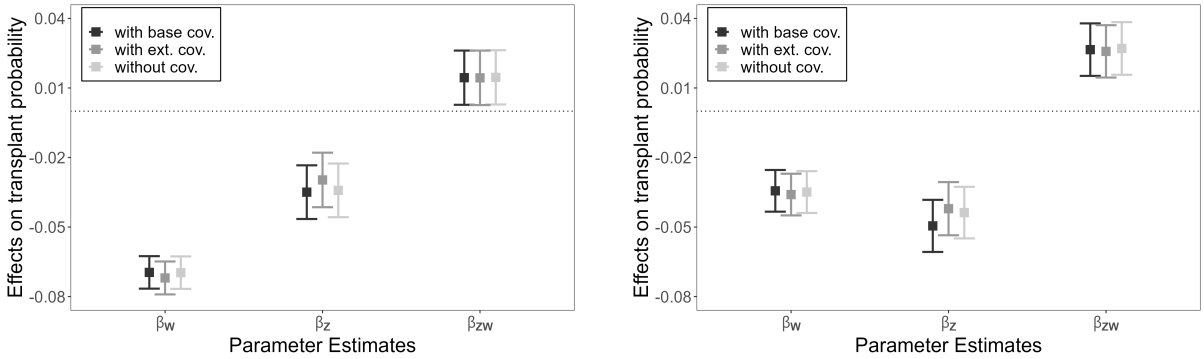


(a) Pre-Transplant Survival

(b) Survival

Figure 6: Survival of candidates on the kidney transplant waitlist depending on Type-II diabetes status

Based on selected sample of Scientific Registry of Transplant Recipients data described in Appendix B. Observations: $N_{t-IIDiab} = 104,490$, $N_{not-IIDiab} = 39,805$.



(a) Old kidney allocation system

(b) New kidney allocation system

Figure 7: Heterogenous effects of blood type and type-II diabetes on probability of transplant within first 3 months on the waitlist

Based on selected sample of Scientific Registry of Transplant Recipients data. Selected sample is described in section 4. (*without cov*) includes no covariates, (*with base cov*) includes offer covariates: age bracket dummies pre-2015, and dummies for all EPTS variables post-2015. (*with ext. cov*) includes all variables presented in Appendix Table A1. Observations (a): $N_{O:no-Tr} = 93,922$, $N_{O:Tr} = 35,989$, $N_{B:no-Tr} = 28,432$, $N_{B:Tr} = 10,303$. Observations (b): $N_{O:no-Tr} = 93,922$, $N_{O:Tr} = 35,989$, $N_{A:no-Tr} = 50,005$, $N_{A:Tr} = 31,637$.

the waitlist, $W_i = 0$ otherwise. In the model we control for all variables relevant in the kidney offer allocation mechanism. Because of this, under $A.Ii$ and given the results of section 5.2, blood-types can be considered randomly allocated and we can credibly interpret the differences in the acceptance of kidney transplants as causal across blood-type regimes. β_0 represents the probability of transplant in the first three months on the waitlist for AB-type candidates without type-II diabetes. β_Z represents the difference in transplant probability for O-types without type-II diabetes. β_W is the difference in transplant probability for AB-types with type-II diabetes relative to AB-types without type-II diabetes. β_{ZW} measures any additional difference in the transplant probability for O-types with type-II diabetes relative O-types without type-II diabetes.

The hypothesis in this section predicts that β_Z should be negative since, given the lower kidney offer rate, the probability of transplant should also be lower for O-blood

types. It also predicts that β_W should be negative if unhealthier candidates with type-II diabetes are more selective about which kidney to choose and therefore delay their decision to accept a kidney. And, finally, the behavioural hypothesis put forward also predicts that β_{ZW} will be positive if, given the lower offer rate in their regime, unhealthy O-type candidates with type-II diabetes are relatively less selective about which kidney to accept than unhealthy AB-type candidates with type-II diabetes. All of these predictions are supported by the results presented in Figure 7.

Taking all the results from this section and previous ones together, our study appears to strongly corroborate with theories of forward looking behavioural changes in response to differences in the offer rate. Were these to be true, they would have important implications on the optimal redesign of kidney transplant mechanisms given the observed differences in pre-transplant survival across blood-type groups. Despite the seemingly ample evidence for the offer-rate response behavioural explanation, the next section shows that these same pre-transplant and period $t = 1$ results can arise mechanically without any adaptive forward looking adjustment to differences in the kidney offer rate.

5.5 Unobserved mechanical selection hypothesis

A final hypothesis which can explain pre-transplant effects does not require any differential responses to the kidney offer rate depending on unobserved variables. We can show that the observed pre-transplant patterns will arise under particular statistical conditions, which are likely to arise in the US kidney transplant setting.

Let us first assume agents are not sophisticated, meaning they do not adjust their behaviour in response to changes in the kidney offer rate as in the previous two sections. Thus, the kidney quality acceptance threshold is invariant to the offer rate across blood-type regimes. We further assume that pre-transplant survival is increasing in variables not relevant in the kidney allocation mechanism. As discussed in previous sections, these may be unobserved health factors known by the candidate and their clinicians, but not relevant in the kidney offer allocation mechanism or held as major mortality variables.

Under this assumption, the pre-transplant empirical patterns documented in Figure 1 of the introduction and the modelled effects of Figure 2 will only arise if the probability of accepting a transplant is also increasing (for early stages on the waitlist) in unobserved health variables.

Table 1 presents a simple stylised one period example to gain intuition for these survival dynamics. 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 good 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 present 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 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 the pre-transplant survival $\beta_z = 1/9$ is higher in the O-blood regime as in our 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 present 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 our kidney setting and our main results.

Table 1: Survival Dynamics in Different Acceptance/Offer Scenarios

	cand.	offered kidneys	accepted kidneys	pre-transpl. survival	pre-transpl. survival ratio	β_z
$\partial \Pr(\text{accept kidney}\|U=u)/\partial u > 0$						
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		

5.5.1 Alternative justification for effects of transplanted at $t = 1$

The hypothesis in this section can also give rise to the transplant effect differences in Figure 5 and the heterogenous treatment effect results in Figure 7. Given the above

described selection, and as noted in Table 1, AB and O blood type populations in both regimes are only comparable in terms of unobservables at $t = 0$. For $t > 0$ there will be relatively fewer surviving positively selected AB blood type candidates than O blood type candidates on the waitlist due to dynamic selection.[33] However, even if candidates are comparable in terms of unobservables, we can show that the results in Figure 5 can be explained without assuming candidates or clinicians adapt to changes in the offer rate.

Assuming candidates and/or clinicians make a simultaneous decision over several kidney offers, and that there are fewer kidney offers in the O-blood type regime, then the expected value of the chosen maximum quality kidney will be lower in the O-blood type regime. A simple proof for this result in presented in Appendix F. These dynamics will give rise to the negative effects documented in Figure 5 simply because O-blood candidates are less likely to be offered, and accept, higher quality kidneys. Based on Agarwal et al. (2021), a crude estimate for the average number of kidney offers per candidate a month is around 17-18. If the trend in the transplant hazards in Figure 1a are a close proxy to the trend in kidney offer rates on the waitlist, the average numbers are slightly higher in initial months. Because candidates and/or transplant centers receive offers some days in advance, it is indeed likely some decisions are made jointly over multiple kidneys.

5.5.2 Alternative justification for probability of transplant at $t = 1$

We can also show that the results on the probability of accepting a kidney at $t = 1$ in Figure 7 will arise in both previously discussed scenarios without behavioural reactions to changes in the kidney offer rate. Take first the scenario in which clinicians are more likely to offer a kidney to higher health candidates than lower health candidates. The consequence of this skewed distribution can be illustrated with the kidney acceptance numbers in Table 1 for the relevant case in which the probability of accepting a kidney is increasing in unobserved health (top frame). β_Z, the difference in the probability of accepting a kidney between O-blood and AB-blood type candidates with high health (no type-II diabetes) is given by $1h/5h - 3h/5h = -2/5$. β_W, the difference in the probability of accepting a kidney between low health (type-II diabetes) and high health (no type-II diabetes) candidates in the AB-blood type regime is also for our stylised numbers given by $1h/5h - 3h/5h = -2/5$. β_{ZW}, the additional difference between low health (type-II diabetes) and high health (no type-II diabetes) candidates in the AB-blood type regime is given by $(0l/5l - 1l/5l) - (1h/5h - 3h/5h) = 1/5$. These results replicate the patterns estimated in Figure 7.

In appendix F.1 we further show that the same coefficient signs for β_Z, β_W and β_{ZW} can arise for the second scenario in which clinicians do not offer more kidneys to higher health candidates but lower health candidates are more likely to reject offered

[33] Recall that we assume treatment occurs before exit each period t. Thus, this statement holds as long as we assume a finite population of candidates on the waitlist. Also, in contrast to the previous section, this selection is not linked to selection due to changing responses to a higher kidney offer rate.

kidneys. Interestingly, an additional requirement to replicate coefficient patterns in Figure 7 for this scenario is that the distribution of offered kidneys in terms of quality is right skewed, meaning there are more undesirable than desirable kidneys. Evidence for this phenomenon is well known and outlined in (Agarwal et al., 2018, 2020).

All in all, this section shows that under certain conditions, the pre-transplant survival differences, and differences in effects documented in Figures 5 and 7 can result directly from positive selection in who receives a kidney transplant. These results do not require candidates to react differently to higher or lower offer rates.

5.6 Which hypothesis does the data generating process and institutional setting support?

The previous section suggests several explanations consistent with the pre-transplant empirical patterns. In contrast to previous behavioural explanations, these hypotheses concerning candidate and clinician or transplant center decisions also imply some constraints on the data generating process.

The first two assumptions to appraise are whether lower health candidates are more likely to reject offers and whether clinicians offer more kidneys to higher health candidates. Several features of the kidney waitlist suggest both to be true in the current design of the US transplant system. In particular, candidates 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, these sub-optimal kidneys. If we assume, as seems plausible, that being informed about an offer of a sub-optimal kidney leads to higher rejection independent of underlying health, we would expect a higher rejection rate from low-health individuals.

From the side of transplant centers, the financial cost of transplants for lower health candidates have been shown to be higher in absolute and marginal terms after medicare compensation (Axelrod et al., 2017, 2018). Financial incentives also create a chain reaction. If transplant centers are less likely to accept sub-optimal kidneys, then the 56 organ procurement organizations - government-chartered nonprofits that collect, test and disseminate organs in their regions - also have lower financial incentives to retrieve these kidneys (Kimberly et al., 2018). This reinforcing process would lead to more kidney offers for higher health individuals.

Beyond these tangible incentives, there is also suggestive evidence of bias in decision-making on behalf of candidates and clinicians in transplant centers. A first piece of evidence is from Zhang (2010) who shows that, for two kidneys from a same donor, the probability of rejecting a kidney increases with the amount of previous rejections when one kidney was randomly rejected more often in initial offers. This suggests the number of rejections, rather than the actual quality of a kidney, is sometimes erroneously used as

a heuristic for rejection decisions by either the patient, the transplant center, or both.

Lastly, recent changes in the measures used to evaluate transplant center performance and initiate official enquiries also pre-suppose a bias in the decision making process for transplant acceptance on behalf of clinicians or transplant centers. Prior to July 2003, transplant center assessments were solely based on a 1-year post-transplant risk-adjusted candidate survival score. This score indicated the relative survival success of transplanted candidates over one year post-transplant adjusting for the donor kidney quality, the candidate health, and the interaction between the two. Because the risk-adjustment score is conditional on donor quality and candidate health, it should not, in principle, have incentivized transplant centers to favour healthier kidneys or healthier candidates. Yet, since July 2023, publicly displayed performance reviews and official enquiries for transplant centers are additionally based on a risk-adjusted offer acceptance rate. In addition, after July 2024 an additional score will be implemented based on the pre-transplant mortality of candidates on a transplant center's waitlist.[34]

These developments, explicitely focused on reducing the excessive waste of viable kidneys, suggest OPTN is attempting to nudge transplant centers to focus more on the mortality and outcomes of less healthy candidates.[35] While the adjustments may partially be attempting to counterbalance other financial incentives, there is also reason to believe they are attempting to counteract behavioural biases in the understanding of the 1-year risk adjustment scores. When it comes to candidate and clinician decisions based on observed metrics, evidence suggests, unsurprisingly, that both are partial to healthy kidneys (Mohan et al., 2018; King et al., 2023).[36] More problematically, anecdotal reports suggest clinicians misinterpret the risk-adjusted 1-year survival score and replace it with the simpler notion of a 1-year survival score (Mohan et al., 2018; Kimberly et al., 2018), which would erroneously incentivize transplant centers to favour healthier kidneys and healthier candidates. As yet, there exists no literature on candidate and clinician decision making and behavioural biases in the context of transplants.

6 Conclusion

Kidney discard rates in the US, nearing 25%, far exceed those of other high-income countries. France 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). Part of the reason for this discrepancy is the unusual transparency in the US system, leaving room for discretion in deciding which organs to accept in the hands of transplant

[34] We further discuss the risk adjusted score model and some of its potential pitfalls in appendix G.

[35] See discussion of the Membership and Professional Standards Committee here and formal information guidelines about change here.

[36] There is also existing evidence of transplant centers attempting to game UNOS/SRTR evaluations (Stith and Hirth, 2016).

centers and candidates on waitlists (Rasmussen et al., 2018).

The results in this paper outline different hypotheses of selection and, in the process, underline which behaviours of clinicians and candidates can realistically be driving the excess discard of viable kidneys. Little is known about which of these behaviours is the leading cause, and important insights could be gained from behavioural research on decision making among the two groups. A common theme does, however, seem to emerge from our research, which is that large amounts of viable kidneys are lost due to misaligned expectations of clinicians and/or candidates, and poor financial incentives for transplant centers. An important policy proposal emerging from this work would be to develop a statistical system in order to re-align expectations. This model could, for each offered kidney, outline the expected amount of time any given candidate within a transplant center can expect to wait until receiving a kidney at least as good as the proposed one.[37] This type of system can easily be built adjacent to the current score models and conveyed to transplant centers and candidates alike. Such new measures, alongside the new performance scores introduced by OPTN in 2023-2024, and other adjustments to the kidney allocation system (Senate hearing: UNOS), may also go to some length in counterbalancing any financial disadvantages of focusing on less health kidneys or candidates.

Our AB vs. O analysis also offers a new insight independently of the question of excess kidney discards and the main cause driving differences in pre-transplant survival. When it comes to the equity of the kidney allocation mechanism, our results indicate that the higher offer rate for AB-blood type candidates allows them to receive higher quality kidneys in the first months on the waitlist which results in longer post-transplant survival.

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[37] An open question is whether, in addition, a candidate specific expected survival rate over that same time period should be presented.

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APPENDIX

A Identification of Causal Effects

In our derivation of identification results we maintain the overlap assumptions in A.II throughout and apply SUTVA/consistency A.III whenever converting potential variables to observed variables. All identification results remain the same when there is censoring if we assume censoring always occurs before treatment and exit at time t, and censored observations are dynamically missing at random. We derive effects in discrete time $t \in \mathbb{N}$ and $s \in \mathbb{N}$.

Proof of Proposition 1 and Corollary 1:

Using the dynamic unconfoundedness assumption A.I, we can straightforwardly derive all causal effects from the general form:

$$\begin{aligned}
\Pr(T^{z,\infty} > \tau) &= \Pr(T^{z,\infty} > \tau | S > \tau, Z = z) \\
&= \prod_{t=0}^{\tau} \Pr(T > t | S > t, T \geq t, Z = z) \\
\Pr(T^{1,s} > \tau) &= \Pr(T^{z,s} > \tau | S = s, Z = z) \\
&= \prod_{t=s}^{\tau} \Pr(T > t | S = s, T \geq t, Z = z) \cdot \prod_{t=0}^{s-1} \Pr(T > t | S > t, T \geq t, Z = z) \\
\Pr(S^z = s) &= \Pr(S^z = s | Z = z) \\
&= \Pr(S = s | S \geq s, T \geq s, Z = z) \cdot \prod_{t=0}^{s-1} \Pr(S > t | S \geq t, T \geq t, Z = z)
\end{aligned}$$

Proof of Proposition 2:

Under assumptions A.I-A.IV, with $T^{1,\infty} \geq T^{0,\infty}$, $\Pr(T^{0,\infty} > \tau | cs_s) = \Pr(T^{0,s} > \tau | cs_s) = 0$ and all probabilities for never-survivors are equal to 0. We then have that,

$$\begin{aligned}
\beta_0 &= \Pr(T^{0,\infty} > \tau) = \Pr(T^{0,\infty} > \tau | as_s) \cdot \Pr(as_s) \\
&= \Big(\prod_{t=s}^{\tau} \Pr(T > t | S > t, T \geq t, Z = 0) \Big) \cdot \Pr(as_s) \\
\beta_s &= [\Pr(T^{0,s} > \tau) - \Pr(T^{0,\infty} > \tau)] = [\Pr(T^{0,s} > \tau | as_s) - \Pr(T^{0,\infty} > \tau | as_s)] \cdot \Pr(as_s) \\
&= \Big(\prod_{t=s}^{\tau} \Pr(T > t | S = s, T \geq t, Z = 0) - \prod_{t=s}^{\tau} \Pr(T > t | S > t, T \geq t, Z = 0) \Big) \cdot \Pr(as_s) \\
\Pr(as_s) &= \Pr(T^{1,\infty} \geq s, T^{0,\infty} \geq s) = \prod_{t=0}^{s-1} \Pr(T > t | S > t, T \geq t, Z = 0) \\
\Pr(cs_s) &= \Pr(T^{1,\infty} \geq s, T^{0,\infty} < s) = \prod_{t=0}^{s-1} \Pr(T > t | S > t, T \geq t, Z = 1) - \Pr(as_s) \\
\Pr(ns_s) &= 1 - \Pr(as_s) - \Pr(cs_s)
\end{aligned} \tag{5}$$

In these derivations it is useful to note that, for example, $\prod_{t=0}^{s-1} \Pr(T > t|S > t, T \geq t, Z = 0) = 1$ for always-survivors.

Under the additional exclusion restriction A.V we have that $\Pr(T^{1,s} > \tau|as_s) = \Pr(T^{0,s} > \tau|as_s)$ from which we can identify $\Pr(T^{1,s} > \tau|cs_s) = [\Pr(T^{1,s} > \tau) - \Pr(T^{1,s} > \tau|as_s) \cdot \Pr(as_s)]/\Pr(cs_s)$. In addition, assuming that for complier-survivors defined at s, $T^{1,\infty} < \tau$ if $\tau >> s$, it follows that $\Pr(T^{1,\infty} > \tau|cs_s) = 0$. Under these restrictions, we can identify all remaining effects,

$$
\begin{aligned}
\beta_z &= [\Pr(T^{1,\infty} > \tau) - \Pr(T^{0,\infty} > \tau)] = [\Pr(T^{1,\infty} > \tau|as_s) - \Pr(T^{0,\infty} > \tau|as_s)] \cdot \Pr(as_s) \\
&= \Big(\prod_{t=s'}^{\tau} \Pr(T > t|S > t, T \geq t, Z = 1) - \prod_{t=s}^{\tau} \Pr(T > t|S > t, T \geq t, Z = 0) \Big) \cdot \Pr(as_s) \\
\beta_{zs} &= [(\Pr(T^{1,s} > \tau) - \Pr(T^{1,\infty} > \tau)) - (\Pr(T^{0,s} > \tau) - \Pr(T^{0,\infty} > \tau))] \\
&= \Pr(T^{1,s} > \tau|cs_s)] \cdot \Pr(cs_s) - [\Pr(T^{1,\infty} > \tau|as_s) - \Pr(T^{0,\infty} > \tau|as_s)] \cdot \Pr(as_s) \\
&= \prod_{t=s}^{\tau} \Pr(T > t|S > t, T \geq t, Z = 1) - \prod_{t=s}^{\tau} \Pr(T > t|S > t, T \geq t, Z = 0) \cdot \Pr(as_s) - \beta_z
\end{aligned}
\tag{6}
$$

B Descriptive Statistics and Data selection

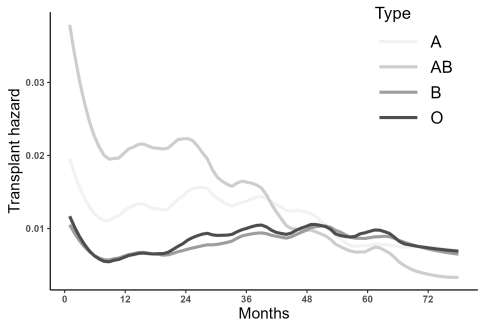
Table A1: Descriptive Statistics

	Pre-treatment Analysis				Treated at $t = 1$	
	O	AB	B	A	O	AB
Age	51.32	51.91	51.56	51.90	49.07	**51.31**
	(13.06)	(13.02)	(12.83)	(13.08)	(13.94)	**(13.57)**
$Transp$	0.10	0.12	0.09	0.12	0.11	0.09
	(0.30)	(0.32)	(0.29)	(0.32)	(0.31)	(0.29)
$Diabetes$	0.35	0.37	0.37	0.36	0.25	**0.32**
	(0.48)	(0.48)	(0.48)	(0.48)	(0.43)	**(0.47)**
$Dialysis$	0.92	0.94	0.92	0.93	1.00	1.00
	(0.27)	(0.24)	(0.27)	(0.26)	(0.04)	(0.06)
BMI	28.41	28.40	28.37	28.47	27.44	27.81
	(5.73)	(5.85)	(5.80)	(5.75)	(5.49)	(5.54)
$Female$	0.39	0.38	0.38	0.38	0.39	0.36
	(0.49)	(0.49)	(0.49)	(0.48)	(0.49)	(0.48)
$PrevMalign$	0.06	0.06	0.05	0.07	0.06	0.06
	(0.24)	(0.24)	(0.23)	(0.25)	(0.24)	(0.24)
$Acpt_{HepB}$	0.56	0.54	0.57	0.54	0.48	0.47
	(0.50)	(0.50)	(0.49)	(0.50)	(0.50)	(0.50)
$Acpt_{HCV+}$	0.06	0.05	0.07	0.05	0.09	**0.07**
	(0.24)	(0.21)	(0.25)	(0.22)	(0.29)	**(0.25)**
$White$	0.62	**0.55**	**0.45**	**0.70**	0.78	**0.68**
	(0.48)	**(0.50)**	**(0.50)**	**(0.46)**	(0.42)	**(0.47)**
$Asian$	0.05	**0.12**	**0.12**	0.05	0.03	**0.07**
	(0.22)	**(0.32)**	**(0.33)**	(0.22)	(0.17)	**(0.26)**
$Black$	0.30	0.32	**0.41**	**0.23**	0.18	**0.24**
	(0.46)	(0.47)	**(0.49)**	**(0.42)**	(0.39)	**(0.43)**
$Other$	0.02	0.01	0.02	0.02	0.01	0.01
	(0.15)	(0.12)	(0.12)	(0.13)	(0.11)	(0.09)

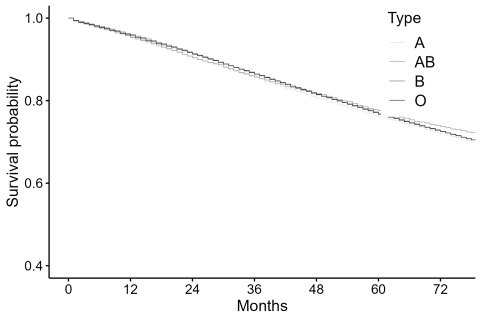
Means for each group presented with standard deviations in parenthesis. We mark in bold any normalized difference relative to the O blood-type group greater or equal to 0.1. Full sample includes 159,150 observations. Age: Candidate age at listing, $Transp$: Received previous transplant (other than kidney transplant), $Diabetes$: Had Diabetes upon entering the waitlist, $Dialysis$: On Dialysis upon entering the waitlist, BMI, $Female$, , $PrevMalign$: Any previous Malignancy, $Acpt_{HepB}$: Will accept an Hepatitis B Core Antibody Positive Donor?, $Acpt_{HCV+}$: Will accept an HCV Positive donor?, $White$: Race-White, $Asian$: Race-Asian, $Black$: Race-Black, $Other$: Race-Other Non-White

Main analysis data selection:

Our initial data contains 1,010,051 observations. We remove candidates with no activation date (954,406), and select only people set to receive a kidney transplant (894,372). We then select for our initial analysis only candidates entering the waitlist between June 1st 2002 and December 1st 2014 (440,926). Among these we only keep the first observed kidney transplant (360,171), for candidates who are over 18 (349,831). We further remove candidates with unusual A1, A2, A1B, A2B blood types (344,882). In the AB vs. O blood types analysis, we also remove the A and B blood types (182,394). We drop candidates with a positive cPRA (170,966) and drop candidates who received a transplant from a living donor (145,274). All remaining reductions in sample for the analysis with covariates result from missing values in the covariate matrix. These in one or the other specification include: $< 1\%$ of missing values for education, $< 0.01\%$ of missing values for BMI, $< 2.8\%$ of missing values for previous malignancy. We take December 1st 2014 as the 2014 reform cutoff date.



(a) Hazard rate for all blood types

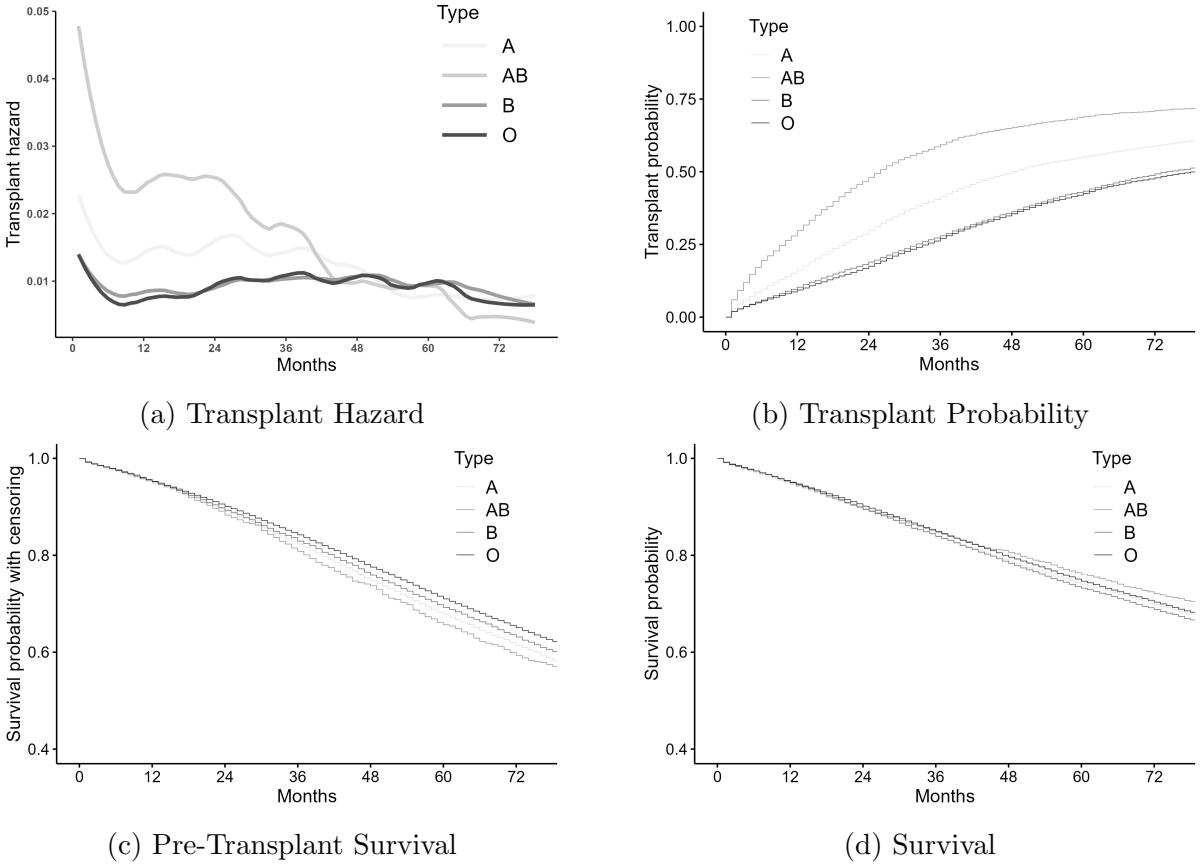


(b) Survival for all blood types

Figure A1: Hazard and overall survival for different blood types

Based on selected sample of Scientific Registry of Transplant Recipients data described in Appendix B. Observations: $N_O = 134,730$, $N_{AB} = 10,544$, $N_B = 40,217$, $N_A = 84,929$.

C Additional Figures on subgroups



(a) Transplant Hazard

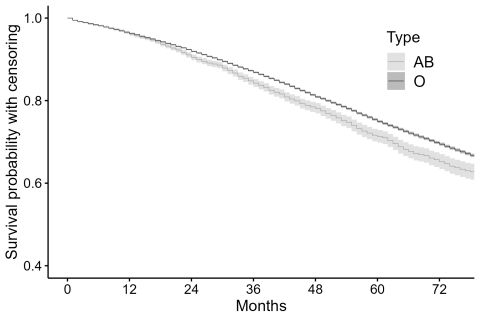
(b) Transplant Probability

(c) Pre-Transplant Survival

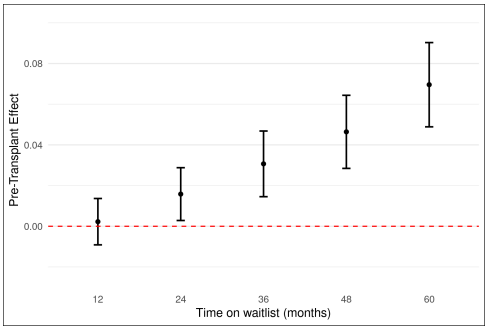
(d) Survival

Figure A2: Hazard, transplant and survival probabilities for different blood types conditional on being White

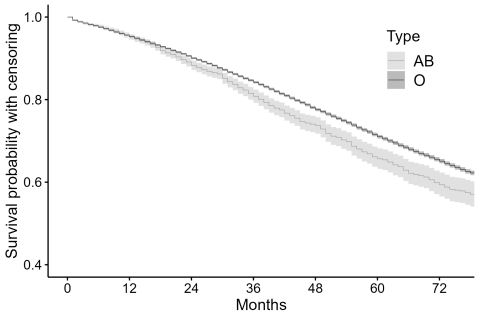
Based on selected sample of Scientific Registry of Transplant Recipients data described in Appendix B. Observations: $N_O = 80,058$, $N_{AB} = 5385$, $N_B = 16,830$, $N_A = 56,877$.



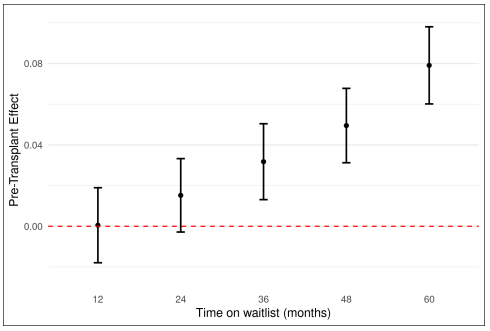
(a) Survival including living donors



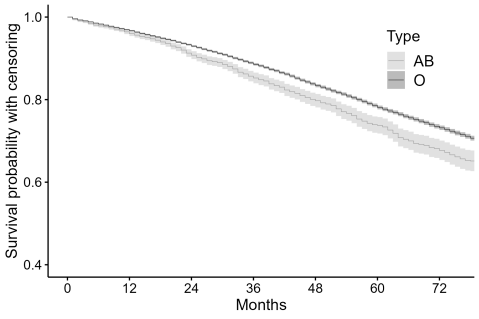
(b) Effects including living donors



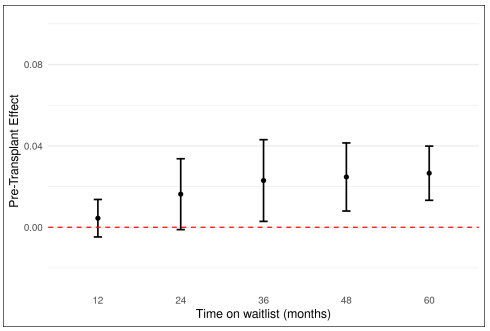
(c) Survival for white people



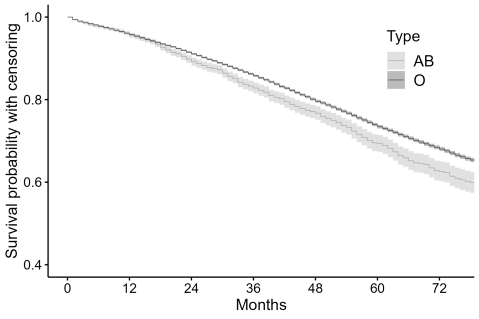
(d) Effects for white people



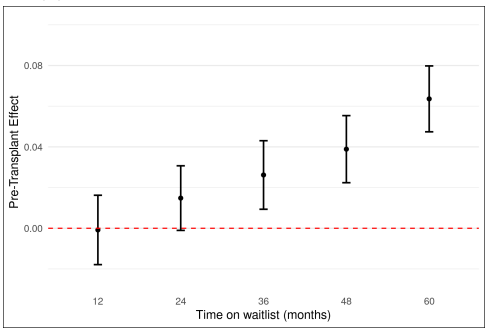
(e) Effects for non-white people



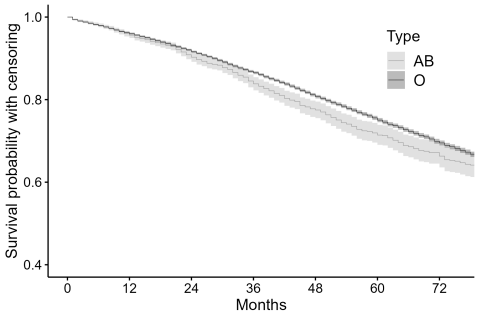
(f) Survival for non-white people



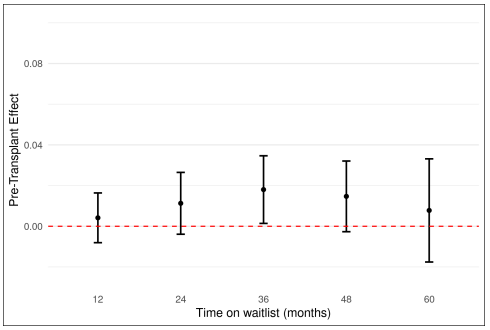
(g) Survival for males



(h) Effects for males



(i) Survival for females



(j) Effects for females

Figure A3: Pre-transplant survival and effect β_z for different subsamples. Based on selected sample of Scientific Registry of Transplant Recipients data described in Appendix B.

D Robustness Checks

Table A2: Causal Effect Decomposition

	(1)	(2)	(3)	(4)	(5)
β_0	0.690	0.679	0.760	0.733	0.713
	(0.043)	(0.150)	(0.104)	(0.133)	(0.138)
	[0.000]	[0.000]	[0.000]	[0.000]	[0.000]
β_z	0.053	0.065	0.073	0.011	0.024
	(0.013)	(0.010)	(0.019)	(0.013)	(0.010)
	[0.000]	[0.000]	[0.000]	[0.365]	[0.015]
β_s	0.171	0.187	0.130	0.101	0.126
	(0.020)	(0.075)	(0.050)	(0.042)	(0.050)
	[0.000]	[0.013]	[0.009]	[0.016]	[0.012]
β_{zs}	-0.094	-0.108	-0.099	-0.021	-0.046
	(0.014)	(0.043)	(0.038)	(0.011)	(0.019)
	[0.000]	[0.012]	[0.009]	[0.062]	[0.017]
$N_{Z=0:no-Tr}$	5125	5125	2241	28,432	50,005
$N_{Z=0:Tr}$	5032	5032	2176	10,303	31,637
$N_{Z=1:no-Tr}$	93,922	93,922	43,338	93,922	93,922
$N_{Z=1:Tr}$	35,989	35,989	11,698	35,989	35,989

Standard errors in parenthesis. P-values in brackets. Effect is estimated over 5 years (τ). Each column presents ex-post transplant effects values $\beta_{s=1}$ and $\beta_{z,s=1}$ for transplant in the first two months. Column (1) presents pre Dec. 2014 decomposition with $Z = 0$: AB-blood types, $Z = 1$: O-blood types, without covariates. Column (2) presents pre Dec. 2014 decomposition with $Z = 0$: AB-blood types, $Z = 1$: O-blood types, with all covariates. Column (3) presents post Dec. 2014 decomposition with $Z = 0$: AB-blood types, $Z = 1$: O-blood types, with all covariates. Column (4) presents pre Dec. 2014 decomposition with $Z = 0$: B-blood types, $Z = 1$: O-blood types, with all covariates. Column (5) presents pre Dec. 2014 decomposition with $Z = 0$: A-blood types, $Z = 1$: O-blood types, with all covariates.

E Dynamic discrete choice model of candidate behaviour

To relate our dynamic treatment effect model to the literature on dynamic discrete choice models we present in this appendix a standard job search model. We then extend it to include a treatment. We loosely adapt the discussion of the search model to our kidney transplant application. For expositional purposes, the model is solved under simplistic assumptions. We then explain how the dynamic discrete choice model relates to the parameters in our proportional hazard model in Section 3.3. Thereafter we describe the data generating process and present simulation results for our estimator.

E.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 our 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 behaviour 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 our 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 our empirical setting to a randomization set by nature but may also be a rule imposed by a policymaker. In our 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 of by specifying a search model of offered kidneys for the accepted kidney. We do not add this layer of complexity since we wish to convey insights for setting of section 5.3 in which the acceptance/offer rates are invariant to the number of offers.

In our 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 our 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,

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

We can solve this model for the reservation utilities after and before treatment,[38]

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

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 our 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. Our 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 our empirical results with the biological predictions of the model is to allow a candidate's behaviour 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 our 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 our dynamic discrete choice search model above.

[38] Full derivation of solutions are presented in subsection E.4

E.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 $\pi_{Z=0} = Pr(S = s|S \geq s, Z = 0) = 0.01$. We impose that the treatment takes place before the exit decision in period t.

Using this model we 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, we 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 we simulate expectations of w_{it} over 1000 draws and iterate over the value function until convergence.

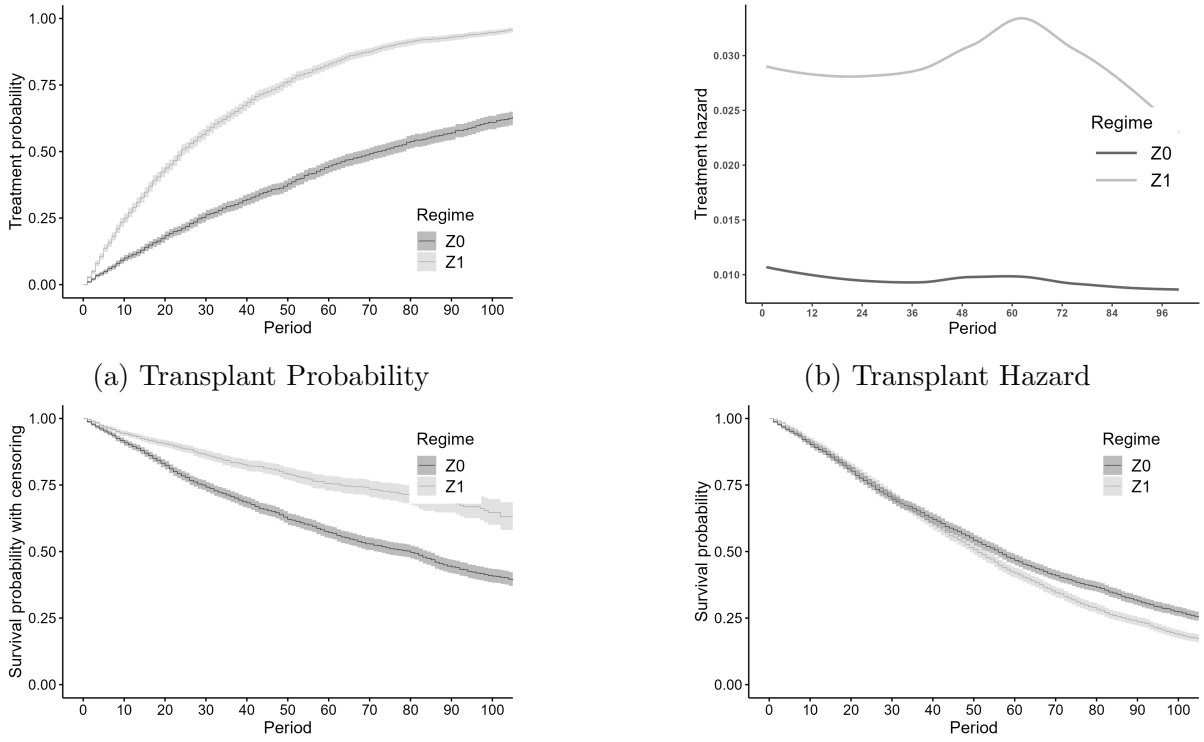
Table A3: Parameter choices in simulations

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

E.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, we 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 we censor all observations greater than $t = 60$ ($\sim$ 43.7%) and apply random right censoring to $\sim$ 6.3% of the remaining observations. We present descriptive survival curves and hazards in Figure A4 in the case of a positive treatment effect and in Figure A5 in the case of a negative treatment effect.



(a) Transplant Probability

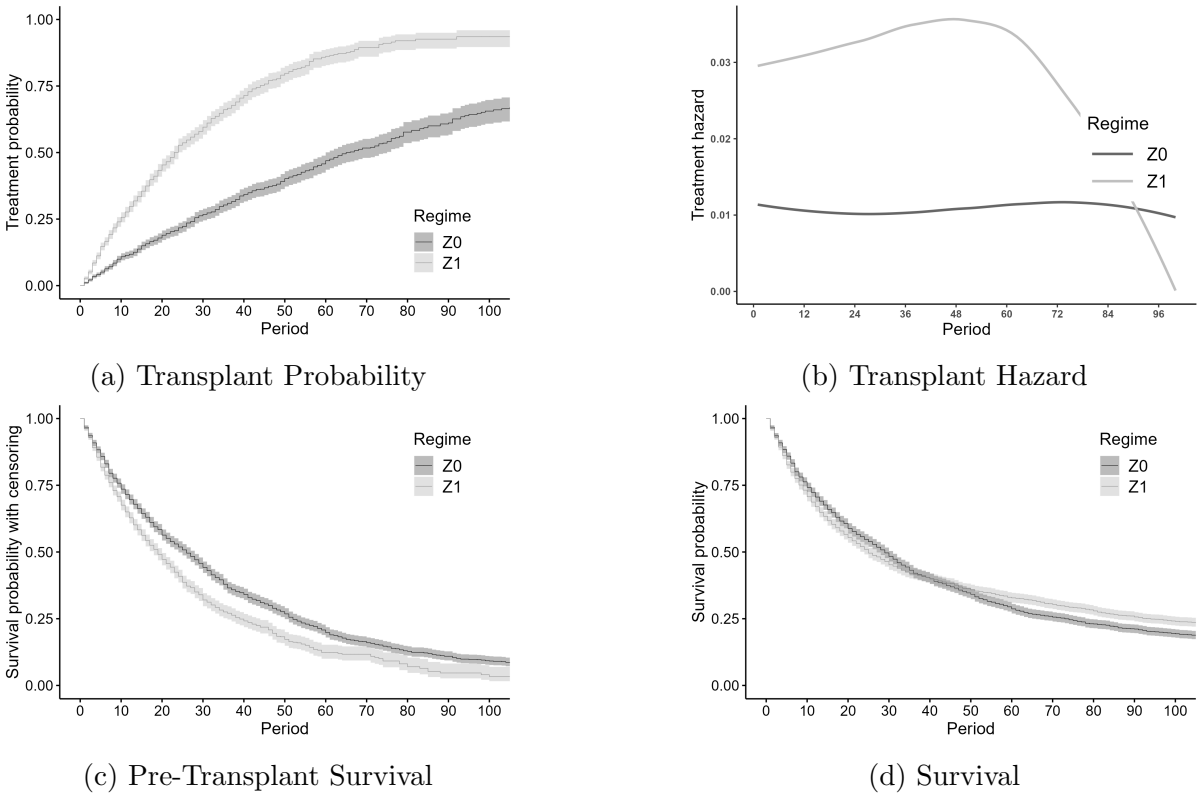
(b) Transplant Hazard

(c) Pre-Transplant Survival

(d) Survival

Figure A4: 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. $N_{Z=0:no-Tr} = 1013$, $N_{Z=0:Tr} = 453$, $N_{Z=1:no-Tr} = 441$, $N_{Z=1:Tr} = 1093$.

Table A4 shows the results when applying our estimation method. We present the average effects for β_0, β_z, $\beta_{(0,s]}$, $\beta_{z(0,s]}$ over the treatment times $s = 1,\ldots,30$ when specifying the duration dependence to six 10 period intervals. The estimator performs relatively well with all sample sizes observed. Figure A6 provide further descriptions on the performance of our estimator. They present the estimates of β_0, β_z, β_s, β_{zs} with τ fixed at 60 over the first 30 periods using a sample of 3000 observations from the full population of 5000. The estimator seems to fit the data very well for all causal estimates, and in the case of β_0, β_s, β_{zs}, also match closely the DGP values.



(a) Transplant Probability

(b) Transplant Hazard

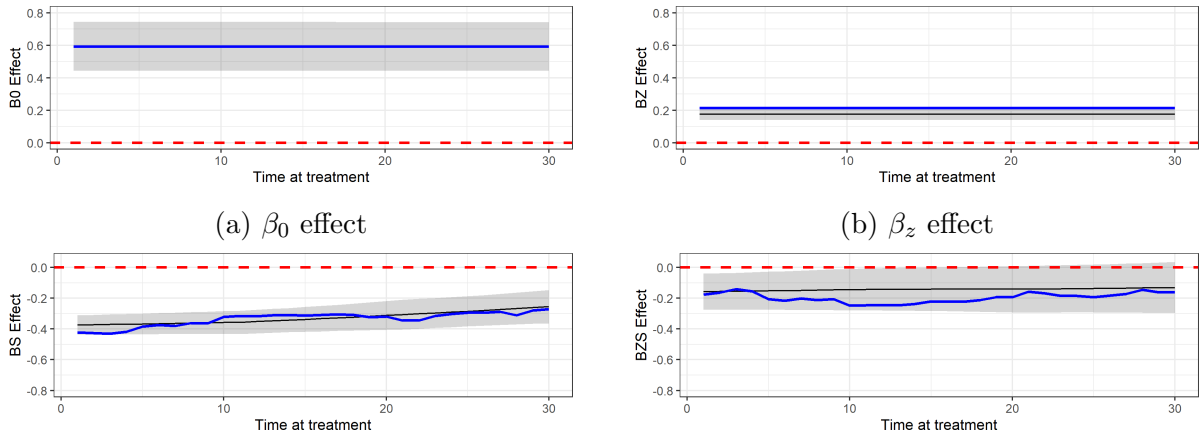
(c) Pre-Transplant Survival

(d) Survival

Figure A5: 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. $N_{Z=0:no-Tr} = 1013$, $N_{Z=0:Tr} = 453$, $N_{Z=1:no-Tr} = 441$, $N_{Z=1:Tr} = 1093$.

Table A4: Simulation results of dynamic discrete choice model, s from 1,. . . ,30 and $s+\triangle$ fixed at 90

	Estimates	Bias	Variance	MSE
N=5000				
β_0	0.590	0.001	0.006	0.006
β_z	0.170	-0.043	0.000	0.002
$\beta_{(0,s]}$	-0.297	0.040	0.002	0.003
$\beta_{z(0,s]}$	-0.180	-0.021	0.004	0.004
N=3000				
β_0	0.593	0.002	0.006	0.006
β_z	0.175	-0.039	0.000	0.002
$\beta_{(0,s]}$	-0.337	0.000	0.002	0.002
$\beta_{z(0,s]}$	-0.144	0.057	0.005	0.008
N=1000				
β_0	0.618	0.027	0.005	0.006
β_z	0.177	-0.036	0.000	0.002
$\beta_{(0,s]}$	-0.322	0.014	0.003	0.003
$\beta_{z(0,s]}$	-0.211	-0.010	0.004	0.004
N=500				
β_0	0.602	0.011	0.006	0.006
β_z	0.203	-0.011	0.001	0.001
$\beta_{(0,s]}$	-0.324	0.013	0.003	0.003
$\beta_{z(0,s]}$	-0.251	-0.050	0.007	0.010



(a) β_0 effect (b) β_z effect

(c) β_s effect (d) β_{zs} effect

Figure A6: Assessing performance of empirical evaluation model
Based on selected sample of Scientific Registry of Transplant Recipients data. Selected sample is described in section 4. $N_{Z=0:no-Tr} = 1013$, $N_{Z=0:Tr} = 453$, $N_{Z=1:no-Tr} = 441$, $N_{Z=1:Tr} = 1093$.

E.4 Reservation utilities in dynamic discrete choice model

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

$$V_{0,t} = w_0 - c + \rho\lambda\mathbb{E}_w[\max\{V_1(w), (1-\pi)V_{0,t+1} + \pi V^{tr}_{0,t+1}\}] + \rho(1-\lambda)[(1-\pi)V_{0,t+1} + \pi V^{tr}_{0,t+1}]$$
$$V^{tr}_{0,t} = w_0 - c + \rho\lambda\mathbb{E}_{w^{tr}}[\max\{V^{tr}_1(w), V^{tr}_{0,t+1}\} + \rho(1-\lambda)V^{tr}_{0,t+1}$$

This second equation can be written as,

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

Since all parameters and distributions in the model are time independent, we have a stationary reservation utility strategy. In a stationary strategy $V^{tr}_{0,t} = V^{tr}_{0,t+1} = V^{tr}_0$ 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^{tr}_0 if $w^{tr} < w^{tr*}$, and $V_1(w^{tr*}) = \frac{w^{tr*}}{1-\rho} = V^{tr}_0$ if $w = w^{tr*}$. It follows that,

$$\begin{aligned} V^{tr}_0 &= w_0 - c + \rho\lambda \int_{w^{tr*}}^{\infty} \left(V_1(w^{tr}) - V^{tr}_0\right) dG^{tr}(w) + \rho V^{tr}_0 \\ &= w_0 - c + \frac{\rho\lambda}{1-\rho} \int_{w^{tr*}}^{\infty} \left(w - w^{tr*}\right) dG^{tr}(w) + \frac{\rho w^{tr*}}{1-\rho} \end{aligned}$$

Replacing again $V^{tr}_0 = \frac{w^{tr*}}{1-\rho}$, rearranging this equation and using integration by parts we obtain the post-treatment reservation utility,

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

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

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

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 $\int_{w^*}^{+\infty} w - w^* dG(w)$,

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

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

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

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.

F Proof that accepted kidney quality is increasing with non-sequential kidney offers

Our 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 $i \in 1, ..., 100$. 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 nth 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.

F.1 Replicating patterns of Figure 7 when higher and lower health candidates have different offer to acceptance rates

We wish to show here that the same coefficient signs for β_Z, β_W and β_{ZW} can arise for the second scenario in which clinicians do not offer more kidneys to higher health candidates but lower health candidates are more likely to reject offered kidneys.

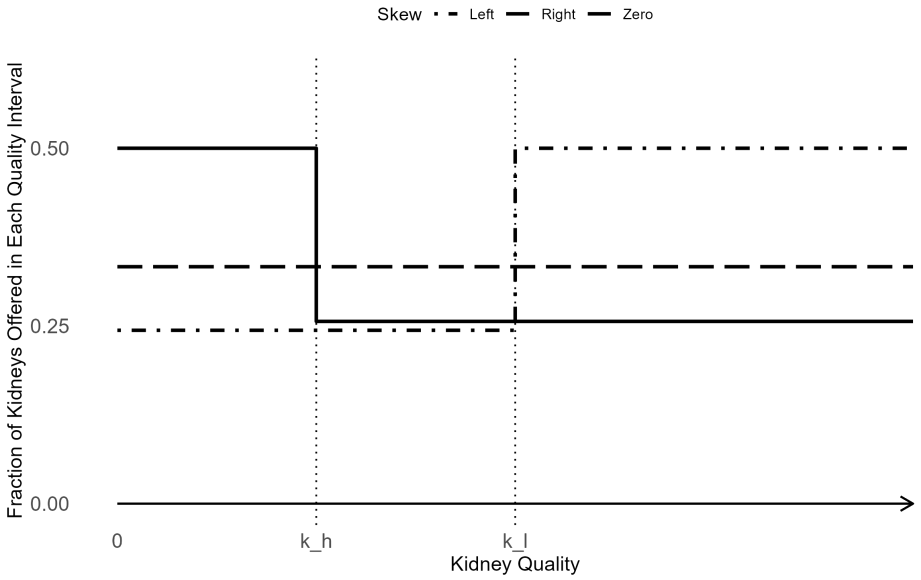


Figure A7: Distribution of offered kidney scenarios

Consider the case in which 2 kidneys are offered in the O-blood regime and 4 are offered in the AB-blood regime. Assume as well for simplicity that clinicians randomly allocate these kidneys regardless of candidate health. As such, higher and lower health candidates can each expect to receive half the kidneys offered. Higher health candidates will further accept any kidney above the threshold k_h while lower health candidates are more selective only accepting kidneys above some threshold k_l. These thresholds can be thought as normalised with respect to the quality of kidneys offered. So, even if in absolute terms both higher and lower health candidates may have the same rejection threshold, the differing thresholds can reflect the lower average quality kidneys offered to lower health candidates. Also assume that when multiple kidneys are offered, they are offered simultaneously.

Take first the case of a right skewed distribution as described in Figure A7. In expectation, O-type higher health candidates will accept $\frac{1}{2}$ offers while AB-type higher health candidates will accept $3 \cdot \frac{1}{2} \cdot \frac{1}{2} = \frac{3}{4}$ offers. Similarly, O-type lower health candidates will accept $\frac{1}{4}$ offers while AB-type lower health candidates will accept $2 \cdot \frac{1}{4} \cdot \frac{3}{4} + \frac{1}{4} \cdot \frac{1}{4} = \frac{1.75}{4}$ offers. As a result, we would obtain $\beta_Z = \frac{2}{4} - \frac{3}{4} = -\frac{1}{4}$, $\beta_W = \frac{1.75}{4} - \frac{3}{4} = -\frac{1.25}{4}$ and $\beta_{ZW} = (\frac{1}{4} - \frac{2}{4}) - (\frac{1.75}{4} - \frac{3}{4}) = \frac{0.25}{4}$. As we can see, the coefficient patterns follow those in Figure A7. Similar calculations will show that in the zero-skew case effects are 0 and in the left-skewed case the coefficient patterns are inversed.

G 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 nto enter the calculation.

Some systematic issues raise concern about the precision of 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, reducing incentives for transplant centers to ignore lower health patients, 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 concerning for the reliability of the model.