Federal Oversight and Strategic Choices of Kidney Transplant Centers *

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Abstract

Kidney transplant centers significantly influence patient survival, yet regulatory oversight of their performance and practices remains limited. This study evaluates a policy designed to penalize centers whose post-transplant mortality exceeds risk-adjusted thresholds. Using variation in policy exposure across centers and novel follow-up data, I employ a difference-in-difference approach to estimate the policy's impact on patient outcomes and center behaviors. The policy reduced post-transplant mortality by 18 - 24%, though the mechanisms driving the improvements evolved over time. Initially, centers responded by performing fewer transplants, thereby avoiding high-risk kidney matches. Over time, transplant volume recovered as centers adapted. They prescribed more potent immunosuppressants to reduce the risk of rejection and intensified patient monitoring to manage side effects. These findings demonstrate that centers initially reduced transplants due to unfamiliarity regarding policy nuances but subsequently shifted toward actively improving post-transplant care.

JEL codes: I11, I18, L38

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1 Introduction

Transplant centers are crucial in helping the 100,000 patients on the national waitlist obtain a kidney transplant and recover from kidney failure. Despite receiving significant reimbursements from the Centers for Medicaid and Medicare Services (CMS) [1], there was limited oversight of center behavior and performance until high-profile issues, such as poor patient outcomes and inefficiencies, came to light in 2005 [2]. These concerns prompted the announcement of a federal oversight program (GAO, 2008). The program enabled CMS to evaluate and guide transplant centers in identifying areas for quality improvement and enhancing the efficiency of care delivery. However, the accompanying financial penalties for poor performance can introduce unintended incentives. For instance, to avoid penalties, centers may cherry-pick patients by prioritizing those with lower-risk profiles, potentially leading to kidney wastage and denying transplants to patients who might benefit the most (Sack, 2012).

This paper examines the effects of federal oversight on post-transplant mortality and treatment decisions. I leverage exogenous variation in penalty exposure created by one of the most extensive oversight programs in the US deceased donor kidney transplant system. Specifically, I study CMS's Conditions of Participation (CoP) policy, announced in February 2005 and implemented in July 2007. The policy penalizes transplant centers for having risk-adjusted post-transplant mortality rates exceeding specified limits. Post-transplant mortality, defined as death or graft failure within 365 days after the transplant, carries significant consequences under the CoP, as centers can lose certification if penalized more than twice over 30 months (Federal Register, 2007). Given CMS's status as the largest purchaser of organ transplantation services, the threat of withdrawal commands immediate attention from center leadership (Hamilton, 2013).

Centers could respond to the threat of punishment in two ways. First, as policymakers intended, centers could improve post-transplant care. For example, acute kidney rejection, the most common post-transplant complication (Gjertson et al., 2002), can be mitigated by intensifying im-

[1] CMS spent $36 billion in 2017 on the care of renal failure patients, with approximately 13% allocated to kidney transplants (Sawani, 2019).

[2] Source: Kaiser puts kidney patients at risk.

munosuppressive regimens and dedicating more resources to monitoring and managing side effects. Secondly, centers may engage in selective behaviors, altering patient or kidney composition to reduce mortality rates. The Organ Procurement and Transplantation Network (OPTN) informs the center of biologically compatible kidneys, but administrators retain discretion over accepting or declining the kidney offers. The CoP's penalties may influence decisions for marginal patient-kidney pairs, as noted by a director in a 2012 *New York Times* article: "*... if you have had a couple of bad outcomes recently you say, 'Well, why should I do this?'... You can always find a reason to turn organs down...*" [3]. These potential trade-offs make oversight policies particularly controversial in kidney transplantation. To address these concerns, I investigate how much of the observed decline in mortality reflects improvements in post-transplant care versus the impact of selection mechanisms.

To motivate the empirical analysis, I consider a stylized model of center behavior to understand how federal oversight affects transplant decisions and post-transplant care. The center observes a noisy signal of patient health and decides whether to select the patient for transplant. Then, it provides post-transplant care. These decisions jointly determine the center's post-transplant mortality. CMS reimburses the center if mortality falls below a specified limit. The center aims to maximize profit by performing as many transplants as possible and providing comprehensive post-transplant care. However, it also faces tradeoffs: performing too many transplants increases the risk of exceeding mortality limits and incurring penalties, while excessive post-transplant care is costly for patients [4]. The model illustrates how the center optimizes these competing objectives. Under the CoP policy, the return to marginal transplants is reduced due to heightened performance scrutiny, while the return to improved post-transplant care increases, incentivizing a shift in behavior.

The primary data sources are administrative follow-up records for all transplant patients, comprehensive patient-kidney offers data, and CMS's CoP report. The dataset spans from 2001 to 2009, covering approximately four years before, two years after the 2005 CoP announcement, and two years after the 2007 implementation. The follow-up data tracks each transplant patient's health

[3] Source: New York Times

[4] For example, patients' coinsurance kicks in or increases the opportunity cost of the patient's time.

status and records prescriptions and medical tests performed during the revisits. The patient-kidney offer dataset records all kidney offers, including information on the final decision, offer dates, reasons for declining, and detailed patient and kidney characteristics. The CoP report documents the center's penalty status, highlighting key center-level characteristics and offering critical insights into how centers were evaluated under the CoP.

The research design exploits two sources of policy-driven variation. First, the announcement and delayed implementation affect centers differentially, creating cross-sectional variation in penalty beliefs. Second, the announcement introduces within-center temporal variation. Centers are not randomly assigned to the penalty, and the panel is crucial in eliminating constant unobserved differences across centers. This setting, therefore, lends itself to a difference-in-differences research design. I follow Gupta (2021) and construct a continuously varying measure of center expectations of exceeding the CoP threshold in the program's first year based on their past mortality and transplant volume. This approach leverages the fact that mortality rates are persistent over time, and hence, past performance is a valuable predictor of future penalty likelihood. This measure incorporates the intensive margin of the penalty incentive, i.e., centers with excellent recent performance are expected to have a lower likelihood of being penalized.

However, estimates obtained via ordinary least squares (OLS) using this measure could be biased upwards due to mean reversion (Chay, McEwan and Urquiola, 2005; Gupta, 2021). I circumvent this problem using an instrumental variable (IV) approach, thereby mitigating concerns about measurement error. The instrument is a predicted mortality rate based on patient-kidney factors estimated using transplant samples from 2002 to 2004. All else equal, centers with a higher proportion of these patients were more likely to be penalized [5]. The identifying assumption is that in the absence of CoP, centers with high versus low predicted mortality, held constant as in 2005, would evolve along parallel trends. To explore the validity of this assumption, I present nonpara-

[5] My estimates might still suffer attenuation bias due to important and potentially unobservable differences in patient composition across centers. To mitigate this issue, I leverage detailed follow-up data to compare outcomes for patients with similar observable characteristics transplanted at the same center before and after the CoP announcement. This approach isolates causal effects based on within-center changes in penalty beliefs. Where feasible, I further strengthen identification by incorporating patient-fixed effects, thus capturing within-patient variation over time.

metric estimates of dynamic effects on all key outcomes.

The baseline IV estimates imply that after CMS announced CoP, a one-standard-deviation increase in center belief resulted in a 2.78 percentage point (pp) (25%) decrease in post-transplant 1-year mortality. The pattern persisted even after CMS implemented CoP. OLS estimates are substantially smaller, consistent with downward bias due to mean reversion. This estimate will understate the aggregate effects of the penalty.

Applying the same research design, I examine how selection and improved post-transplant care influenced mortality across different policy phases. Initially, detailed patient-kidney offer data reveal that after CMS announced CoP, centers became 16% less likely to transplant a given patient-kidney pair. This cautious approach led to more high-risk kidneys being discarded, inadvertently reducing mortality due to fewer risky transplants. However, this selective behavior dissipated as CMS implemented CoP. Using follow-up data, I quantify subsequent improvements in post-transplant care during the implementation period. Centers became 7-13% more likely to prescribe the potent immunosuppressant, tacrolimus, to patients during follow-up revisits, complemented by increased patient monitoring. These clinical enhancements significantly reduced infection-related mortality, a key side-effect of the heightened tacrolimus regimen. These findings, combined with anecdotal evidence, indicate that initial policy uncertainty triggered cautious selection, but centers quickly adapted by restoring transplant volumes and markedly improving post-transplant care.

Several patterns suggest a causal interpretation of these results. First, there are no differential pretrends across centers at different levels of penalty risk. Second, the timing of the changes coincides with the announcement of the CoP policy. Third, I find statistically insignificant effects on otherwise similar outcomes that were not incentivized by the program, such as post-transplant mortality rates beyond the first year, diabetes, return to dialysis, wait time for transplant, and waitlist mortality. Fifth, the estimates are robust to alternative specification checks.

1.1 Related literature

This paper contributes to three main strands of literature. First, it engages with the economic debate on centralized quality disclosure [6]. Closely related studies, such as Dranove et al. (2003); Jin and Sorensen (2006); Bundorf et al. (2009); Ramanarayanan and Snyder (2012); Feng Lu (2012); Kolstad (2013); Gupta (2021); Vatter (2023), examine provider responses to such policies in healthcare contexts, including coronary artery bypass grafts, fertility clinics, nursing homes, hospital readmissions, and health plan ratings. My paper adds to existing work by identifying a transitory adjustment period during which uncertainty or adaptive behavior led to unintended short-term resource wastage. This finding highlights how government agencies can facilitate organizational learning and minimize unintended consequences during policy transition.

Second, this paper contributes to economic research on deceased donor organ transplants, which predominantly examines the design of allocation systems (Su and Zenios, 2005; Zhang, 2010; Bloch and Cantala, 2017; Agarwal, Hodgson and Somaini, 2020; Agarwal et al., 2021; Leshno, 2022; Doval et al., 2024; Sweat, 2024). Related work, such as Dickert-Conlin, Elder and Teltser (2019) and Bae (2024), investigates how state-level policies and changes to donor service area boundaries affect allocation and mortality rates. My paper adds to existing work by analyzing how federal oversight policy directly influences the behavior of transplant centers, highlighting the underexplored channel of post-transplant care and its impact on patient outcomes.

Third, this paper contributes to the literature on the causal effects of CoP by addressing limitations in previous studies that rely on cross-sectional variation in center penalty status (Schold, Arrington and Levine, 2010; Schold et al., 2013; Hamilton, 2013) or within-center temporal variation (White et al., 2014). Closely related is Stith and Hirth (2016), which employs a difference-in-differences design but focuses on centers transitioning in and out of treatment status, complicating causal interpretation. My paper contributes to existing work by utilizing novel follow-up data to highlight the impact of CoP on post-transplant care practices. Moreover, the 2.5-year gap between

[6] Dranove and Jin (2010) reviews the theoretical and empirical literature on quality disclosure. Their paper highlights various examples from healthcare, finance, and education.

CoP's announcement and implementation provides a unique opportunity to mitigate concerns about changing treatment status and anticipatory behavior, strengthening the credibility of causal inferences.

1.2 Roadmap

I organize the rest of the paper as follows. Section 2 describes the institutional details and the CoP policy. Section 3 describes the model. Section 4 describes the data. Section 5 describes the research design. Section 6 presents results on mortality and the various mechanisms at different CoP phases. Section 7 presents robustness checks of the main results. Section 8 concludes.

2 Institutional background

A patient diagnosed with end-stage renal disease (ESRD) has two options: dialysis or kidney transplant [7]. Dialysis requires two to three treatments a week. Sessions are time-consuming; patients can be infected if nurses do not disinfect stations appropriately after use. These disadvantages make kidney transplants the cheaper alternative (Matas and Schnitzler, 2004). In this paper, I focus exclusively on deceased donor kidney transplant that accounts for 60% of all kidney transplants in the U.S. (AKF, 2003) [8]. This section describes how patients are added to the waitlist, how the centralized system allocates kidneys, what post-transplant follow-up care entails, and the details of the Conditions of Participation (CoP). Figure I summarizes the patient experience on the deceased donor kidney transplant waitlist.

[Figure I about here.]

[7] Dialysis is a treatment that removes waste and excess water from the blood. There are two types of dialysis: hemodialysis and peritoneal dialysis.

[8] Kidney exchange is an alternative way of getting a kidney transplant (Roth, Sonmez and Unver, 2004). However, patients need a willing living donor, which can sometimes be logistically cumbersome. Hence, kidney exchange is considered a different program from a deceased donor kidney transplant.

2.1 Getting on the waitlist

The physician refers patients to a local transplant center when they have kidney failure [9]. The center's selection committee will evaluate if the patient is eligible for a kidney transplant (i.e., started dialysis or had a glomerular filtration rate (GFR) below 20mL per minute). The center will then register accepted patients on the national deceased donor waitlist and upload important information, such as immunological profiles, health conditions, and factors to be computed into the UNet system (AKF, 2003).

2.2 Kidney allocation and transplant process

The Organ Procurement and Transplantation Network (OPTN) designs and administers the centralized allocation process for deceased donor kidneys. Centers upload a deceased donor's medical history and organ condition into UNet when brain or cardiac death is imminent. The system identifies biologically compatible patients and ranks them according to their priority order. Many factors contribute to the order, including, but not limited to, blood type, duration on the waitlist, the patient's location, and, in some instances, weight and size compared to the donor.

Recovered kidneys become unsuitable for transplants after 24-36 hours. So, UNet simultaneously contacts multiple transplant centers about their compatible patients to speed up the matching process. When contacted, a transplant center has 1 hour to decide which patient receives the kidney offer. During this hour, surgeons receive information about the donor's medical history and can request additional information from the donor's hospital. At the same time, surgeons also evaluate their patients' health conditions and decide if the patient is eligible or suitable for the transplant. For example, the patient's condition might have deteriorated since the last evaluation, or the patient might be unavailable due to a family emergency. The transplant center does not contact every compatible patient due to the tight deadline [10]. It usually informs the patient after UNet confirms

[9] Patients usually follow the physician's recommendation because the local transplant center is logistically convenient and does not disrupt their dialysis routine (Schaffhausen et al., 2019). The average distance between a patient's home and the nearest center is 23 miles (Purnell and McAdams-DeMarco, 2020).

[10] Furthermore, no regulations mandate that transplant centers notify patients of their kidney offers (OPTN, 2023).

the center's acceptance (King et al., 2023; Husain et al., 2025)

If UNet receives multiple acceptances, the center with the highest-priority patient will receive the kidney. After receiving the kidney, the center conducts a final blood test using samples from both the patient and the donor [11]. Otherwise, the center declines the kidney offer, and UNet contacts the next center. UNet removes the patient from the waitlist 24 hours after a successful transplant. In the case of a declined kidney offer, the patient returns to the waitlist without any penalty on their priority for the next kidney offer (OPTN, 2023).

There are two channels through which the center affects the type of kidney its patients are matched with. First, the center can set acceptable donor criteria for each patient on UNet. For example, the center can limit the patient's maximum donor age to 80. As a result, kidneys from donors over the age of 80 will not be offered to the patient, even if they are biologically compatible. Second, due to the tight one-hour deadline, the center usually accepts or declines incoming kidney offers on the patient's behalf. I leverage the patient's acceptable donor criteria and patient-kidney offer data to examine how CoP affects these two channels.

2.3 Post-transplant care and acute kidney rejection

Centers typically discharge patients within 8 to 14 days post-transplant. After discharge, patients will visit the center for regular check-ups at defined intervals (e.g., 6 months, 1 year, 2 years, etc.) to monitor their recovery and kidney function.

Acute kidney rejection, an immune response typically occurring within the first 12 months post-transplant, is the most common post-transplant complication [12]. During rejection episodes, the patient's immune system, especially T-cells and antibodies, attacks the transplanted kidney, potentially leading to impairment and graft failure (Becker et al., 2022). To mitigate rejection risk, centers prescribe maintenance immunosuppressants, most commonly calcineurin inhibitors

[11] This blood test is called a serum crossmatch. It mixes the donor cells with the patient's blood to determine if the antibodies will bind to the donor cells and cause kidney damage. Source: Blood tests for transplant

[12] Approximately $15-20\%$ of transplanted patients will experience some degree of kidney rejection. Source: Cleveland Clinic.

(CNIs), such as cyclosporine and tacrolimus. These drugs inhibit calcineurin, preventing T-cell activation and subsequent immune response against the transplanted kidney (Lee, Myoung and Kim, 2023). Medicare Part B covers the patient's immunosuppressive drugs for the first 36 months post-transplant, after which Medicare will stop paying if the patient is under 65 years old and does not suffer from any disability [13].

In Section 6.3, I utilize follow-up data that tracks patient health outcomes and immunosuppressant prescriptions to evaluate how CoP impacts the center's post-transplant practices, particularly in terms of immunosuppressant prescribing patterns and the management of potential side effects.

2.4 Conditions of Participation (CoP)

Before July 2007, the OPTN was the primary organization responsible for monitoring a transplant center's number of post-transplant survival, but it only twice recommended to the Department of Health and Human Services that a transplant center's certification be removed citep Gao2008. Following several high-profile problems that came into light in 2005, CMS became concerned that the lack of severe penalties for poor performance may have led to a decline in the quality of kidney transplants [14].

CMS announced CoP in February 2005 and implemented it in July 2007. The policy provides a foundation to *(i) protect other potential Medicare beneficiaries who are waiting for organs for transplantation; (ii) establish sufficient quality and procedural standards to ensure that transplants are performed safely and efficiently; and (iii) reduce Medicare expenses by decreasing the likelihood that a transplant will fail* (Federal Register, 2005). Centers submit the 1-year post-transplant outcomes of a rolling 2.5-year cohort to the Scientific Registry of Transplant Recipients (SRTR) in the first week of every January and July [15]. CMS penalizes a transplant center for poor performance if all of the following criteria are satisfied:

[13] Patients pay the Part B deductible and a 20% coinsurance. Source: Medicare and anti-rejection drugs.

[14] Source: Los Angeles Times.

[15] Figure C.III in the appendix illustrates an example of a rolling 2.5-year cohort. The January 2011 submission (black box) consists of transplants from July 1, 2007, to December 31, 2009 (black line). Similarly, the July 2011 submission (red box) includes transplants from January 1, 2008, to June 30, 2010 (red line).

1. $O/E \geq 1.5$

2. $O - E \geq 3$

3. $Pr(O = E) \leq 0.05$

O is the center's observed number of patient deaths or graft failures within 1 year post-transplant; E is the center's expected number of patient deaths or graft failures within 1 year post-transplant. SRTR calculates E by estimating a Cox regression model (Cox, 1972), using all the transplants in the rolling 2.5-year cohorts submitted by each transplant center. The model utilizes extensive patient, donor, and match characteristics, including, but not limited to, age, race, diabetic status, donor cause of death, and human leukocyte antigen (HLA) matching. However, the model does not include center characteristics because *"center characteristics and practices may be associated with the differences we are trying to identify and therefore should not be risk-adjusted away."* (Dickinson et al., 2008). Criterion one states that the center's observed deaths have to exceed expected deaths by 50%. Criterion two states that the difference between the observed and expected deaths must be greater than 3. Finally, criterion three states that if observed deaths differ from expected deaths, the difference must be statistically significant at the 95% significance level. Intuitively, criteria one and two state that the center cannot have too many observed deaths; criteria three can be interpreted as CMS's attempt to protect low-volume transplant centers from statistical anomalies in patient deaths. For example, a patient death is more likely to push a low-volume center's OE death ratio in criteria one above the 1.5 limit compared to a high-volume center (Federal Register, 2005) [16].

Once CMS penalizes a center for poor performance, it implements a data-driven quality assessment and performance improvement (QAPI) system. If CMS identifies the center again within the next 30 months, it risks losing its program certification and Medicare funding. However, most centers have 210 days to appeal that their poor performance is due to mitigating circumstances. I present an example of a CoP report in Figure C.IV.

[16] I account for transplant volume and unadjusted mortality in Section 5.1 when constructing center penalty expectations.

3 Conceptual framework

In this section, I formalize the incentives of the transplant center and examine how CoP influences decision-making. I present a stylized model in which the center observes a noisy signal of patient health and then determines the transplant eligibility threshold and the amount of post-transplant care. The center must balance the tradeoffs between profit, patient welfare, and CoP compliance. Specifically, it weighs the revenue from transplant procedures and post-transplant care against the regulatory penalties associated with high patient mortality rates. The model delivers two predictions about the center's response to CoP implementation. First, CoP raises the marginal cost of each transplant by increasing the penalty for poor outcomes, leading centers to reduce the number of transplants. Second, by penalizing poor outcomes, CoP incentivizes centers to improve post-transplant care despite the associated costs. In subsequent analysis, I model patient mortality in my setting, describe the center's objective function, and characterize the optimal transplant decision and post-transplant care. Finally, I provide comparative statics on key parameters and present proofs in Appendix A. Figure II illustrates the center's timeline and decision-making[17].

[Figure II about here.]

3.1 Setup

Patient health is denoted as x, where $x \sim N(\mu_x, \sigma_x^2)$ [18]. However, when deciding whether to transplant, centers only observe a noisy signal of patient health, $\tilde{x} = x + u$, where $u \sim N(0, \sigma_u^2)$ is independent of x. Thus, $\tilde{x}$ is an unbiased signal for patient health x. After the transplant, centers observe x and decide on post-transplant care $q(x)$. Transplant patients die if the latent variable $y > 0$, where $y = \varepsilon - x - q(x)$ and $\varepsilon \sim N(0, \sigma^2)$ is a normally distributed idiosyncratic shock. Let the probability that a patient with health x and post-transplant care $q(x)$ die to be $P(x, q(x)) =$

[17] For brevity, I abstract from the kidney decision in my current model. In Appendix B, I include an additional stage where the center chooses either a good or a bad kidney. Both models have similar results on transplant threshold and post-transplant care.

[18] Patients with higher x are deemed healthier and more suitable for transplant (OPTN, 2023).

$1 - \Phi\left(\frac{q(x)+x}{\sigma}\right)$, which is decreasing in q and x: more post-transplant care or healthier patient reduces the likelihood of transplant deaths. Conditional on transplant decision and post-transplant care, the center expects $\int_{\tilde{x}} A(\tilde{x}) \int_x P(x, q(x)) p(x|\tilde{x}) dx dF(\tilde{x})$ patients to die, where $p(x|\tilde{x})$ is the posterior distribution of x given $\tilde{x}$ and can be derived with Bayes' rule.

I follow Clemens and Gottlieb (2014); Dickstein (2017); Alexander (2020); Shi (2023) and model the center's objective function as a weighted combination of profit and concern for patient utility. The weight placed on profit is ρ and can be interpreted as the center's belief in punishment. In my setting, the center becomes more altruistic and places more weight on patient utility when the likelihood of punishment is low (i.e., low ρ). CMS pays the center a fixed reimbursement π for each transplant and a reimbursement rate α for each unit of post-transplant care, $q(x)$. Thus, the center profit is $\pi + \alpha q(x)$. A center's concern for patient welfare can be understood as altruism on behalf of the patient or as the center acting to preserve its reputation (Alexander, 2020).

The patient's utility from post-transplant care is concave in $q(x)$, reflecting diminishing returns to care. Healthier patients (higher x) derive greater benefits from transplants, but excessive care imposes costs due to coinsurance or opportunity cost on patients' time (Senanayake et al., 2020). The patient receives zero if the center does not perform a transplant. The center maximizes utility and chooses $A(\tilde{x}), q(x)$ to maximize a weighted average of their profit and the patient's utility from transplant[19]:

$$\max_{A(\tilde{x}),q(x)} \int_{\tilde{x}} A(\tilde{x}) \int_x \left[\rho \overbrace{[\pi + \alpha q(x)]}^{\text{center profit}} + (1-\rho) \overbrace{\left[x q(x) - \frac{\gamma}{2} q^2(x) \right]}^{\text{patient utility}} \right] p(x|\tilde{x}) dx \quad dF(\tilde{x}) \tag{1}$$

$$\text{s.t.} \quad \overbrace{\int_{\tilde{x}} A(\tilde{x})}^{\text{small center discount}} \quad \overbrace{\int_x P(x, q(x)) p(x|\tilde{x}) dx}^{\text{"not too many deaths"}} dF(\tilde{x}) \leq \tau$$

[19]The notation $q(x)$ indicates that centers observe patient health status when choosing post-transplant care.

τ is the CoP limit, and the rest of the terms in the constraint reflect the CoP conditions in Section 2.4. $\int_x P(x, q(x))p(x|\tilde{x})dx$ mimics conditions 1 and 2: there cannot be too many post-transplant deaths. However, even if it does, the center is exempted if condition 3 fails (i.e., the sample size is so small that differences between observed and expected deaths are statistically insignificant). $\int_{\tilde{x}} A(\tilde{x})dF(\tilde{x})$ mimics condition 3 and serves as a scaling factor that makes it less likely for small centers to exceed the CoP limit, τ.

Intuitively, the center balances competing incentives. On one hand, it seeks to maximize profit by performing more transplants and providing reimbursable care. On the other hand, performance concerns and patient welfare impose constraints: (i) transplanting too many patients increases the likelihood of exceeding the CoP mortality limit; (ii) patients dislike excessive post-transplant care due to the marginal cost $\gamma > 0$. The center optimally trades off these incentives by adjusting the transplant decision $A(\tilde{x})$ and post-transplant care $q(x)$. Next, I characterize the optimal $A^*(\tilde{x}), q^*(x)$ and present the proofs in Appendix A.

Proposition 1. *The optimal $q^*(x)$ is an implicit solution to the equation 10. $A^*(\tilde{x})$ takes the form of a cutoff strategy as defined in equation 11, and t^* is the transplant threshold where patients with $\tilde{x} \geq t^*$ will receive transplants and post-transplant care. Conversely, patients with $\tilde{x} < t^*$ will receive no transplants nor post-transplant care.*

3.2 Comparative statics

In this stylized model, the pre-CoP announcement reflects $\tau \to \infty$, meaning no effective regulatory constraints on the product of transplants and mortality, allowing centers to optimize without restrictions. The post-CoP announcement reflects $\tau < \infty$, introducing binding regulatory constraints. The following result illustrates the comparative statics for the transplant threshold t^* and post-transplant care $q^*(x)$ as CMS announces CoP (i.e., τ decreases). I present the proofs in Appendix A.

Proposition 2. *As CMS announces CoP (i.e., τ decreases), the transplant threshold t^* increases*

$\left(\frac{\partial t^*}{\partial \tau} < 0\right)$*; post-transplant care* $q^*(x)$ *increases* $\left(\frac{\partial q^*(x)}{\partial \tau} < 0\right)$.

Proposition 2 predicts that the CoP announcement decreases the fraction of patients receiving transplants. This reduction is not necessarily due to centers selecting healthier patients, but rather a higher threshold t^* increases the likelihood that patients with better true health x surpass it. Consequently, the average health of transplanted patients rises (i.e., $\mathbb{E}[x|\tilde{x} \geq t^*]$ increases with t^*). The magnitude of this increase depends on how well the noisy signal $\tilde{x}$ reflects x. When $\tilde{x}$ is highly informative (low Var(u)), the stricter threshold effectively excludes less-healthy patients, substantially improving the average health of transplanted patients. Conversely, when $\tilde{x}$ is weakly informative (high Var(u)), the threshold has little effect on health composition.

4 Data and descriptive analysis

This paper uses two administrative datasets from the OPTN: the Standard Transplant Analysis Research (STAR) and Potential Transplant Recipient (PTR) data. The OPTN data system includes data on all donors, waitlisted candidates, and transplant recipients in the U.S. submitted by its members.

4.1 Sample construction

The STAR dataset provides detailed information on patient and donor characteristics, as well as survival outcomes. Crucially, patients who receive a transplant are also included in the follow-up data, which tracks their health status over time and records all immunosuppressant prescriptions and medical tests performed during subsequent visits. The PTR dataset comprises all kidney offers generated by the system, as well as records of acceptance or decline decisions. These datasets are populated using information gathered during the allocation process, forms submitted by transplant centers from patient follow-ups after a transplant, and patient death dates merged from social security records.

I restrict attention to patients who received a transplant between January 1st, 2001, and July

31st, 2009, which approximately spans 4 years before and 4 years after the CoP announcement in February 2005 [20]. From this set, I exclude patients who required multiple organ transplants, those who received a kidney from a living donor, and patients from pediatric transplant centers. Correspondingly, I only use data on donor offers and acceptance decisions for my sample of patients. This paper uses three different units of analysis. Section 6 uses patient-appointment information to analyze post-transplant mortality and post-transplant care. Section 6.2 uses patient-kidney offers to analyze transplant center accept-decline decisions. Section 6.2.2 uses kidney-level information to analyze kidney utilization.

4.2 Descriptive analysis

Figure III presents a time-series plot of the post-transplant 1-year mortality rate from 2001 to 2009, showing a steady decline from approximately 12% in 2001 to 9% in 2009 [21]. This reflects significant improvements in post-transplant survival over time. The downward trend appears to have accelerated after the CoP announcement in February 2005, suggesting that the CoP announcement may have contributed to these further improvements. For subsequent analysis, the period before February 2005 is considered the pre-CoP period, February 2005 to July 2007 is the post-CoP announcement period, and the period after July 2007 is the post-CoP implementation period. This timeline provides a natural framework for evaluating the impact of CoP on transplant outcomes.

[Figure III about here.]

Table I presents summary statistics for the sample, with each row representing different follow-up intervals, while panels group key variables. Between 2001 and 2009, 85,496 patients received deceased donor kidney transplants, distributed across three periods (43% in 2001-2004, 31% in

[20] I restrict my analysis sample to this period because the U.S. Food and Drug Administration (FDA) approved generic tacrolimus (Sandoz) in August 2009. This approval likely reduced the cost of maintenance immunosuppressants, which may have incentivized transplant centers to perform more transplants. As a result, the approval could confound the estimated causal effects of CoP on transplant behavior.

[21] Post-transplant 1-year mortality measures the percentage of patients who die within one year after receiving a kidney transplant. For example, among patients transplanted in 2001, 12% died within one year.

2005-2007, and 26% in 2007-2009). Panel A shows high attendance rates at follow-up appointments, indicating strong patient compliance. Attendance, however, declines over time primarily due to post-transplant mortality, which accounts for 92% of missed visits (Table D.I). Panels B and C reveal diverging trends in prescription use, with tacrolimus use increasing and cyclosporine use decreasing over subsequent follow-up intervals. Panel D illustrates stable hospitalization rates across different CoP phases. Overall, the patterns highlighted in Table I suggest high patient compliance and provide preliminary evidence of evolving center practices aimed at improving the prevention of kidney rejection. These findings are further explored in Section 6.3.

[Table I about here.]

Tables D.II and D.III compare transplant kidney and patient characteristics pre-CoP, post-CoP announcements, and implementation. A comparison of columns in both tables reveals no significant differences in overall transplant profiles. However, Table D.III highlights a notable spike in dialysis patients receiving kidney transplants post-CoP. This trend is likely unrelated to centers favoring dialysis patients but instead reflects the broader expansion and consolidation of the two major dialysis chains, Davita and Fresenius, during this time, which increased the number of patients undergoing dialysis treatment (Eliason et al., 2019). Overall, these comparisons provide strong preliminary evidence that, while the total number of transplants has decreased, there is no clear indication that centers are selecting against specific transplant profiles. These patterns are further examined in Section 6.2.

5 Research design

The February 2005 announcement of CoP created both cross-sectional and within-center temporal variation in penalty incentives, providing a suitable context for a difference-in-difference (DiD) analysis of its causal effect. However, three empirical challenges arise in identifying these effects, which the proposed design addresses.

First, CoP penalizes centers based on historical mortality performance adjusted for patient and kidney risk profiles. Since penalized and non-penalized centers differ systematically, cross-sectional comparisons alone may be biased. Thus, I focus on within-center estimates to control for any time-invariant factors influencing center behavior.

Second, treatment status is ambiguous because forward-looking center administrators strategize based on their expectations of exceeding CoP limits rather than waiting for actual penalty. Following Gupta (2021), I model center responses based on their expectations of exceeding CoP thresholds, conditional on information available at the end of the prior six-month window. The linear equation below represents a static version of this economic model:

$$Y_{ickt} = \alpha_c + \delta_t + \sum_{s \in \{ann, imp\}} \beta_s \mathbb{E}[\mathbf{1}(\mathbf{CoP_{c,t_0+5}} > \bar{\mathbf{CoP}}) | I_{t0}] \times \mathbf{1}(t = s) + X'_{ik}\gamma + \varepsilon_{ickt} \quad (2)$$

Here, Y_{ickt} denotes the outcome (e.g., patient mortality, immunosuppressant prescription, patient-kidney offer decision, etc.), α_c controls for time-invariant center characteristics, while δ_t accounts for common shocks affecting all centers within a six-month window, t. The key term represents each center's expectation, given the prior periods' information (I_{t_0}), of exceeding the CoP threshold ($\bar{\mathbf{CoP}}$). This forward-looking approach differs from existing literature, which focuses solely on post-CoP implementation behaviors, allowing me to identify anticipatory adjustments that occur between the CoP announcement and implementation [22]. Finally, X_{ik} controls for patient and kidney risk factors, while ε_{ickt} captures omitted factors influencing outcomes. The parameter β_s measures the average change in outcomes after the CoP announcement or implementation associated with a 10 pp (or one standard deviation) increase in centers' penalty expectations.

5.1 Measure of center expectation

Center beliefs about exceeding the CoP threshold, while central to identification, are unobserved. I address this by constructing an empirical analog based on two simplifying assumptions:

[22] Penalties, such as system reviews or decertification, strongly incentivize centers to adjust their practices preemptively (Hamilton, 2013).

centers form rational expectations about being penalized using their past mortality performance and transplant volumes. Specifically, in February 2005 (t_0), centers predict their probability of being penalized in July 2007 $(t_0 + 5)$ based on their unadjusted post-transplant mortality and transplant volume over the period from July 2001 to December 2004.

Following Gupta (2021), I nonparametrically predict each center's expectation of future penalty via kernel regression of actual penalty status on the relevant unadjusted post-transplant mortality and transplant volume:

$$\begin{aligned} \mathbb{E}[\mathbf{1}(\mathbf{CoP}_{\mathbf{c},\mathbf{t_0+5}} > \bar{\mathbf{CoP}})|I_{t0}] &= f(R_{ct}, TX_{ct}) + \xi_{ct} \\ \widehat{\mathbb{E}}[\mathbf{1}(\mathbf{CoP}_{\mathbf{c},\mathbf{t_0+5}} > \bar{\mathbf{CoP}})|I_{t0}] &= \widehat{f}(R_{ct}, TX_{ct}) \end{aligned} \tag{3}$$

Intuitively, this measure predicts the likelihood of a penalty based on the experience of neighboring centers falling within the kernel bandwidth. One problem is that the penalty status released in July 2007 is not exogenous, as it includes post-announcement transplants. Thus, I circumvent this issue using earlier penalty status from January 2005, July 2005, and January 2006, whose 2.5-year rolling cohorts predate CoP's announcement (illustrated in Figure C.V). The resulting probability of penalty is denoted as ρ_c.

[Figure IV about here.]

My analysis focuses on the 209 centers during the CoP announcement period. Figure IV shows that the average (median) center faces a 10 (7)% probability of future penalties, significantly lower than the approximately 50% penalty likelihood observed in the Hospital Readmissions Reduction Program (HRRP) setting (Gupta, 2021). This difference arises because CoP's penalty criteria are more stringent and include safeguards for low-volume centers (condition 3), reducing overall penalty likelihoods [23].

Despite low penalty probabilities, the expected cost for penalized centers is substantial, includ-

[23] HRRP penalties apply when hospitals exceed the national average for 30-day readmissions, leading to a higher baseline penalty likelihood. By contrast, CoP penalties require centers to meet all three specified conditions simultaneously. Figure C.VI further illustrates the differences in penalty expectations between CoP and HRRP.

ing potential system reviews, temporary shutdowns, or decertification. These significant consequences strongly incentivize centers to adjust their behavior and proactively improve post-transplant outcomes.

5.2 Mean reversion

The OLS regression in the previous subsection could underestimate the effect of the CoP announcement due to the possibility of mean reversion (Chay, McEwan and Urquiola, 2005; Gupta, 2021). Transplant centers may have escaped penalty due to a temporary downswing in their mortality rate above their "true" mean, just as the penalty status was first determined. They adjust their behavior, knowing that their performance will revert to their true, lower-quality self in the future. Hence, OLS estimates would suggest limited behavioral responses from the policy. To overcome this, I employ an instrumental variables approach, relying on variation in center quality from 2002 to 2004, before the CoP announcement, to generate exogenous variation in penalty probability under CoP. This approach assumes stable underlying center quality, isolating exogenous variation from temporary fluctuations.

Following established literature on dynamic models (Anderson and Hsiao, 1981; Amemiya and MaCurdy, 1986; Arellano and Bond, 1991), I instrument penalty expectations (ρ_c) using predetermined center characteristics characteristics (Arellano and Bover, 1995; Acemoglu and Finkelstein, 2008; Gupta, 2021). Accordingly, I use a center-level instrument Z_c predicted using baseline CMS covariates [24]. The IV approach also mitigates concerns of measurement error in constructing center expectations.

Equation 5 presents the empirical version of the conceptual model in equation 2, where I replace

[24] These include cold ischemia time, donor medical history, patient and kidney diagnosis, age, BMI, creatinine levels, race, insurance coverage, etc.

the expectation term with ρ_c. Equation 4 is the first-stage equation:

$$\rho_c \times \mathbb{1}(t = s) = \pi_{1c} + \pi_{2t} + \lambda Z_c \times \mathbb{1}(t = s) + X'_{ik}\pi_3 + u_{ickt} \quad ; \quad s \in \{ann, imp\} \tag{4}$$

$$Y_{ickt} = \alpha_c + \delta_t + \sum_{s \in \{ann,imp\}} \beta_s \widehat{\rho_c} \times \mathbb{1}(t = s) + X'_{ik}\gamma + \varepsilon_{ickt} \tag{5}$$

I estimate the two rows of equations jointly using two-stage least squares (2SLS), such that the endogenous variable, ρ_c, is replaced by the predicted value, $\widehat{\rho_c}$ generated using the first stage. The baseline instrument, Z_c, is an expected mortality rate calculated using data on patient and kidney risk factors from transplanted samples collected between 2002 and 2004, ensuring exogeneity and removing transient noise. The key identifying assumption is parallel trends; centers with low and high baseline expected mortality would exhibit similar trends absent CoP. To examine this, I estimate a dynamic nonparametric model (equation 6, comparing centers with high and low instrument values over time:

$$Y_{ickt} = \alpha_h + \delta_t + \sum_{s \neq 2003h2} \beta_s \mathbf{1}(d_{Z_c=1}) \times \mathbf{1}(t = s) + \varepsilon_{ickt} \tag{6}$$

Here, $d_{Z_c=1}$ is an indicator for centers in the upper half of the baseline mortality risk, representing those with the greatest incentive to improve their performance.

5.3 Subsample

To identify the causal effect of the CoP policy on post-transplant mortality and center behaviors, I compare patients from the same transplant center whose follow-up periods do not overlap with the CoP announcement (illustrated as solid lines in Figure C.VII). Excluding these overlapping cases mitigates temporal confounding, as mortality risks naturally evolve over time, ensuring comparisons reflect outcomes exclusively influenced by pre- or post-CoP conditions and thereby enhancing internal validity.

However, the above approach could still be biased if the composition of patients within centers

changed significantly after the CoP announcement, introducing unobservable differences [25]. To address this, whenever feasible, I supplement the previous analysis by using patients with overlapping follow-up timelines. In these cases, I employ patient-fixed effects regressions, leveraging within-patient variation over time to isolate the causal effects of CoP [26]. The following equation represents the patient-fixed effects model:

$$Y_{ict} = \alpha_i + \delta_t + \sum_{s \in \{ann, imp\}} \beta_s \rho_c \times \mathbb{1}(t = s) + X'_{it}\gamma + \varepsilon_{ict} \tag{7}$$

Y_{ict} is the relevant outcome variable. α_i controls for time-invariant patient characteristics, while δ_t accounts for common shocks affecting all patients within a 6-month window.

6 Effects on post-transplant mortality and mechanisms

This section quantifies the effects of the CoP policy on post-transplant 1-year mortality, the program's targeted metric, to establish its top-line impact. Using patient-kidney offers and follow-up data, I then analyze how the selection and post-transplant care channel drive the changes in post-transplant mortality at different phases of the CoP policy, respectively.

6.1 Targeted metric

[Figure V about here.]

Figure V plots the coefficients β_s of equation 6 for 6-month windows between 2001 and 2009, with 2003h2 as the reference period, to examine changes in the probability of post-transplant 1-year mortality. The plot reveals two key insights. First, no preexisting differential trends exist

[25] Section 6.2.1 and 7.1.3 demonstrate that centers do not appear to systematically discriminate against specific patient profiles or kidney types at the transplant or admission stages. These results mitigate concerns about potential selection bias that could undermine the above identification strategy.

[26] A limitation of analyzing patients with overlapping follow-up timelines is that Medicare covers 80% of immunosuppressive medications expenses through Part B for the first three years post-transplant. Beyond that, coverage requires eligibility based on age or disability, or patients must obtain coverage through other insurance plans or Medicare Part D when eligible. This institutional feature makes it difficult to disentangle the policy's causal impact from effects arising from the loss of Medicare subsidies, especially after CMS implemented CoP.

between centers with low and high values of Z_h, indicating that the parallel trends assumption might hold in my setting. Second, after the CoP announcement in February 2005 (first dashed vertical line), there was a statistically significant and economically meaningful decline in mortality for centers with higher penalty risks. The pattern persisted even when CMS implemented CoP (second dashed vertical line). My results suggest that the no-anticipatory assumption in prior studies, which focus on behavior post-CoP implementation, may overlook essential center responses during the announcement period.

Table II presents OLS (Column 1) and IV (Column 2) estimates, showing a 2.78 pp reduction in post-transplant 1-year mortality for a one-standard-deviation increase in a center's belief after CMS announced CoP. The IV estimates are larger than the OLS estimates, consistent with concerns that mean reversion may underestimate the CoP response.

For context, in 2004, 11% of the 10,370 kidney transplant recipients died within a year. A 2.78 pp decline implies that 853 patients died post-transplant, compared to 1,147 previously—a 24% decrease. The improvements persisted even after CMS implemented CoP in July 2007, although at a smaller magnitude (2.11 pp, 18% of the baseline).

[Table II about here.]

Further analysis, as shown in Figure C.VIII and Table D.IV, using granular time intervals, reveals that mortality improvements are most pronounced within the first two weeks and six months after receiving a kidney transplant. This suggests that transplant centers concentrated their mitigation efforts on these critical periods, such as closer monitoring and an adjusted immunosuppressant regimen. While early and intermediate post-transplant periods show substantial improvements, the policy had little or no effect on mortality beyond two years. These findings suggest that CoP significantly improved early transplant outcomes and that centers prioritize immediate and intermediate recovery stages to achieve these gains. In the following subsection, I examine how both selection and post-transplant care channels contributed to the improvements in post-transplant mortality.

6.2 Selection channel

Quantifying the role, if any, of distortions in producing the decline in post-transplant 1-year mortality reported above is vital. This subsection examines how centers select patients and kidneys for transplantation and their effect on kidney utilization.

6.2.1 Selection into transplant

[Figure VI about here.]

The CoP penalty reduces the financial attractiveness of performing transplants, incentivizing centers to accept fewer patient-kidney offers and potentially wait for better matches to minimize post-transplant mortality. Figure VI plots the estimated effects on acceptance probabilities for patient-kidney pairs in each 6-month window, using equation 6 with an acceptance indicator, A_{ickt}, as the dependent variable. The figures show that centers expecting greater penalties decreased acceptance rates for patient-kidney pairs after the CoP announcement. However, acceptance rates for these centers were already trending lower in 2004, suggesting that pre-existing trends may partly explain the observed changes. The trend rebounded after CMS implemented CoP, highlighting a potential uptick in acceptance behavior. On average, Table III indicates a 1.42 pp decline in acceptance probability, corresponding to a 16.2% decrease given the mean acceptance rate of 8.79%, after CMS announced CoP. But such behavior dissipated as CMS implemented CoP.

[Table III about here.]

To test whether centers avoided certain patient or kidney profiles to reduce mortality risk, I estimated triple-difference models interacting center penalty risk with patient and kidney covariates from the CoP risk-adjustment model. Tables D.V and D.VI show no statistically significant results, indicating effective risk adjustment that did not incentivize strategic selection based on included covariates [27]. However, risk-adjustment models might omit critical covariates predictive of

[27] See SRTR website and Table 1 of Weinhandl et al. (2009) for consistently included covariates.

survival, potentially discouraging centers from transplanting these riskier profiles—an unintended consequence of CoP (Weinhandl et al., 2009; Kasiske et al., 2012). Further triple-difference regressions (Tables D.VII, D.VIII) find no evidence supporting this concern [28].

6.2.2 Kidney discard

While CoP incentivized centers to become less likely to accept a given patient-kidney pair, OPTN only discards deceased donor kidneys if no center is willing to transplant them. Each discarded kidney represents a missed opportunity to save or improve a patient's life, particularly given the significant organ shortage and growing number of patients on the waitlist [29]. While some kidneys are discarded due to legitimate medical concerns, such as poor quality or high risk of complications, a substantial proportion of discarded kidneys might still be viable for transplantation. Analyzing the broader implications of CoP on kidney utilization is essential to identify whether CoP exacerbates these issues by incentivizing overly cautious behavior.

To investigate this, I aggregate data on all patient-kidney offers to the kidney level and construct a weighted average of center penalty exposure, $Exposure_k$ [30]. The following equation represents the discard model:

$$D_{kdt} = \alpha_d + \delta_t + \sum_{s \in \{ann, imp\}} \beta_s Exposure_k \times \mathbf{1}(t = s) + X_k' \gamma + \varepsilon_{kt} \tag{8}$$

Here, D_{kdt} is the discard indicator, α_d represents donor service area fixed effects, δ_t accounts for six-month window fixed effects, and X_k is a vector of kidney characteristics. The parameter of interest, β_s, captures whether kidneys offered to more exposed centers are more likely to be discarded after the CoP announcement or implementation. Table D.IX presents the results. Column 1 finds no statistically significant increase in overall kidney discard rates after CoP implementation. Column 2, which introduces a triple-difference specification interacting exposure with a high-risk

[28] The omitted covariate list is non-exhaustive. For instance, cardiovascular disease or treatments that remove donor-specific antibodies—highlighted by Kasiske et al. (2012)—are not adjusted for or collected in my data.

[29] Nearly 30% of recovered kidneys are discarded each year (McKenney et al., 2024).

[30] I use the proportion of patients from the same transplant center as weights.

kidney indicator, suggests that high-risk kidneys were 3.58 pp (23.8%) more likely to be discarded than low-risk kidneys following CMS's CoP announcement. However, this effect attenuated after CoP implementation, suggesting an adaptive response among transplant centers.

Columns 3 and 4 examine the number of patients offered high-risk kidneys to explore the mechanism behind this trend. Following CMS's CoP announcement, high-risk kidneys were offered to 37.8 patients, a 17% increase relative to low-risk kidneys, suggesting greater difficulty finding an accepting center. However, post-implementation, high-risk kidneys were offered to 95.7 fewer patients (a 33.9% relative decrease), indicating that centers became more receptive.

Why did patient-kidney acceptance and kidney discard rebound? Anecdotal evidence suggests that uncertainty surrounding the initial CoP proposal prompted transplant centers to adopt a cautious approach to decision-making. For example, communications between members of the American Society of Transplant Surgeons (ASTC) and CMS regulators highlight significant concerns about the ambiguous appeal processes in the original CoP proposal:

> *"Although the joint task force still had some fundamental disagreements with the final rule, it was felt to be a clear improvement over the initial proposal, ... It also clarified due process rights available to transplant centers in the event of an unfavorable review and provided for the consideration of mitigating circumstances when outcome and volume criteria were not met."* (Abecassis et al., 2008)

This excerpt highlights how uncertainty regarding penalties and appeals initially led to cautiousness in transplant decisions. Once CMS clarified these procedural ambiguities and implemented CoP, transplant acceptance rates rebounded, aligning with the empirical patterns depicted in Figure VI [31].

[31] In Section 7.2, I demonstrate that despite the initial drop in patient-kidney acceptance rates, this did not worsen waiting times nor waitlist mortality.

6.3 Post-transplant care channel

In Section 3, I presented a stylized model describing the center's incentives and how CoP affects the center's behavior. Under this model, optimal post-transplant care, $q^*(x)$, will equate the marginal cost of incremental care with the marginal benefit to the patient and center. CoP incentivizes centers to provide optimal post-transplant care, thereby decreasing the patient's probability of post-transplant mortality and, in turn, the center's likelihood of exceeding the CoP threshold. Hence, CoP nudges the center to increase post-transplant care on average (i.e., $\frac{\partial q^*(x)}{\partial \tau} > 0$). Using novel follow-up data tracking patient immunosuppressant prescriptions and non-targeted patient outcomes during revisits, I empirically assess how centers changed their treatment protocols in response to CoP.

6.3.1 Maintenance immunosuppressants

The immune system naturally identifies and attacks foreign bodies, posing challenges for kidney transplant recipients whose new kidney is perceived as foreign. Maintenance immunosuppressants, particularly calcineurin inhibitors (CNIs), are crucial in preventing acute kidney rejection by suppressing the immune response and reducing the risk of kidney rejection [32]. CNIs, specifically cyclosporine and tacrolimus, are widely prescribed due to their potency in suppressing immune activity [33]. In this section, I analyze prescription patterns for these two common CNIs.

[Figure VII about here.]

Figure VII presents an event-study analysis of cyclosporine and tacrolimus prescription rates at the 2-week follow-up interval across different CoP policy phases. Following the CoP announcement, the prescription rates for cyclosporine decreased, while those for tacrolimus increased. Tables D.X and D.XI further quantify these shifts via DiD estimates. Specifically, Column 1 of each

[32] Three types of immunosuppressants are utilized during transplantation: (i) *induction medicines*—potent intravenous medications administered at transplant to initially suppress the immune system; (ii) *maintenance medicines*—ongoing treatments used to prolong graft viability; and (iii) *rejection medicines*—used to treat rejection episodes. Source: UNOS, types of immunosuppressants.

[33] In my data, CNIs are prescribed in 93% of a patient's immunosuppressant regimen and are often used in combination with antimetabolites and corticosteroids.

table shows that 2-week cyclosporine prescriptions decreased by 5.65 pp (a 44% decline from the 12.7% baseline), while 2-week tacrolimus prescriptions increased by 5.89 pp (an 8% rise from the 80% baseline). These changes persisted post-CoP implementation and across subsequent follow-up intervals [34].

These findings indicate centers with higher penalty beliefs systematically substituted tacrolimus for cyclosporine. This substitution aligns with randomized controlled trials reporting improved post-transplant 1-year mortality — the key metric targeted by CoP — due to reduced acute kidney rejection after taking tacrolimus (Webster et al., 2005). I find similar evidence in Table D.IV showing decreased kidney rejection-related deaths at 6 months and 1 year follow-ups post-CoP implementation.

6.3.2 Side effects and non-targeted outcomes

While tacrolimus is more effective in preventing kidney rejection, it increases the risk of gastrointestinal disturbances and diabetes compared to cyclosporine (Lee, Myoung and Kim, 2023). Next, I assess whether increased tacrolimus prescriptions worsened non-targeted patient outcomes (i.e., readmissions, diabetes, return to dialysis, etc.). Column 1 of Table D.XV shows a 3.72 pp (16.9%) increase in readmissions during the 1-year follow-up period relative to a baseline of 22.1%, following the CoP announcement. However, this rise in hospitalization likely reflects centers' increased vigilance and conservative discharge criteria rather than deteriorating patient health. This interpretation is supported by statistically insignificant DiD estimates for other non-targeted outcomes in Columns 2-4 in Table D.XV and Columns 1-4 in Table D.XVI [35].

Additionally, the increased use of tacrolimus likely coincided with improved care quality and better management of viral infections associated with immunosuppression. This is evidenced in Table D.XIV, where Column 1 indicates a 0.4 pp (46%) reduction in deaths due to viral infections within 2 weeks post-transplant (baseline: 0.96%) after CoP's announcement, a pattern persisting post-CoP implementation and across follow-up intervals.

[34] In the appendix, I obtain similar results in Table D.XII using patient-fixed effects, as described in Section 5.3.
[35] Unfortunately, I do not observe the reasons nor the length of hospitalization in the follow-up data.

Overall, the analysis indicates transplant centers shifted toward prescribing the more potent tacrolimus and intensified patient monitoring for potential side effects. This shift has significant economic implications for CMS. James and Mannon (2015) estimated annual maintenance costs at $16,000 for tacrolimus versus $8,400 for cyclosporine. While more expensive upfront, prescribing tacrolimus with enhanced monitoring remains considerably cheaper than treating acute kidney rejection ($22,407 per episode) or managing kidney failure through dialysis ($70,581 annually) or retransplantation ($106,373) (Gheorghian et al., 2012). A conservative back-of-the-envelope calculation, assuming an 8% kidney rejection probability (Hart et al., 2017), indicates this shift generated approximately $8,350 in savings per patient in the first post-transplant year [36].

7 Robustness checks

This section discusses alternative mechanisms, the effect of CoP on non-targeted metrics, and tests the sensitivity of the estimates to modeling assumptions.

7.1 Alternative mechanisms

7.1.1 Compulsory documentation

The CoP policy introduced new documentation requirements, mandating that transplant centers maintain accurate and up-to-date medical records for both pre- and post-transplant care (Federal Register, 2007). Abecassis et al. (2008) suggests that these mandates could divert resources away from patient care. To test this, I use teaching status and patient volume as proxies for centers' administrative capacity and estimate triple-difference models. Table D.XVII shows no evidence that CoP differentially affected post-transplant mortality or organ acceptance rates at centers with lower administrative capacity, suggesting that documentation requirements did not compromise clinical performance.

[36] These calculations are conservative and exclude potential increases in hospitalization claims, additional medical checks, and logistical dialysis costs.

7.1.2 Donor filtering

Transplant centers can proactively filter donor offers for patients listed in the UNet system (King et al., 2022; Yu et al., 2024), such as setting maximum donor age criteria independent of biological compatibility. The concern is that CoP incentivizes centers to impose stricter donor criteria to avoid risky kidney profiles. Leveraging patients whose waitlist tenure overlaps with CoP announcement and implementation, I employ patient-fixed effects regressions across various donor characteristics [37]. Tables D.XVIII and D.XIX reveal no consistent evidence of stricter criteria. Instead, centers appear to loosen certain donor criteria (e.g., accepting higher creatinine levels, longer ischemic times) to expand their kidney pool [38].

7.1.3 Strategic admission

Another way centers could influence post-transplant outcomes is by selectively admitting patients. For example, White et al. (2014) argues that centers might adopt stricter admission criteria, especially targeting socioeconomic factors affecting patient compliance with post-transplant care [39]. However, examining various socioeconomic and health indicators among admitted patients, I find no support for this hypothesis in Tables D.XX and D.XXI. Since CoP penalizes centers for post-transplant—but not waitlist—mortality, admitting patients without immediate transplant commitments does not directly affect performance metrics, which may explain why admission practices remained unchanged.

7.1.4 CMV testing

Cytomegalovirus (CMV), a virus that can reactivate in immunosuppressed kidney transplant recipients, is routinely monitored in transplant care protocols. Regular testing enables early identifi-

[37] To minimize temporal confounding, I restrict the analysis to patients listed between 2004–2009 who waited fewer than six months initially and remained on the waitlist for at least three years.

[38] Characteristics such as diabetes, obesity, donors outside the service area, and death by cardiac arrest were omitted from the result tables as centers did not restrict these offers, reflecting minimal donor filtering.

[39] White et al. (2014) uses within-center temporal variation to analyze the patient admission strategies of penalized centers.

cation of viral infections, allowing for timely antiviral treatment to prevent immune-mediated damage (Hasanzamani et al., 2016; Al Atbee and Tuama, 2022). Table D.XXII finds no evidence that centers significantly altered CMV testing across various follow-up intervals. Given its standardization in transplant care, CMV testing practices likely required minimal adjustment in response to the CoP policy [40].

7.2 Non-targeted patient outcomes

In Section 6.3.2, I presented an array of patient outcomes not targeted by CoP. I do not find evidence that CoP worsened patient health during the recovery process. Next, I examine CoP's effect on the patient's waitlist experience.

Transplant wait times are a critical measure of the system's efficiency and fairness, as prolonged waits increase the risk of complications and reduce the likelihood of successful transplantation. A potential concern is that the CoP policy may incentivize centers to modify their decision-making processes to avoid penalties, potentially altering patients' likelihood of receiving a transplant or remaining on the waitlist for extended periods. Table D.XXIII examines this concern and finds no evidence that either wait times or the probability of being removed from the waitlist increased after CMS announced CoP.

These results support the findings in section 6.2.1, which indicate that centers do not use selective practices when determining which patients receive transplants. These findings suggest that the CoP policy did not negatively impact patient wait times or waitlist mortality, further emphasizing the policy's neutrality in pre-transplant patient management.

7.3 Changing specifications

I test the sensitivity to changing key modeling assumptions. First, I use the entire patient sample and include patients whose post-transplant mortality timeline overlaps with the CoP announcement. Table D.XXIV reproduces the coefficients for equation 5 using outcome variables: post-transplant

[40] Source: Managing Kidney Transplant Recipients.

mortality, tacrolimus, and cyclosporine prescription. The estimates are similar to the specifications that exclude overlapping patients.

Second, I use an alternate approach to construct center penalty beliefs. A useful benchmark is to assume that centers had perfect foresight and accurately predicted their actual penalty status in July 2007 under CoP. This can be implemented straightforwardly by replacing the penalty probabilities with actual first-time penalty status indicators in equation 5. Table D.XXV presents estimates that are largely similar in terms of signs but are statistically insignificant. The results here suggest that perfect foresight might not be an appropriate assumption in my context.

Third, I also changed the main specification, allowing the center penalty probability to vary over time. Then, I include this measure in the model along with the interaction term and estimate a standard differences-in-differences specification. The estimates in Table D.XXVI are considerably smaller. Still, they remain statistically significant for most cases [41].

8 Conclusion

This paper examines a large-scale federal oversight program in the U.S. deceased donor kidney transplant setting. While the CoP policy reduced post-transplant mortality rates by 18-24%, the mechanisms driving these improvements evolved across different phases of the CoP policy. Initially, centers became more conservative in transplanting a given patient-kidney pair due to unfamiliarity with the policy's appeal process. This cautious approach resulted in more high-risk kidneys being discarded and a reduction in mortality due to fewer risky transplants. Over time, as centers became more familiar with the policy's details, they restored transplant volumes and prescribed more potent immunosuppressants to prevent kidney rejection. Furthermore, they complemented these efforts with enhanced patient monitoring to manage side effects related to immunosuppression. Back-of-the-envelope calculations suggest improved management of kidney rejection

[41] A weakness of this approach is that the beliefs computed for later periods rely on performance after the first penalty status was known. If penalized centers responded by differentially lowering their mortality rate, their beliefs would decrease in subsequent years, and the model would estimate a lower differential response across centers. Hence, this approach may understate the true response, but it still offers a useful specification check.

generated savings of $8,350 per patient.

While the findings reveal temporary inefficiencies in kidney utilization after the CoP announcement, they indicate that CMS largely achieved its stated goals: (i) protecting potential Medicare beneficiaries awaiting transplantation; (ii) establishing standards for safe and efficient transplants; and (iii) reducing Medicare expenses by lowering transplant failure risks (Federal Register, 2005). Three factors likely contributed to this success. First, CoP's incentive structure balanced meaningful penalties with safeguards against disproportionate harm. Second, frequent communication among American Society of Transplant Surgeons (ASTS) council members fostered collaboration and guidance rather than punishment, easing adaptation (Abecassis et al., 2008). Third, although imperfect, the CMS risk-adjustment model effectively accounted for key patient and kidney-related covariates, mitigating concerns of strategic patient and kidney-related selection based on omitted risk factors.

The findings point to several directions for future research. The analysis highlights the broader potential of integrating policy design with mechanism design to address inefficiencies in the deceased donor kidney program. Exploring how such frameworks can be expanded to other aspects of organ allocation and post-transplant care could yield valuable insights for improving system-wide outcomes.

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List of Figures

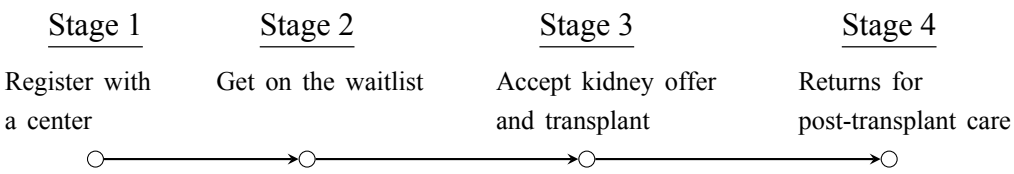


Figure I: Overview of patient experience on the deceased donor kidney transplant waitlist

Note: Between Stage 2 and 3, if the center declines the offer, the patient retains their priority and continues waiting for future offers. The kidney will then be offered to the next patient.

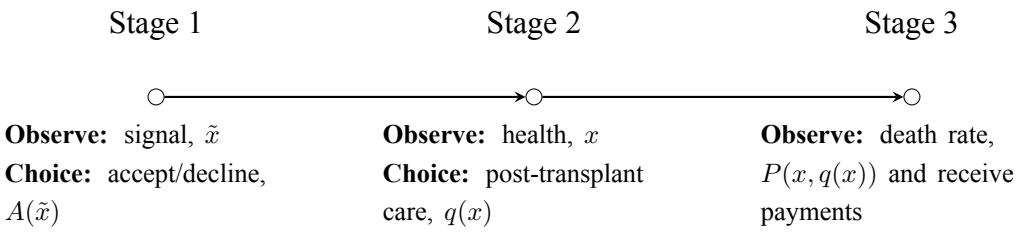


Figure II: Timeline of the center behavior

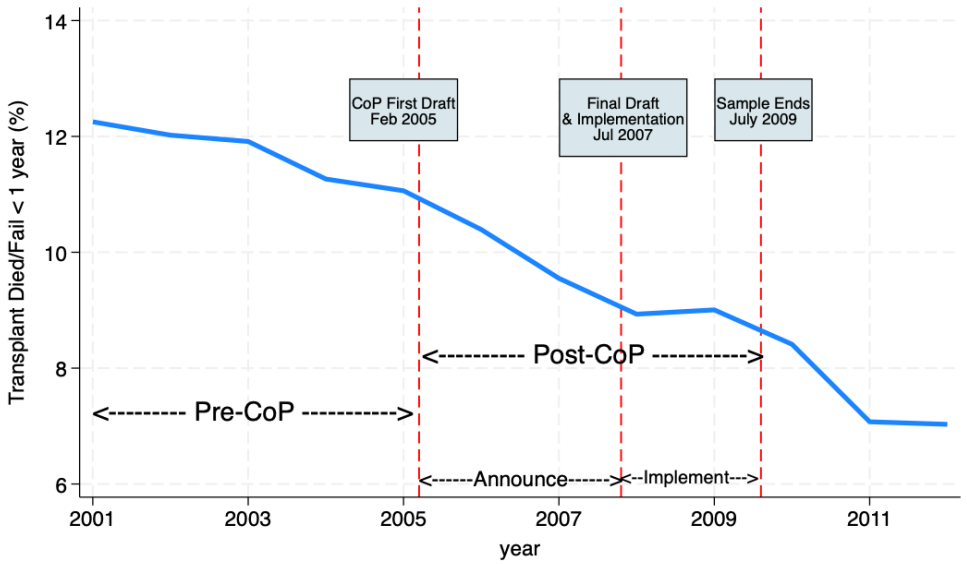


Figure III: Post-transplant mortality decreasing from 2001-2012 (main analysis period)

Note: CMS announced CoP in February 2005, represented by the first red-dotted vertical line. CMS implemented CoP in July 2007, represented by the second red-dotted vertical line. I define the pre-CoP period as January 2001 to February 2005, the post-CoP announcement period as February 2005 to July 2007, and the post-CoP implementation period as July 2007 to December 2009.

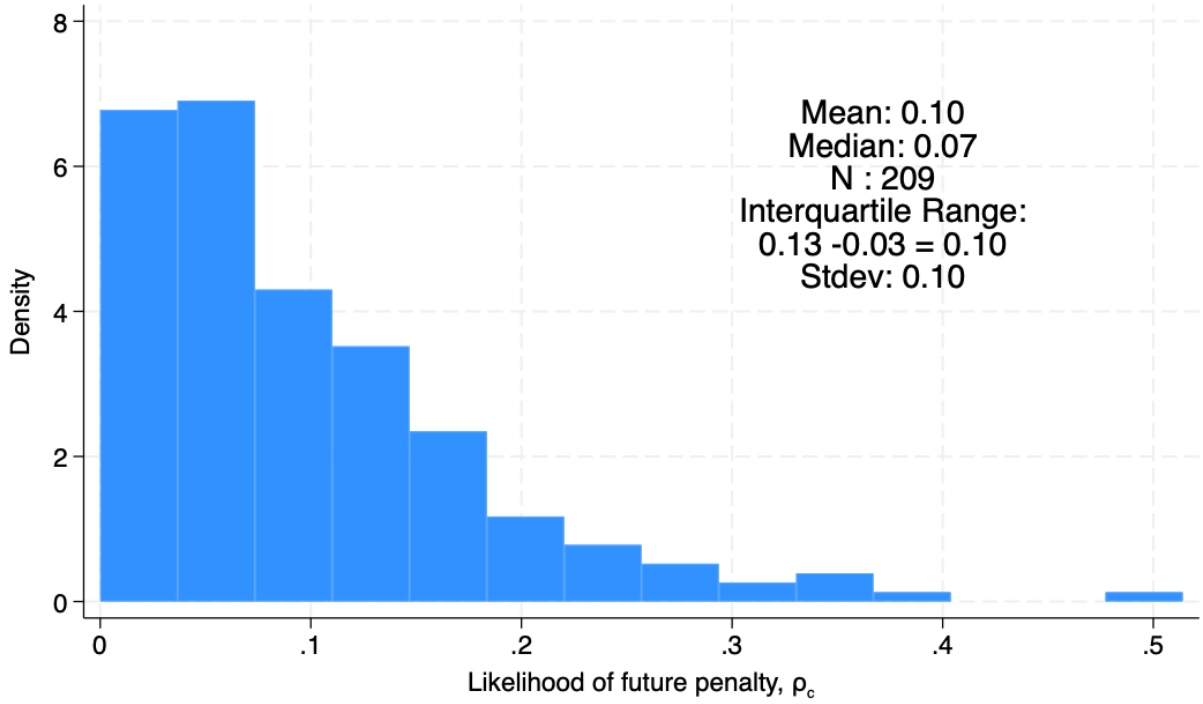


Figure IV: Density of center likelihood of future penalty

Note: This figure illustrates the density of the constructed continuously varying measure of a forward-looking center's expectation of being penalized in the future.

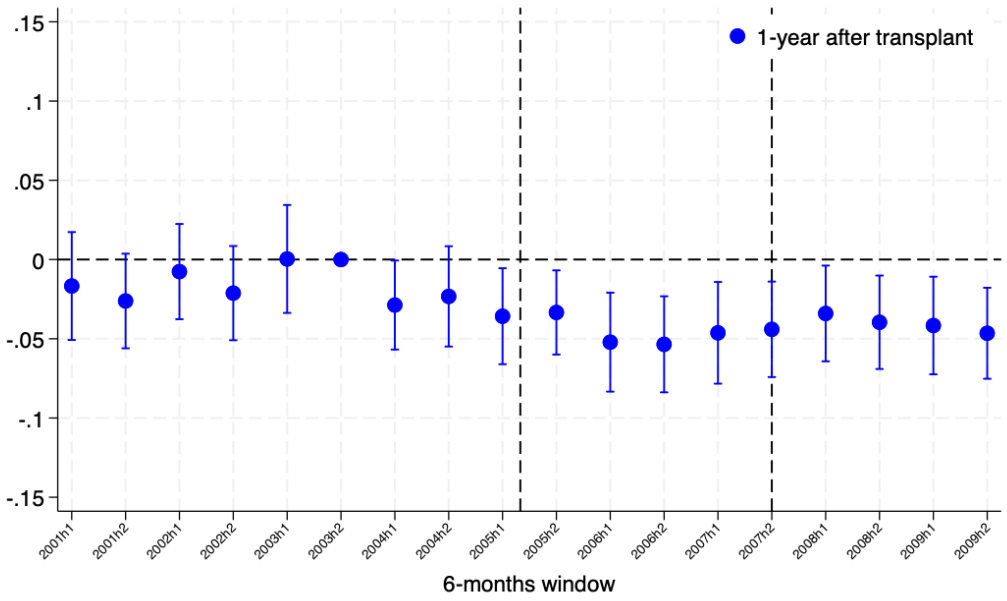


Figure V: Impact on post-transplant 1-year mortality

Note: The figure presents the estimated effects on the probability of post-transplant 1-year mortality, obtained using equation 6 with the instrument Z_h and 2003h2 as the reference 6-month window. The first dashed vertical line is the CoP announcement, and the second line is the CoP implementation. I cluster standard errors at the transplant center level. Error bars indicate 95 percent confidence intervals.

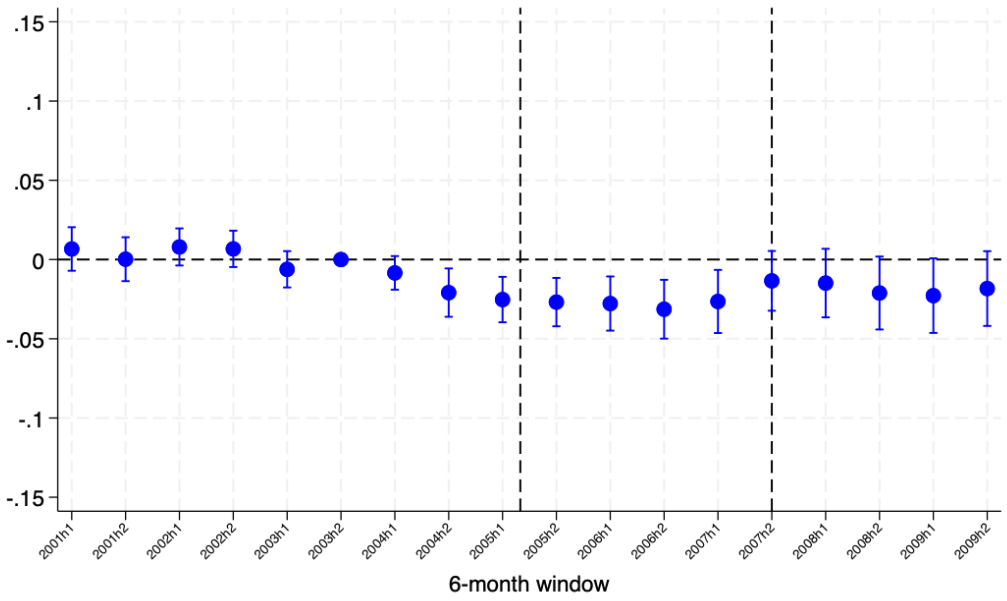


Figure VI: Impact on patient-kidney offer acceptance

Note: The figure presents the estimated effects on the probability of accepting a patient-kidney offer, obtained using equation 6 with the instrument Z_h and 2003h2 as the reference 6-month window. I cluster standard errors at the transplant center level. Error bars indicate 95 percent confidence intervals.

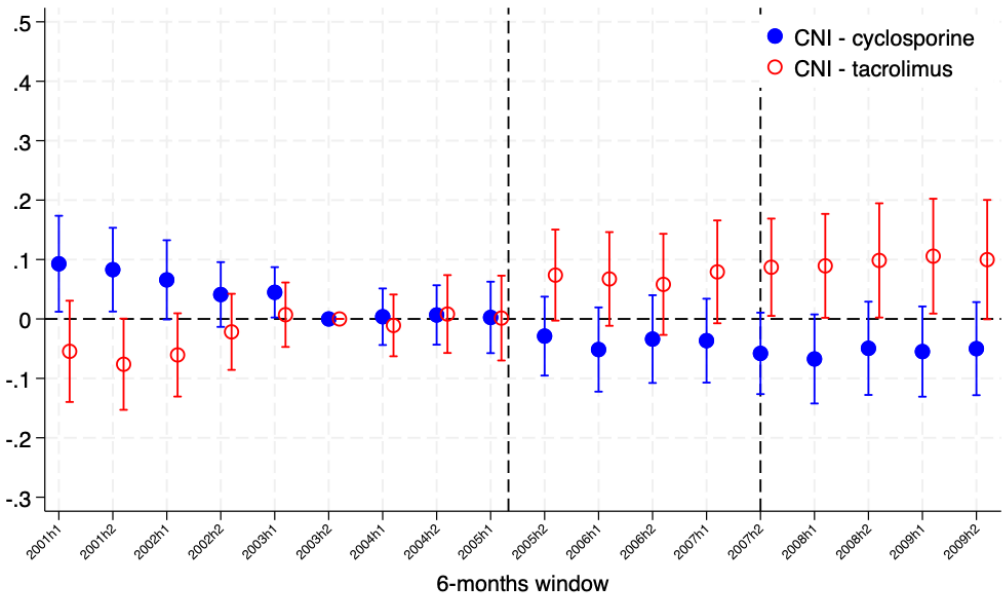


Figure VII: Impact on maintenance immunosuppressant prescription

Note: The figure presents the estimated effects on the probability of prescribing cyclosporine and tacrolimus at the 2-week follow-up interval, obtained using equation 6 with the instrument Z_h and 2003h2 as the reference 6-month window. The first dashed vertical line is the CoP announcement, and the second line is the CoP implementation. I cluster standard errors at the transplant center level. Error bars indicate 95% confidence intervals.

List of Tables

Table I: Follow-up outcomes before, after CoP announcement and implementation

Outcome Measure	Pre-CoP	Post-Announce	Post-Implement
Panel A: follow-up compliance			
2 weeks	98.4%	98.5%	98.7%
6 month	91.6%	92.6%	93.7%
1 year	87.8%	89.1%	90.5%
Panel B: tacrolimus prescription			
2 weeks	64.9%	80.7%	87.8%
6 months	65.8%	74.8%	68.7%
1 year	65.7%	79.2%	84.5%
Panel C: cyclosporine prescription			
2 weeks	26.5%	11.5%	7.0%
6 months	25.4%	10.5%	5.7%
1 year	24.8%	11.0%	7.0%
Panel D: hospitalizations			
6 months	32.6%	33.5%	34.7%
1 year	22.9%	22.5%	21.7%
2 years	19.7%	19.4%	20.2%
Number of Observations	36446	27052	21998

Notes: This table presents summary statistics for follow-up outcomes before CoP, after CoP announcement, and implementation at different follow-up intervals. Panel A shows the percentage of patients who attended follow-up appointments. 92% non-compliers are due to deaths. Panels B, C, and D are conditional on patients attending follow-up appointments. Panel B shows the prescription rate for tacrolimus. Panel C shows the prescription rate of cyclosporine. Panel D shows hospitalization rates during follow-up.

Table II: Impact on targeted metric, post-transplant 1-year mortality

	(1) OLS	(2) IV
Post-Announce	-0.01517*	-0.02782**
	(0.00615)	(0.01031)
Post-Implement	-0.01508*	-0.02112*
	(0.00631)	(0.01061)
Y mean	0.11767	0.11767
F-statistic		56.76781
Fixed Effects	Center, 6-months	Center, 6-months
Observations	78832	78832

Note: This table presents an estimated effect on the probability of post-transplant 1-year mortality, obtained by estimating equation 5 (1st column, OLS) and jointly estimating equations 4 and 5 (2nd column, IV), respectively on the subsample of patients whose post-transplant mortality timeline do not overlap with the CoP announcement described in Section 5.3. I cluster standard errors at the transplant center level. *$p < 0.05$, **$p < 0.01$, ***$p < 0.001$.

Table III: Impact on selection into transplant

	(1) OLS	(2) IV
Post-Announce	-0.00967*	-0.01453**
	(0.00396)	(0.00535)
Post-Implement	-0.01064*	-0.01566
	(0.00531)	(0.00802)
Y mean	0.08273	0.08273
F-statistic		39.82527
Fixed Effects	Center, 6-months	Center, 6-months
Observations	642699	642699

Note: This table presents an estimated effect on the probability of accepting a patient-kidney offer, obtained using equation 2 (1st column, OLS) and 4 (2nd column, IV). I cluster standard errors at the transplant center level. *$p < 0.05$, **$p < 0.01$, *** $p < 0.001$

A Stylized model of center behavior without kidney choices

This section provides the proof for Propositions 1 and 2 in Section 3. The center chooses its transplant decision $A(\tilde{x})$ and post-transplant care $q(x)$ to maximize its expected payoff:

$$\max_{A(\tilde{x}),q(x)} \int_{\tilde{x}} A(\tilde{x}) \int_{x} \overbrace{\left[\rho \overbrace{\left[\pi + \alpha q(x)\right]}^{\text{center profit}} + (1-\rho) \overbrace{\left[x q(x) - \frac{\gamma}{2} q^2(x) \right]}^{\text{patient utility}} \right]}^{\Pi(x,q(x))} p(x|\tilde{x}) dx dF(\tilde{x}) \tag{9}$$

$$\text{s.t.} \int_{\tilde{x}} A(\tilde{x}) \left[\int_{x} P(x, q(x)) p(x|\tilde{x}) dx \right] dF(\tilde{x}) \leq \tau$$

We solve the maximization problem via backwards induction.

A.1 Solving for $q^*(x)$

Let $\lambda \geq 0$ be the Lagrange multiplier on the constraint. Define the Lagrangian:

$$\mathcal{L} = \int_{\tilde{x}} A(\tilde{x}) \int_{x} \Big[\Pi(x, q(x)) \Big] p(x|\tilde{x}) dx \, dF(\tilde{x}) \; - \; \lambda \Big[\int_{\tilde{x}} A(\tilde{x}) \int_{x} P(x, q(x)) p(x|\tilde{x}) dx dF(\tilde{x}) - \tau \Big].$$

Step 1: If the constraint is slack ($\lambda = 0$). For each x, we differentiate $\Pi(x, q(x))$ with respect to $q(x)$:

$$\rho\, \alpha + (1-\rho) \big[x - \gamma\, q(x) \big] = 0 \quad \Longrightarrow \quad (1-\rho)\, \gamma\, q(x) = \rho\, \alpha + (1-\rho)\, x.$$

Hence

$$q^{\text{uncon}}(x) = \frac{\rho\, \alpha + (1-\rho)\, x}{(1-\rho)\, \gamma}.$$

Step 2: If the constraint binds ($\lambda > 0$). For each x, we need

$$\frac{\partial \mathcal{L}}{\partial q} = \int A(\tilde{x}) \frac{\partial \Pi(.)}{\partial q} p(x|\tilde{x}) dx dF(\tilde{x}) - \lambda \int A(\tilde{x}) \frac{\partial P(.)}{\partial q} p(x|\tilde{x}) dF(\tilde{x}) = 0$$

where

$$\frac{\partial}{\partial q} \overbrace{\Big\{\rho(\pi + \alpha\, q(x)) + (1-\rho)\big[x\, q(x) - \tfrac{\gamma}{2} q^2(x)\big]\Big\}}^{\Pi(x,q(x))} = \rho\,\alpha + (1-\rho)\big[x - \gamma\, q\big].$$

and

$$\frac{\partial}{\partial q(x)} \overbrace{\Big[1 - \Phi\big(\tfrac{x+q(x)}{\sigma}\big)\Big]}^{P(x,q(x))} = -\,\phi\Big(\tfrac{x+q(x)}{\sigma}\Big)\,\frac{1}{\sigma},$$

Rearrange for $q^*(x)$:

$$(1-\rho)\,\gamma\, q^*(x) = \rho\,\alpha + (1-\rho)\,x + \lambda\,\frac{1}{\sigma}\,\phi\Big(\tfrac{x+q^*(x)}{\sigma}\Big).$$

Thus we have the *implicit* solution:

$$q^*(x) = \frac{\rho\,\alpha + (1-\rho)\,x}{(1-\rho)\,\gamma} \;+\; \frac{\lambda}{(1-\rho)\,\gamma\,\sigma}\,\phi\Big(\tfrac{x+q^*(x)}{\sigma}\Big). \tag{10}$$

If $\lambda = 0$, we revert to the unconstrained optimum. Otherwise, $q^*(x)$ exceeds the unconstrained level, reflecting a desire to reduce mortality.

A.2 Solving for the acceptance rule $A(\tilde{x})$

Define the net benefit function, $NB(\tilde{x})$

$$NB(\tilde{x}) = \int \Pi(x, q(x)) p(x|\tilde{x}) dx - \lambda \int P(x, q(x)) p(x|\tilde{x}) dx$$

Since the posterior distribution of $p(x|\tilde{x})$ is increasing in $\tilde{x}$, $NB(\tilde{x})$ is a monotonic function of $\tilde{x}$, $A^*(\tilde{x})$ takes the form of a cutoff strategy:

$$A^*(\tilde{x}) = \begin{cases} 1 & \text{if } \tilde{x} \geq t^* \\ 0 & \text{if } \tilde{x} < t^* \end{cases} \tag{11}$$

where t^* is such that $NB(t^*) = 0$. This completes the proof for proposition 1.

A.3 Comparative Statics: Effect of Decreasing τ

As τ decreases, the regulatory constraint tightens, and the Lagrange multiplier λ increases. This forces the center to reduce the product

$$(\text{\# transplanted}) \times (\text{\# expected deaths}).$$

They can do this in two ways:

- **Raise t^* (fewer transplants).** Since

$$NB(\tilde{x}) = \int \Pi(x, q(x)) p(x|\tilde{x}) dx - \lambda \int P(x, q(x)) p(x|\tilde{x}) dx$$

  increases in $\tilde{x}$, a higher threshold means fewer people qualify for a transplant.

- **Raise q^* (improve post-transplant care).** From

$$q^*(x) = \frac{\rho\,\alpha + (1-\rho)\,x}{(1-\rho)\,\gamma} + \frac{\lambda}{(1-\rho)\,\gamma\,\sigma}\,\phi\Big(\tfrac{x+q^*(x)}{\sigma}\Big),$$

  a larger λ makes $q^*(x)$ bigger for each x—the center "overspends" on care (relative to the unconstrained level) to reduce mortality.

This completes the proof for proposition 2.

B Stylized model of center behavior with kidney choices

In this section, I formalize the transplant center's incentives and explore how CoP affects decision-making. I present a stylized model where the center observes a noisy signal of patient health and then chooses the transplant eligibility threshold, the kidney type, and the amount of post-transplant care. The center must balance the tradeoffs between profit, patient welfare, and compliance with CoP constraints. Specifically, the center considers the revenue from transplants and post-transplant care, the relative scarcity of good kidneys, and the regulatory penalties from high patient mortality. The model delivers three predictions about the center's response to CoP implementation. First, CoP raises the marginal cost of each transplant by increasing the penalty for poor outcomes, leading centers to reduce transplants. Second, CoP's stricter death constraints increase the marginal benefit of the safer, "expensive" good kidney, resulting in a shift away from bad kidneys. Third, by penalizing poor outcomes, CoP incentivizes centers to increase post-transplant care despite its cost. In subsequent analysis, I model patient mortality in my setting, describe the center's objective function, and characterize the optimal transplant threshold, kidney choice, and post-transplant care. Finally, I provide comparative statics on key parameters and present proofs in the Appendix. Figure B.I illustrates the center's timeline and decision-making.

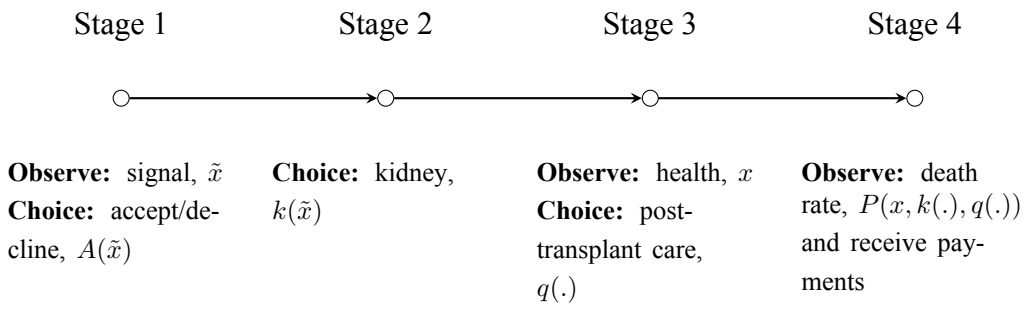


Figure B.I: Timeline of the center behavior

B.1 Setup

Patient health is denoted as x, where $x \sim N(\mu_x, \sigma_x^2)$ [42]. However, when deciding whether to transplant, centers only observe a noisy signal of patient health, $\tilde{x} = x + u$, where $u \sim N(0, \sigma_u^2)$ is independent of x. Thus, $\tilde{x}$ is an unbiased signal for patient health x. Next, the center matches the patient with the good (g) or bad (b) kidney. The good kidney is less risky $(\sigma_g < \sigma_b)$. After the transplant, centers observe x and decide on post-transplant care $q(x, k)$. Transplant patients die if the latent variable $y > 0$, where $y = \varepsilon_k - x - q(x, k)$ and $\varepsilon_k \sim N(0, \sigma_k^2)$. ε_k is a normally distributed idiosyncratic shocks with mean 0 and variance σ_k^2. Let the likelihood that a patient with health x, kidney k, and post-transplant care $q(x, k)$ die be $P(x, k, q(x, k)) = 1 - \Phi\left(\frac{q(x,k)+x}{\sigma_k}\right)$, which is decreasing in q and x: more post-transplant care or healthier patient reduces the likelihood of transplant deaths. Similarly, the good kidney reduces mortality due to its lower variance σ_g^2. Conditional on transplant decision, kidney choice and post-transplant care, the center expects $\int_{\tilde{x}} A(\tilde{x}) \int_x P(x, k, q(x, k)) p(x|\tilde{x}) dF(\tilde{x})$ patients to die, where $p(x|\tilde{x})$ is the posterior distribution of x given $\tilde{x}$ and can be derived with Bayes' rule.

I follow Clemens and Gottlieb (2014); Dickstein (2017); Alexander (2020); Shi (2023) and model the center's objective function as a weighted combination of profit and concern for patient utility. The weight placed on profit is ρ and can be interpreted as the center's belief in punishment. In my setting, the center becomes more altruistic and places more weight on patient utility when the likelihood of punishment is low (i.e., low ρ). CMS pays the center a fixed reimbursement, π for each transplant, and a reimbursement rate α for each unit of post-transplant care, $q(x, k)$. Thus, the center profit is $\pi + \alpha q(x, k)$. A center's concern for patient welfare can be understood as altruism on behalf of the patient or as the center acting to preserve its reputation (Alexander, 2020).

The patient's utility from post-transplant care is concave in $q(x, k)$, reflecting diminishing returns to care. Healthier patients (higher x) derive greater benefits from transplants, but excessive care imposes costs due to coinsurance or opportunity costs on patient's time (Senanayake et al., 2020). The patient also faces a waiting cost of g to receive a good kidney, reflecting the scarcity

[42] Patients with higher x are deemed healthier and more suitable for transplant(OPTN, 2023).

of good kidneys. The patient receives zero if centers do not perform a transplant. The center maximizes utility and chooses $A(\tilde{x}), k(\tilde{x}), q(x,k)$ to maximize a weighted average of their profit and the patient's utility from transplant[43]:

$$\max_{A(\tilde{x}),k(\tilde{x})\in\{g,b\},q(x,k)} \int_{\tilde{x}} A(\tilde{x}) \int_{x} \left[\rho \overbrace{\left[\pi + \alpha q(.)\right]}^{\text{center profit}} + (1-\rho) \overbrace{\left[xq(.) - \frac{\gamma}{2} q^2(.) - \mathbf{1}_{\{k=g\}} g \right]}^{\text{patient utility}} \right] p(x|\tilde{x}) dx dF(\tilde{x})$$

$$\text{s.t.} \quad \overbrace{\int_{\tilde{x}} A(\tilde{x})}^{\text{"small center discount"}} \quad \overbrace{\int_{x} P(x,k,q(.))p(x|\tilde{x})dx \, dF(\tilde{x})}^{\text{"not too many deaths"}} \leq \tau \tag{12}$$

τ is the CoP limit, and the rest of the terms in the constraint reflect the CoP conditions in Section 2.4. $\int_x P(x,k,q(x,k))p(x|\tilde{x})dx$ mimics conditions 1 and 2: there cannot be too many post-transplant deaths. However, even if it does, the center is exempted if condition 3 fails (i.e., the sample size is so small that differences between observed and expected deaths are statistically insignificant). $\int_{\tilde{x}} A(\tilde{x})dF(\tilde{x})$ mimics condition 3 and serves as a scaling factor that makes it less likely for small centers to exceed the CoP limit, τ.

Intuitively, the center balances competing incentives. On one hand, it seeks to maximize profit by performing more transplants, using cheaper bad kidneys, and providing reimbursable care. On the other hand, performance concerns and patient welfare impose constraints: (i) transplanting too many patients and using the bad kidney increases the likelihood of exceeding the CoP mortality limit; (ii) patients dislike excessive post-transplant care due to the marginal cost $\gamma > 0$. The center optimally trades off these incentives by adjusting the transplant decision $A(\tilde{x})$, kidney choice $k(\tilde{x})$, and post-transplant care $q(x,k)$. Next, I characterize $A^*(\tilde{x}), k^*(\tilde{x}), q^*(x,k)$ and present the proofs in Appendix.

[43] **Note:** The notation $q(x,k)$ is to indicate that post-transplant care is chosen when x and k are observed.

Proposition 3. *The optimal $q^*(x,k)$ is an implicit solution to the equation 14. $A^*(\tilde{x})$ takes the form of a cutoff strategy 17 and t^* is the transplant threshold where patients with $\tilde{x} \geq t^*$ will receive transplants and post-transplant care. Conversely, patients with $\tilde{x} < t^*$ will receive no transplants nor post-transplant care. The optimal kidney allocation $k^*(\tilde{x})$ is defined as:*

$$k^*(\tilde{x}) = \begin{cases} g & t^* \leq \tilde{x} < t_g^*, \\ b & \tilde{x} \geq t_g^*, \\ \textit{(no transplant)} & \tilde{x} < t^*. \end{cases}$$

where t_g^*, the good kidney threshold, is the root to equation 16.

Because the center cannot observe a patient's true health x and instead relies on the noisy signal $\tilde{x}$, Proposition 3 implies a negative□sorting allocation rule based on $\tilde{x}$. Specifically, patients whose signals lie in an intermediate range, $\tilde{x} \in [t^*, t_g^*)$, receive the safer (good) kidney, while patients with strong signals, $\tilde{x} \geq t_g^*$, receive the riskier (bad) kidney. The intuition is that for borderline (moderate) signals, the good kidney's lower mortality risk ($\sigma_g < \sigma_b$) provides a significant survival benefit that justifies incurring its waiting cost g. By contrast, for sufficiently high signals $\tilde{x} \geq t_g^*$, that survival benefit diminishes and no longer outweighs g, prompting the center to assign the cheaper (bad) kidney. This tradeoff in expected benefit versus cost naturally yields a cutoff $\tilde{x} = t_g^*$ above which the center switches from good to bad kidneys.

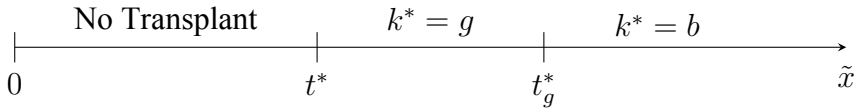
B.2 Comparative statics

In this stylized model, the pre-CoP announcement reflects $\tau \to \infty$, meaning no effective regulatory constraints on the product of transplants and mortality, allowing centers to optimize without restrictions. The post-CoP announcement reflects $\tau < \infty$, introducing binding regulatory constraints. The following result illustrates the comparative statics for the transplant threshold t^*, kidney choice t_g^*, and post-transplant care $q^*(x,k)$ as CMS announces CoP (i.e., τ decreases). I present the proofs in the Appendix.

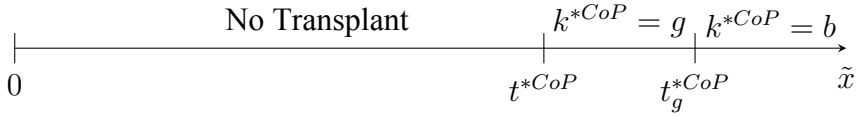
Proposition 4. *As CMS announces CoP (i.e., τ decreases), the transplant threshold t^* increases $\left(\frac{\partial t^*}{\partial \tau} < 0\right)$; post-transplant care $q^*(x,k)$ increases $\left(\frac{\partial q^*(x,k)}{\partial \tau} < 0\right)$; the good kidney threshold t_g^* increases $\left(\frac{\partial t_g^*}{\partial \tau} < 0\right)$.*

Proposition 4 predicts that as CMS announces CoP, the fraction of patients receiving a transplant decreases. However, this does not imply that enters are actively selecting healthier patients. Instead, the higher threshold t^* makes it more likely for a patient with better true health x to surpass it. Consequently, average health among the smaller set of transplanted patients rises (i.e., $E[x|\tilde{x} > t^*)$ is increasing in t^*). The extent of this rise depends on the informativeness of the noisy signal $\tilde{x}$. When $\tilde{x}$ closely tracks x (i.e., low Var(u)), the stricter threshold effectively excludes less-healthy patients, strongly skewing the transplanted group toward high health. Conversely, if $\tilde{x}$ is weakly informative (i.e., high Var(u)), the higher threshold barely alters the health composition of transplanted patients.

Furthermore, Proposition 4 predicts fewer bad kidney transplants after CMS implements CoP. Using Figure B.II as an example, this decrease is because the center substitutes the bad kidneys with the good kidneys for patients with a strong signal, $\tilde{x} \in [t_g^*, t_g^{*CoP}]$. However, this does not imply more good kidney transplants because patients with intermediate signal, $\tilde{x} \in [t^*, t^{*CoP}]$ will not receive a transplant due to more stringent performance limits. Thus, the effect of CoP on good kidney transplants is ambiguous and depends on the model's parameter values (e.g., high/low waiting cost, g).



(a) Kidney matching when CoP limit, $\tau \to \infty$ (before CoP)



(b) Kidney matching when CoP limit, $\tau < \infty$ (after CoP)

Figure B.II: Kidney matching before CoP v.s. after CoP

Note: Panel A depicts the scenario when the CoP limit is not stringent (e.g., $\tau \to \infty$). Panel B depicts the scenario when the CoP limit is very stringent (e.g., $\tau < \infty$). The model predicts fewer bad kidney transplants because centers substitute the bad kidneys for the good kidneys for patients with a strong signal, $\tilde{x} \in [t_g^*, t_g^{*CoP}]$. On the other hand, patients with intermediate signal, $\tilde{x} \in [t^*, t^{*CoP}]$, do not receive a transplant. Thus, the effect of CoP on good kidney transplants is ambiguous and depends on the parameter value of the model (e.g., high/low waiting cost, g).

B.3 Proofs for Proposition 3 and 4

From equation 12, the center's objective function is

$$\max_{A(\tilde{x}),k\in\{g,b\},q} \int_{\tilde{x}} A(\tilde{x}) \int_{x} \overbrace{\left[\rho \overbrace{[\pi + \alpha q]}^{\text{center profit}} \quad + \quad (1-\rho) \overbrace{\left[xq - \frac{\gamma}{2} q^2 - \mathbf{1}_{\{k=g\}} g \right]}^{\text{patient utility}} \right]}^{\Pi(x,k,q)} p(x|\tilde{x}) dx dF(\tilde{x})$$

$$\text{s.t.} \int_{\tilde{x}} A(\tilde{x}) \left[\int_{x} P(x,k,q) p(x|\tilde{x}) dx \right] dF(\tilde{x}) \leq \tau \tag{13}$$

We solve the maximization problem via backwards induction.

B.3.1 Solving for q^*

Let $\lambda \geq 0$ be the Lagrange multiplier on the constraint. Define the Lagrangian:

$$\mathcal{L} = \int_{\tilde{x}} A(\tilde{x}) \int_{x} \Big[\Pi(x,k,q) \Big] p(x|\tilde{x}) dx \, dF(\tilde{x}) \; - \; \lambda \Big[\int_{\tilde{x}} A(\tilde{x}) \int_{x} P(x,k,q) p(x|\tilde{x}) dx dF(\tilde{x}) - \tau \Big].$$

Step 1: If the constraint is slack ($\lambda = 0$). For each x, we differentiate $\Pi(x,k,q)$ with respect to q:

$$\rho\,\alpha + (1-\rho)\big[x - \gamma\, q\big] = 0 \quad \Longrightarrow \quad (1-\rho)\,\gamma\, q = \rho\,\alpha + (1-\rho)\,x.$$

Hence

$$q^{\text{uncon}}(x) = \frac{\rho\,\alpha + (1-\rho)\,x}{(1-\rho)\,\gamma}.$$

Step 2: If the constraint binds ($\lambda > 0$). For each x, we need

$$\frac{\partial \mathcal{L}}{\partial q} = \int A(\tilde{x}) \frac{\partial \Pi(.)}{\partial q} p(x|\tilde{x}) dx dF(\tilde{x}) - \lambda \int A(\tilde{x}) \frac{\partial P(.)}{\partial q} p(x|\tilde{x}) dF(\tilde{x}) = 0$$

where

$$\frac{\partial}{\partial q} \overbrace{\Big\{ \rho(\pi + \alpha\, q) + (1-\rho)\big[x\, q - \tfrac{\gamma}{2} q^2 - \mathbf{1}_{\{k=g\}} g\big] \Big\}}^{\Pi(x,k,q)} = \rho\, \alpha + (1-\rho)\big[x - \gamma\, q\big].$$

and

$$\frac{\partial}{\partial q} \overbrace{\Big[1 - \Phi\big(\tfrac{x+q}{\sigma_k}\big)\Big]}^{P(x,k,q)} = -\,\phi\Big(\tfrac{x+q}{\sigma_k}\Big)\, \frac{1}{\sigma_k},$$

Rearrange for q^*:

$$(1-\rho)\, \gamma\, q^* = \rho\, \alpha + (1-\rho)\, x + \lambda\, \frac{1}{\sigma_k}\, \phi\Big(\tfrac{x+q^*}{\sigma_k}\Big).$$

Thus we have the *implicit* solution:

$$q^*(x,k) = \frac{\rho\, \alpha + (1-\rho)\, x}{(1-\rho)\, \gamma} \;+\; \frac{\lambda}{(1-\rho)\, \gamma\, \sigma_k}\, \phi\Big(\tfrac{x+q^*(x,k)}{\sigma_k}\Big). \tag{14}$$

If $\lambda = 0$, we revert to the unconstrained optimum. Otherwise, $q^*(x,k)$ exceeds the unconstrained level, reflecting a desire to reduce mortality.

B.3.2 Solving for $k^*(\tilde{x})$

Upon seeing $\tilde{x}$, the center forms a posterior over x, where $p(x|\tilde{x})$ is derived from Bayes' rule, with priors $x \sim N(\mu_x, \sigma_x^2)$ and $u \sim N(0, \sigma_u^2)$. x and u are assumed to be independent. $\Pi(x,k,q^*)$ is the payoff for a transplanted patient of true health x given kidney k as defined in the previous section. Thus, the center chooses k^* at each $\tilde{x}$ such that:

$$k(\tilde{x}) = \arg\max_{k \in \{g,b\}} \overbrace{\int \left[\Pi(x,k,q^*) \;- \lambda P(x,k,q^*)\right] p(x|\tilde{x})\, dx}^{\widetilde{\Pi}(x,k,q^*)} \tag{15}$$

Next, we define:

$$D(\tilde{x}) = \widetilde{\Pi}(x, g, q^*) - \widetilde{\Pi}(x, b, q^*) \tag{16}$$

As $\tilde{x}$ increases, the posterior shifts to higher x. Since $\widetilde{\Pi}(x, g, q^*)$ and $\widetilde{\Pi}(x, b, q^*)$ differ mainly by the cost g and the difference in survival benefits, then $D(\tilde{x})$ is decreasing in $\tilde{x}$: when $\tilde{x}$ is large, the expected incremental survival benefit of g is smaller, so $D(\tilde{x})$ may become negative, favoring kidney b. Thus, $D(\tilde{x})$ crosses zero exactly once, giving a unique cutoff t_g^*. We have the following cutoff rule:

$$k^*(\tilde{x}) = \begin{cases} g & t \leq \tilde{x} < t_g^*, \\ b & \tilde{x} \geq t_g^*, \\ \text{(no transplant)} & \tilde{x} < t^*. \end{cases}$$

B.3.3 Solving for the acceptance rule $A(\tilde{x})$

Define the net benefit function, $NB(\tilde{x})$

$$NB(\tilde{x}) = \int \Pi(x, k, q) p(x|\tilde{x}) dx - \lambda \int P(x, k, q) p(x|\tilde{x}) dx$$

Since the posterior distribution of $p(x|\tilde{x})$ is increasing in $\tilde{x}$, $NB(\tilde{x})$ is a monotonic function of $\tilde{x}$, $A(\tilde{x})$ takes the form of a cutoff strategy:

$$A(\tilde{x}) = \begin{cases} 1 & \text{if } \tilde{x} \geq t^* \\ 0 & \text{if } \tilde{x} < t^* \end{cases} \tag{17}$$

where t^* is such that $NB(t^*) = 0$. This completes the proof for proposition 3.

B.3.4 Comparative Statics: Effect of Decreasing τ

As τ decreases, the regulatory constraint tightens, and the Lagrange multiplier λ increases. This forces the center to reduce the product

$$(\#\text{ transplanted}) \times (\#\text{ expected deaths}).$$

They can do this in two ways:

- **Raise t^* (fewer transplants).** Since

$$NB(\tilde{x}) = \int \Pi(x, q(x))p(x|\tilde{x})dx - \lambda \int P(x, q(x))p(x|\tilde{x})dx$$

  increases in $\tilde{x}$, a higher threshold means fewer people qualify for a transplant.

- **Raise t_g^* (fewer bad kidney transplants).** From

$$D(\tilde{x}) = \widetilde{\Pi}(x, g, q^*) - \widetilde{\Pi}(x, b, q^*)$$

  a larger λ means centers can afford fewer expected deaths than before. This reduces the marginal benefit of the bad kidney and raises t_g^*

- **Raise q^* (improve post-transplant care).** From

$$q^*(x,k) = \frac{\rho\,\alpha + (1-\rho)\,x}{(1-\rho)\,\gamma} + \frac{\lambda}{(1-\rho)\,\gamma\,\sigma_k}\,\phi\Big(\tfrac{x+q^*(x)}{\sigma_k}\Big),$$

  a larger λ makes $q^*(x,k)$ bigger for each x—the center "overspends" on care (relative to the unconstrained level) to reduce mortality.

This completes the proof for proposition 4.

C Supplementary Figures

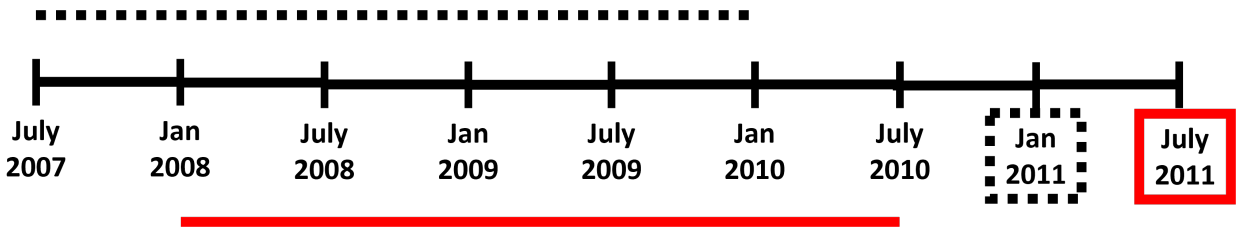


Figure C.III: An illustration of the rolling 2.5-year cohort for CoP

Note: The January 2011 submission (black box) consists of transplants from July 1, 2007, to December 31, 2009 (black line). Similarly, the July 2011 submission (red box) contains transplants from January 1, 2008, and June 31, 2010 (red line).

Line		Center 1 Year	National 1 Year
	Adult (Age 18+)		
1	Transplants (n=number)	90	10,781
2	**Percent (%) of Patients Surviving at End of Period**		
3	Observed at this Center	87.78	86.26
4	Expected, based on national experience	89.41	
5	**Deaths During Follow-up Period**		
6	Observed at this center	11	1,392
7	Expected, based on national experience	8.48	1,392
8	Ratio: Observed to Expected (O/E)	1.30	1.00
9	(95% Confidence Interval)	(0.65-2.32)	
10	P-value (2-sided), observed v. expected	0.469	
11	How does this center's survival compare to what is expected for similar patients?	Not Significantly Different (a)	
12	Percent retransplanted	5.5	4.4
13	Follow-up days reported by center (%)	91.7	93.9
14	Maximum Days of Follow-up (n)	365	365

Figure C.IV: An example of a transplant center's CoP report

Note: This table is from Dickinson et al. (2008) and provides an example of a center that did not get penalized for poor performance. The CoP conditions from Section 2.4 can be calculated from this table. For example, Condition 1 is in line 8 (e.g., $O/E = 1.3 < 1.5$); Condition 2 is calculated by taking the difference between lines 6 and 7 (e.g., $O - E = 2.52 < 3$); Condition 3 is in line 10 (e.g., $Pr(O = E) = 0.469 > 0.05$).

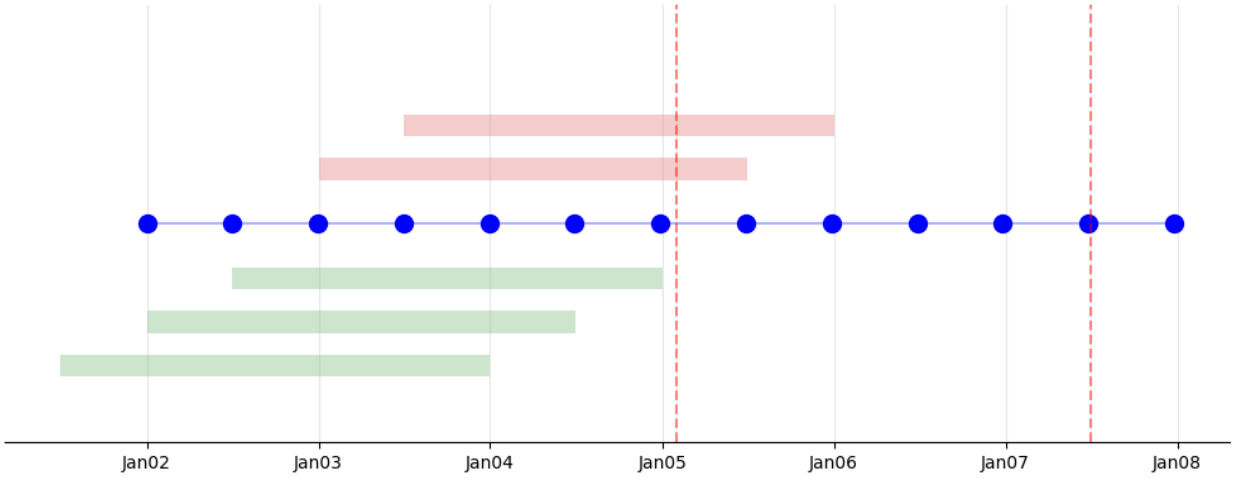


Figure C.V: CoP reports and their 2.5-years rolling cohorts

Note: The green bars highlight the 2.5-year rolling cohort for the penalty status of the CoP report in January 2005, July 2005, and January 2006. These reports are based on transplants before the CoP announcement (1st red dotted line). The red bars represent CoP reports from July 2006 and January 2007, which were built on 2.5-year rolling cohorts that overlapped with the CoP announcement.

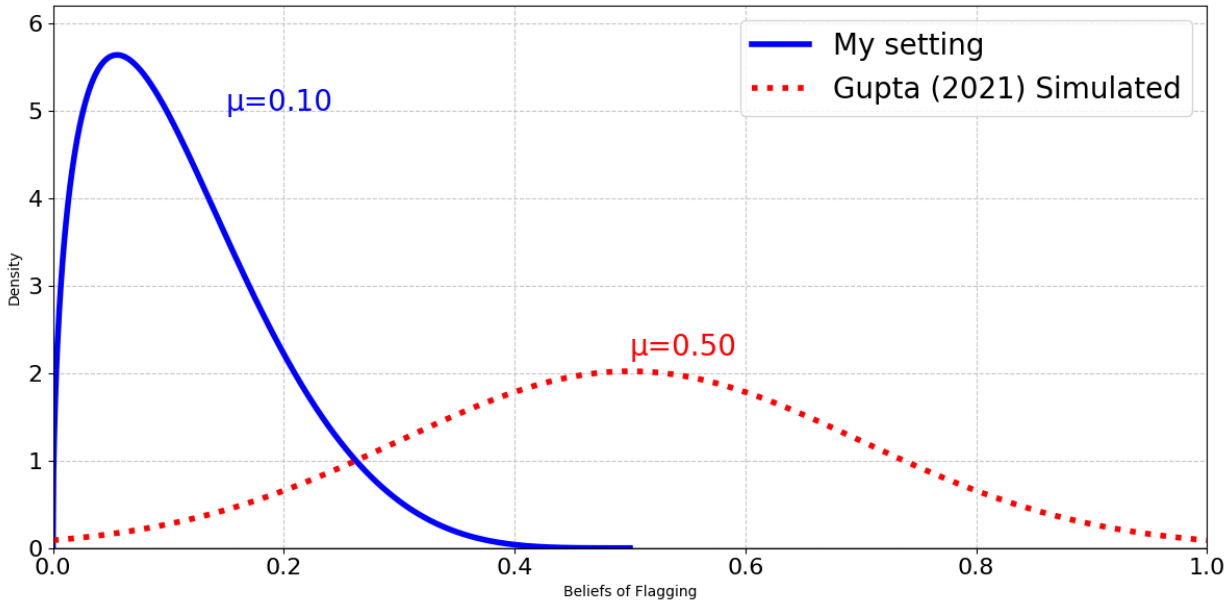


Figure C.VI: Density of actual penalty beliefs in my setting versus simulated beliefs in Gupta (2021)

Note: The blue solid lines are the density of my estimated penalty beliefs ρ_c. The density is similar to a beta distribution with parameters $(\alpha = 1.5, \beta = 5)$ and support $[0, 0.5]$. The red dotted lines are simulated density for Gupta (2021) using a truncated standard normal density with parameter $(\mu = 0.5, \sigma = 0.2)$ and support $[0, 1]$

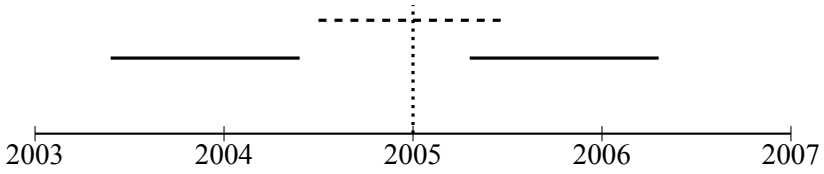
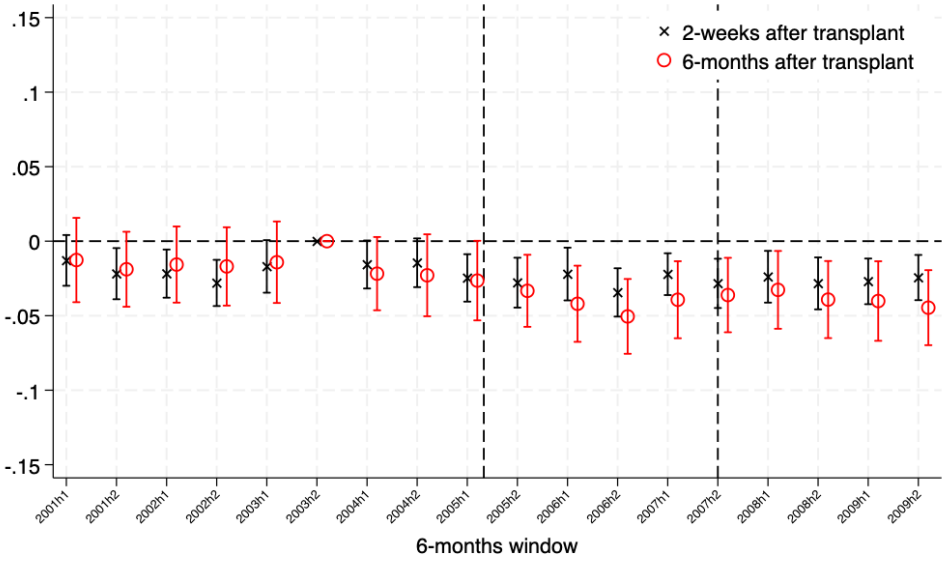


Figure C.VII: Non-overlapping (solid) and overlapping (dashed) patients

Note: This figure highlights my regression subsample as described in Section 5.3. The length of the lines indicates the patient's post-transplant mortality timeline and varies according to the outcome of interest. Patients whose post-transplant mortality timeline does not overlap with the CoP announcement have solid lines, and those with dashed lines have post-transplant mortality that overlaps with the CoP announcement.

Panel A



Panel B

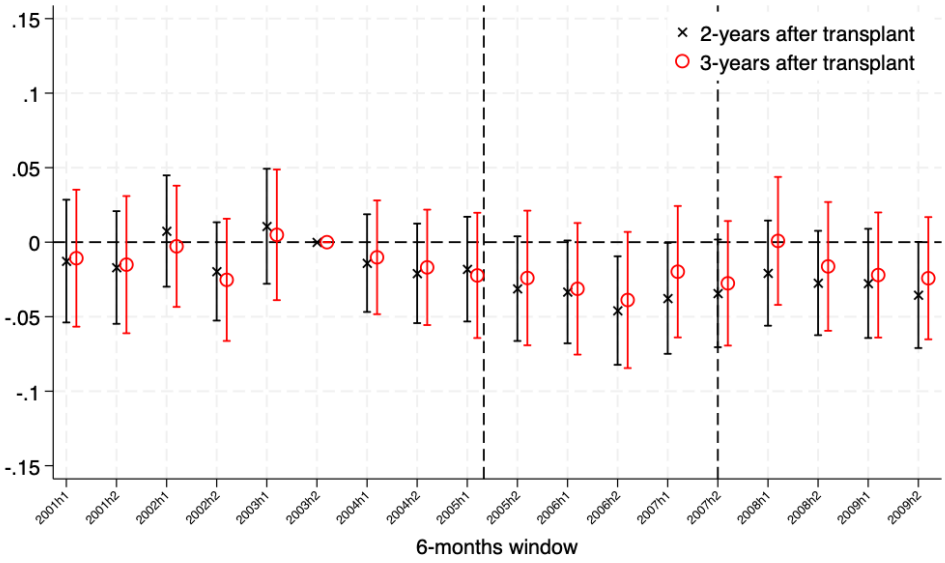
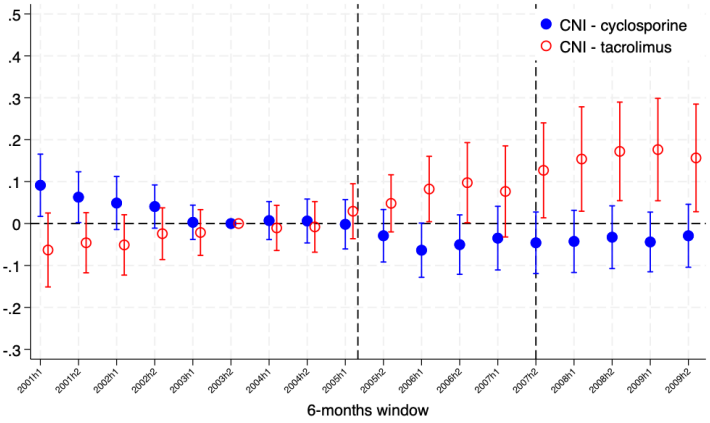


Figure C.VIII: Impact on post-transplant mortality at different periods

Note: The figure presents the estimated effect on post-transplant mortality at 2-week/6-month/2-year/3-year, obtained using equation 6 with the instrument Z_c and 2003h2 as the reference 6-month window. I cluster standard errors at the transplant center level. Error bars indicate 95 percent confidence intervals.

Panel A: 6-months follow-up



Panel B: 1-year follow-up

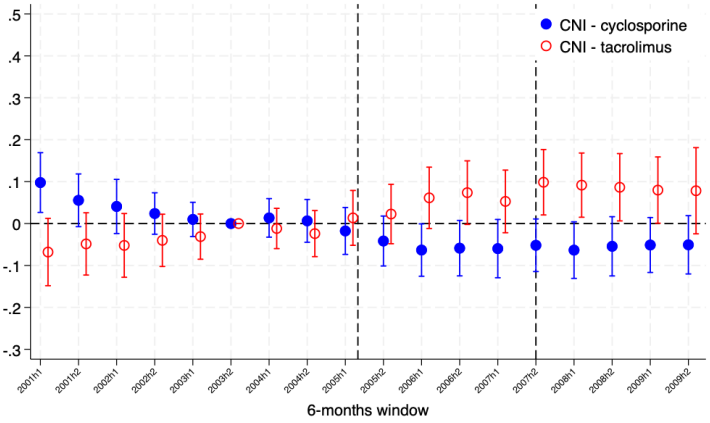


Figure C.IX: Impact on maintenance immunosuppressant - tacrolimus and cyclosporine prescription at different follow-up intervals

Note: The figure presents the estimated effect on tacrolimus and cyclosporine prescription at 6-months and 1-year follow-up, obtained using equation 6 with the instrument Z_c and 2003h2 as the reference 6-month window. I cluster standard errors at the transplant center level. Error bars indicate 95 percent confidence intervals.

D Supplementary Tables

Table D.I: Death rates among patients missing follow-up care

Time Period	Pre-CoP	Post-Announce	Post-Implement
Dead within 2 weeks	100.0%	100.0%	100.0%
	(N=593)	(N=398)	(N=357)
Dead within 6 months	92.0%	92.8%	95.3%
	(N=3078)	(N=1996)	(N=1731)
Dead within 1 year	94.0%	95.0%	95.8%
	(N=4451)	(N=2950)	(N=2580)

Notes: This table shows the proportion of patients who missed their follow-up appointments due to death. Death rates are calculated as the proportion of patients who died within the specified timeframe among those who did not show up for their scheduled follow-up care.

Table D.II: Tranplanted and discarded kidney characteristics pre and post-CoP

	Transplanted			Discarded		
	Pre-CoP	Post-Ann	Post-Impl	Pre-CoP	Post-Ann	Post-Impl
Age	35.6	36.7	37.0	52.5	54.1	52.7
	(17.3)	(17.1)	(17.3)	(17.0)	(16.2)	(16.5)
Creatinine Levels	1.1	1.1	1.1	1.4	1.4	1.5
	(1.0)	(0.7)	(0.9)	(1.1)	(0.9)	(1.2)
Kidney Risk	0.4	0.4	0.4	0.7	0.8	0.8
	(0.3)	(0.3)	(0.3)	(0.2)	(0.2)	(0.2)
Male	59.6%	60.5%	60.9%	52.2%	52.4%	53.3%
	(49.1)	(48.9)	(48.8)	(50.0)	(49.9)	(49.9)
White	72.4%	68.6%	68.0%	72.2%	68.5%	69.0%
	(44.7)	(46.4)	(46.7)	(44.8)	(46.5)	(46.3)
Death - Stroke	37.8%	36.3%	34.3%	65.9%	65.9%	58.6%
	(48.5)	(48.1)	(47.5)	(47.4)	(47.4)	(49.3)
Death - Head Trauma	47.2%	44.9%	41.3%	19.8%	17.1%	17.7%
	(49.9)	(49.7)	(49.2)	(39.9)	(37.7)	(38.2)
Hypertension	19.6%	24.1%	25.5%	52.7%	61.6%	60.3%
	(39.7)	(42.8)	(43.6)	(49.9)	(48.6)	(48.9)
Total Offers	95.0	61.8	139.9	796.4	470.9	1122.3
	(505.2)	(259.0)	(592.4)	(2352.7)	(915.9)	(2008.0)
Observations	37975	28099	28625	5750	5307	6508

Notes: This table presents means and standard deviations (in parentheses) for kidney donor characteristics. The sample is split between transplanted and discarded kidneys before and after the CoP announcement and implementation. If a pair of kidneys were recovered, but only one was transplanted, each would count as an observation in both the transplanted and discarded columns.

Table D.III: Tranplant patient characteristics pre and post-CoP

	Pre-CoP	Post-CoP	
		Announcement	Implementation
Age	47.9	49.3	50.2
	(14.5)	(15.3)	(15.3)
White	51.7%	48.7%	47.4%
	(50.0)	(50.0)	(49.9)
Years on WL	2.2	2.3	2.4
	(1.9)	(2.0)	(2.1)
Completed Univ.	14.2%	15.8%	17.4%
	(34.9)	(36.5)	(37.9)
Medicare	60.5%	61.0%	62.7%
	(48.9)	(48.8)	(48.4)
Diabetic	31.2%	33.5%	35.7%
	(46.4)	(47.2)	(47.9)
On Dialysis	55.9%	75.6%	76.8%
	(49.7)	(43.0)	(42.2)
Total Offers	55.9	69.2	95.1
	(72.3)	(100.8)	(147.7)
Expected Post-TX Survival	31.2	34.9	37.7
	(29.7)	(30.9)	(31.9)
Observations	36446	27052	27356

Notes: This table presents means and standard deviations (in parentheses) for transplant patient characteristics. The three columns cover transplants performed over the pre-CoP (January 2001 - February 2005), post-CoP announcement (February 2005 - July 2007), and post-CoP implementation (July 2007 - July 2009) periods.

Table D.IV: Impact on different post-transplant timeline mortality

	Post-transplant $\leq$ 1-year			Post-transplant $>$ 1-year	
	(1)	(2)	(3)	(4)	(5)
	2-weeks	6-months	1-year	2-years	3-years
Post-Announce	-0.01044***	-0.02695***	-0.02782**	-0.00943	0.00986
	(0.00302)	(0.00598)	(0.01031)	(0.01660)	(0.02124)
Post-Implement	-0.01330***	-0.02820***	-0.02112*	-0.00103	0.02895
	(0.00383)	(0.00727)	(0.01061)	(0.01601)	(0.02201)
Y mean	0.03028	0.08074	0.11767	0.18191	0.24566
F-statistic	57.05134	56.98470	56.76781	56.27769	55.55284
Fixed Effects	Center, 6-months	Center, 6-months	Center, 6-months	Center, 6-months	Center, 6-months
Observations	87371	83137	78832	70774	62880

Note: This table relates to the analysis in Section 6.1. It presents the estimated effect on the probability of deaths at different post-transplant timelines, obtained by jointly estimating equations 4 and 5. Each column uses the subsample of patients whose post-transplant mortality timeline does not overlap with the CoP announcement described in Section 5.3. I cluster standard errors at the transplant center level. *$p < 0.05$, **$p < 0.01$, *** $p < 0.001$

Table D.V: Impact on acceptance decision across patient subgroups

	(1) Age>65	(2) BMI>30	(3) Prior TX	(4) On dialysis	(5) Medicare
Post-Ann (Tri)	0.00879	0.00385	0.00461	0.00551	0.00353
	(0.00557)	(0.00393)	(0.00539)	(0.00414)	(0.00360)
Post-Imp (Tri)	0.00359	0.00536	0.00288	0.00403	0.00928
	(0.00648)	(0.00417)	(0.00645)	(0.00509)	(0.00527)
Y mean	0.08273	0.08273	0.08273	0.08273	0.08273
Fixed Effects	Center, 6-months	Center, 6-months	Center, 6-months	Center, 6-months	Center, 6-months
Observations	646983	646983	646983	646983	646983

Note: This table relates to the analysis in section 6.2.1. It presents the estimated effect on the center's acceptance decision across different subgroups, obtained from triple differences regression interacted with different patient characteristics included in the risk-adjustment model. I cluster standard errors at the transplant center level. *$p < 0.05$, **$p < 0.01$, *** $p < 0.001$

Table D.VI: Impact on acceptance decision across kidney subgroups

	(1) Age>65	(2) Diabetic	(3) Hypertension	(4) Death-Stroke	(5) Death-Head Trauma
Post-Ann (Tri)	0.01489**	0.00708	0.00623	0.00004	-0.00108
	(0.00568)	(0.00431)	(0.00455)	(0.00325)	(0.00379)
Post-Imp (Tri)	0.00668	0.00390	0.00538	0.00248	-0.00154
	(0.00754)	(0.00469)	(0.00465)	(0.00425)	(0.00527)
Y mean	0.08273	0.08273	0.08273	0.08273	0.08273
Fixed Effects	Center, 6-months	Center, 6-months	Center, 6-months	Center, 6-months	Center, 6-months
Observations	646983	646983	646983	646983	646983

Note: This table relates to the analysis in section 6.2.1. It presents the estimated effect on the center's acceptance decision across different subgroups, obtained from triple differences regression interacted with different kidney characteristics included in the risk-adjustment model. I cluster standard errors at the transplant center level. *$p < 0.05$, **$p < 0.01$, *** $p < 0.001$

Table D.VII: Impact on acceptance decision across non-adjusted patient or kidney subgroups (i)

	(1) Pat - Uni. Grad	(2) Pat - Malignant	(3) Kid - Cancer Hist.	(4) Kid - BMI>30
Post-Ann (Tri)	0.00356	-0.01443	0.00508	0.00582*
	(0.00500)	(0.00861)	(0.00574)	(0.00289)
Post-Imp (Tri)	0.00141	-0.00593	0.01158	-0.00091
	(0.00460)	(0.00865)	(0.00635)	(0.00296)
Y mean	0.08273	0.08273	0.08273	0.08273
Fixed Effects	Center, 6-months	Center, 6-months	Center, 6-months	Center, 6-months
Observations	646983	625011	646983	646983

Note: This table relates to the analysis in section 6.2.1. It presents the estimated effect on the center's acceptance decision across different subgroups, obtained from triple differences regression interacted with various non-adjusted patient and kidney characteristics. I cluster standard errors at the transplant center level. *$p < 0.05$, **$p < 0.01$, *** $p < 0.001$

Table D.VIII: Impact on acceptance decision across non-adjusted patient or kidney subgroups (ii)

	(1) Risky Pat.	(2) Risky Kid.	(3) Risky Pat. x Kid.
Post-Ann (Tri)	0.00410	0.00851	0.00722
	(0.00511)	(0.00559)	(0.00475)
Post-Imp (Tri)	0.00709	0.00524	0.00652
	(0.00475)	(0.00696)	(0.00493)
Y mean	0.08273	0.08273	0.08273
Fixed Effects	Center, 6-months	Center, 6-months	Center, 6-months
Observations	642699	642699	642699

Note: This table relates to the analysis in section 6.2.1. It presents the estimated effect on the center's acceptance decision across different subgroups, obtained from triple differences regression interacted with predetermined patient and kidney risk measures. I use the kidney donor profile index (KDPI) and estimated post-transplant survival (EPTS) as measures of kidney and patient risk, respectively. A kidney is risky if $KDPI \geq 0.5$, and a patient is risky if $EPTS \geq 0.5$. A patient-kidney pair is considered high-risk if both the patient and the kidney are high-risk. I cluster standard errors at the transplant center level. *$p < 0.05$, **$p < 0.01$, *** $p < 0.001$

Table D.IX: Impact on kidney discard

	Kidney Discards		No. of Patients Offered	
	(1)	(2)	(3)	(4)
	Baseline	Risky Kidney	Baseline	Risky Kidney
Post-Announce	0.00755	-0.00737*	18.93656***	4.42829
	(0.00424)	(0.00331)	(4.36242)	(4.07778)
Post-Implement	-0.00557	-0.01254***	-58.35275***	-24.85710***
	(0.00443)	(0.00353)	(6.74354)	(5.99234)
Post-Announce (Tri)		0.03575***		37.87207***
		(0.00956)		(9.02274)
Post-Implement (Tri)		0.01550		-75.35143***
		(0.00980)		(13.64219)
Y mean	0.15008	0.15008	222.44305	222.44305
Fixed Effects	DSA, 6-months	DSA, 6-months	DSA, 6-months	DSA, 6-months
Observations	76304	76304	76304	76304

Note: This table relates to the analysis in Section 6.2.2. It presents the estimated effect on kidney discard, obtained by estimating equation 8. I use the kidney donor profile index (KDPI) as a measure of kidney risk. A kidney is risky if $KDPI \geq 0.5$. I cluster standard errors at the donor service area level. *$p < 0.05$, **$p < 0.01$, *** $p < 0.001$

Table D.X: Impact on the prescription of maintenance immunosuppressants - Cyclosporine

	(1) 2-weeks	(2) 6-months	(3) 1-year	(4) 2-years
Post-Announce	-0.05704*	-0.04914*	-0.05119*	-0.05327
	(0.02801)	(0.02351)	(0.02410)	(0.02770)
Post-Implement	-0.08137*	-0.06742*	-0.06505*	-0.06560*
	(0.03308)	(0.02972)	(0.02988)	(0.03073)
Y mean	0.15978	0.14875	0.14809	0.11643
F-statistic	55.20009	54.06380	51.35394	50.61915
Fixed Effects	Center, 6-months	Center, 6-months	Center, 6-months	Center, 6-months
Observations	82768	72703	66157	55193

Note: This table relates to the analysis in Section 6.3.1. It presents the estimated effect on the probability of prescribing cyclosporine at different follow-up timelines, obtained by jointly estimating equations 4 and 5. Each column uses the subsample of patients whose post-transplant mortality timeline does not overlap with the CoP announcement described in Section 5.3. I cluster standard errors at the transplant center level. *$p < 0.05$, **$p < 0.01$, ***$p < 0.001$.

Table D.XI: Impact on the prescription of maintenance immunosuppressants - Tacrolimus

	(1) 2-weeks	(2) 6-months	(3) 1-year	(4) 2-years
Post-Announce	0.05705	0.08183**	0.06288*	0.10958*
	(0.02926)	(0.02933)	(0.03021)	(0.05171)
Post-Implement	0.10307**	0.14781**	0.10432**	0.13865*
	(0.03673)	(0.05245)	(0.03346)	(0.05935)
Y mean	0.75790	0.69038	0.76061	0.61734
F-statistic	55.20009	54.06380	51.35394	50.61915
Fixed Effects	Center, 6-months	Center, 6-months	Center, 6-months	Center, 6-months
Observations	82768	72703	66157	55193

Note: This table relates to the analysis in Section 6.3.1. It presents the estimated effect on the probability of prescribing tacrolimus at different follow-up timelines, obtained by jointly estimating equations 4 and 5. Each column uses the subsample of patients whose post-transplant mortality timeline does not overlap with the CoP announcement described in Section 5.3. I cluster standard errors at the transplant center level. *$p < 0.05$, **$p < 0.01$, ***$p < 0.001$.

Table D.XII: Impact on the prescription of maintenance immunosuppressants

	(1) Cyclosporine	(2) Tacrolimus
Post-Announce	-0.00620**	0.01440***
	(0.00218)	(0.00266)
Y mean	0.21836	0.67635
Fixed Effects	Patients, 6-months	Patients, 6-months
Observations	100543	100543

Note: This table relates to the analysis in Section 6.3.1. It presents the estimated effect on the probability of prescribing cyclosporine or tacrolimus at different follow-up timelines, obtained by estimating equation 7. Each column uses the subsample of patients whose post-transplant mortality timeline overlaps with the CoP announcement as described in Section 5.3. I cluster standard errors at the patient level. *$p < 0.05$, **$p < 0.01$, ***$p < 0.001$.

Table D.XIII: Impact on deaths by kidney rejections

	(1) 2-weeks	(2) 6-months	(3) 1-year	(4) 2-years
Post-Announce	-0.00004	-0.00047	-0.00182	0.00055
	(0.00087)	(0.00154)	(0.00176)	(0.00251)
Post-Implement	-0.00163	-0.00456*	-0.00528*	-0.00432
	(0.00115)	(0.00216)	(0.00250)	(0.00292)
Y mean	0.00287	0.00845	0.01256	0.01903
F-statistic	55.26085	55.21618	55.02377	54.60244
Fixed Effects	Center, 6-months	Center, 6-months	Center, 6-months	Center, 6-months
Observations	82266	78032	73727	65669

Note: This table relates to the analysis in Section 6.3.1. It presents the estimated effect on the probability of death due to kidney rejection at different post-transplant timelines, obtained by jointly estimating equations 4 and 5. Each column uses the subsample of patients whose post-transplant mortality timeline does not overlap with the CoP announcement described in Section 5.3. I cluster standard errors at the transplant center level. *$p < 0.05$, **$p < 0.01$, *** $p < 0.001$

Table D.XIV: Impact on deaths by viral infections

	(1) 2-weeks	(2) 6-months	(3) 1-year	(4) 2-years
Post-Announce	-0.00438*	-0.00982**	-0.01079**	-0.01200**
	(0.00203)	(0.00334)	(0.00383)	(0.00447)
Post-Implement	-0.00627***	-0.01077***	-0.01214***	-0.01433***
	(0.00179)	(0.00272)	(0.00323)	(0.00368)
Y mean	0.00959	0.01922	0.02336	0.02834
F-statistic	55.26085	55.21618	55.02377	54.60244
Fixed Effects	Center, 6-months	Center, 6-months	Center, 6-months	Center, 6-months
Observations	82266	78032	73727	65669

Note: This table relates to the analysis in Section 6.3.2. It presents the estimated effect on the probability of death by viral infection at different post-transplant timelines, obtained by jointly estimating equations 4 and 5. Each column uses the subsample of patients whose post-transplant mortality timeline does not overlap with the CoP announcement described in Section 5.3. I cluster standard errors at the transplant center level. *$p < 0.05$, **$p < 0.01$, *** $p < 0.001$

Table D.XV: Impact on non-targeted patient outcomes (i)

	(1) Hospitalization	(2) Acute Kidney Rejection	(3) Dialysis	(4) Diabetes
Post-Announce	0.03724*	0.01252	0.00003	0.00921
	(0.01759)	(0.00955)	(0.00144)	(0.01907)
Post-Implement	0.03701	0.01316	0.00090	-0.00818
	(0.02369)	(0.01079)	(0.00128)	(0.01799)
Y mean	0.22096	0.03613	0.00394	0.10450
F-statistic	51.35394	51.35394	51.35394	51.35394
Fixed Effects	Center, 6-months	Center, 6-months	Center, 6-months	Center, 6-months
Observations	66157	66157	66157	66157

Note: This table relates to the analysis in sections 6.3.2 and 7.2. It presents the estimated effect on non-targeted patient outcomes, obtained by jointly estimating equations 4 and 5. Each column represents a different patient outcome measured during the patient's 1-year follow-up. I cluster standard errors at the transplant center level. *$p < 0.05$, **$p < 0.01$, *** $p < 0.001$

Table D.XVI: Impact on non-targeted patient outcomes (ii)

	(1) Malignancy	(2) Kidney Failure	(3) Creatinine	(4) Positive CMV
Post-Announce	-0.00204	-0.00028	-0.01561	-0.00979
	(0.00145)	(0.00064)	(0.01571)	(0.01817)
Post-Implement	0.00085	0.00062	-0.04744*	0.00008
	(0.00192)	(0.00063)	(0.01907)	(0.02646)
Y mean	0.00710	0.00093	1.49528	0.63007
F-statistic	51.35394	51.35394	50.60337	51.35394
Fixed Effects	Center, 6-months	Center, 6-months	Center, 6-months	Center, 6-months
Observations	66157	66157	65006	66157

Note: This table relates to the analysis in sections 6.3.2 and 7.2. It presents the estimated effect on non-targeted patient outcomes, obtained by jointly estimating equations 4 and 5. Each column represents a different patient outcome measured during the patient's 1-year follow-up. I cluster standard errors at the transplant center level. *$p < 0.05$, **$p < 0.01$, *** $p < 0.001$

Table D.XVII: Impact of compulsory documentation on mortality and transplant decision

	Post-transplant mortality		Pat-Kid Acceptance	
	(1)	(2)	(3)	(4)
	Teaching Center	Large Center	Teaching Center	Large Center
Post-Ann (Tri)	0.00294	-0.00154	-0.00392	0.00296
	(0.01474)	(0.01833)	(0.00869)	(0.00840)
Post-Imp (Tri)	-0.00543	-0.02449	0.00313	-0.00181
	(0.01528)	(0.01984)	(0.01037)	(0.01195)
Y mean	0.11904	0.11904	0.08273	0.08273
Fixed Effects	Center, 6-months	Center, 6-months	Center, 6-months	Center, 6-months
Observations	74175	74175	646983	646983

Note: This table relates to the analysis in section 7.1.1. It presents the estimated effects on post-transplant mortality/ the center's acceptance decision across different subgroups, obtained from triple-differences regression interacted with different administrative capacities. I cluster standard errors at the transplant center level. *$p < 0.05$, **$p < 0.01$, *** $p < 0.001$

Table D.XVIII: Impact on acceptable donor criteria (i)

	(1) Min. Age	(2) Max. Age	(3) HLA Mismatch	(4) Creatinine
Post-Announce	0.02900	-0.53994**	0.00647	0.63170
	(0.02441)	(0.16927)	(0.01274)	(0.67081)
Post-Implement	0.04581	1.04754**	0.00863	3.41333**
	(0.06314)	(0.36648)	(0.01437)	(1.27404)
Y mean	1.23015	80.92366	5.94159	21.81707
Fixed Effects	Patient, Quarters	Patient, Quarters	Patient, Quarters	Patient, Quarters
Observations	43723	43723	43025	43723

Note: This table relates to the analysis in section 7.1.2. It presents the estimated effect on centers setting more stringent donor criteria, obtained by estimating equation 7. Each column represents a different modifiable donor criterion. I cluster standard errors at the patient level. *$p < 0.05$, **$p < 0.01$, *** $p < 0.001$

Table D.XIX: Impact on acceptable donor criteria (ii)

	(1) Hypertension	(2) Cold Ischemic Time	(3) Warm Ischemic Time	(4) Expanded Criteria
Post-Announce	-0.00006	2.32549*	-1.10574*	0.00396
	(0.00007)	(1.11263)	(0.55112)	(0.00735)
Post-Implement	-0.00039	9.47421***	0.29125	-0.02113*
	(0.00039)	(1.89632)	(1.09701)	(0.01041)
Y mean	0.99943	68.08462	65.95272	0.37520
Fixed Effects	Patient, Quarters	Patient, Quarters	Patient, Quarters	Patient, Quarters
Observations	43723	43723	35696	43723

Note: This table relates to the analysis in section 7.1.2. It presents the estimated effect on centers setting more stringent donor criteria, obtained by estimating equation 7. Each column represents a different modifiable donor criterion. I cluster standard errors at the patient level. *$p < 0.05$, **$p < 0.01$, *** $p < 0.001$

Table D.XX: Impact on admitted patient characteristics (i)

	(1) Age	(2) White	(3) No education	(4) Working	(5) Medicare
Post-Announce	-0.11703	-0.00313	-0.00085	-0.00905	0.01535
	(0.15165)	(0.00413)	(0.01724)	(0.00556)	(0.01056)
Post-Implement	-0.15069	-0.00183	-0.02809	0.00545	0.00568
	(0.16484)	(0.00536)	(0.02463)	(0.00859)	(0.01156)
Y mean	49.16298	0.48686	0.15842	0.18485	0.48500
Fixed Effects	Centers, 6-months	Centers, 6-months	Centers, 6-months	Centers, 6-months	Centers, 6-months
Observations	261313	261313	261313	261313	261313

Note: This table relates to the analysis in section 7.1.3. It presents the estimated effect on admitted patient characteristics, obtained by estimating equation 5. I cluster standard errors at the transplant center level. *$p < 0.05$, **$p < 0.01$, *** $p < 0.001$

Table D.XXI: Impact on admitted patient characteristics (ii)

	(1) BMI	(2) Diabetic	(3) On dialysis	(4) Blood type O
Post-Announce	-0.13743**	-0.00008	-0.00236	0.12877
	(0.05193)	(0.00606)	(0.00874)	(0.31113)
Post-Implement	-0.18323**	-0.00280	0.00498	0.36779
	(0.06640)	(0.00779)	(0.00975)	(0.33629)
Y mean	27.62767	0.39092	0.76889	48.56857
Fixed Effects	Centers, 6-months	Centers, 6-months	Centers, 6-months	Centers, 6-months
Observations	253966	261313	261313	261313

Note: This table relates to the analysis in section 7.1.3. It presents the estimated effect on admitted patient characteristics, obtained by estimating equation 5. I cluster standard errors at the transplant center level. *$p < 0.05$, **$p < 0.01$, *** $p < 0.001$

Table D.XXII: Impact on the rate of CMV testing

	(1) 2-weeks	(2) 6-months	(3) 1-year	(4) 2-years
Post-Announce	-0.01628	0.03586	0.03447	0.05219
	(0.01615)	(0.02627)	(0.02904)	(0.04475)
Post-Implement	0.00754	0.02102	0.05379	0.02890
	(0.02026)	(0.03059)	(0.05436)	(0.05679)
Y mean	0.94816	0.11720	0.24254	0.28579
F-statistic	55.20396	54.06380	51.35394	50.61915
Fixed Effects	Center, 6-months	Center, 6-months	Center, 6-months	Center, 6-months
Observations	83118	72703	66157	55193

Note: This table examines the proposed detection channel in section 7.1.4. It presents the estimated effect on the probability of centers performing a CMV test at different follow-up timelines, obtained by jointly estimating equations 4 and 5. Each column uses the subsample of patients whose post-transplant mortality timeline does not overlap with the CoP announcement described in Section 5.3. I cluster standard errors at the transplant center level. *$p < 0.05$, **$p < 0.01$, ***$p < 0.001$.

Table D.XXIII: Impact on patient waitlist experience

	Time on waitlist		Removed from waitlist	
	(1)	(2)	(3)	(4)
	OLS	IV	OLS	IV
Post-Ann	-0.12275	-0.26685**	0.00238	0.00464
	(0.09087)	(0.09936)	(0.00554)	(0.00586)
Y mean	2.79544	2.79544	0.09271	0.09271
F-statistic		134.14564		127.64148
Fixed Effects	Center, 6-months	Center, 6-months	Center, 6-months	Center, 6-months
Observations	107249	107252	186374	186376

Note: This table relates to the analysis in section 7.3. It presents the estimated effect on transplant wait time and waitlist mortality, obtained by estimating equations 4 and 5. I cluster standard errors at the transplant center level. *$p < 0.05$, **$p < 0.01$, *** $p < 0.001$

Table D.XXIV: Full patient sample

	Post-transplant mortality	Tacrolimus prescription	Cyclosporine prescription
2-weeks after transplant			
Post-Ann	-0.003	0.049**	-0.048**
	(0.002)	(0.022)	(0.022)
Post-Imp	-0.006**	0.070**	-0.061**
	(0.002)	(0.029)	(0.024)
6-months after transplant			
Post-Ann	-0.011***	0.060***	-0.047***
	(0.004)	(0.019)	(0.017)
Post-Imp	-0.013**	0.072***	-0.050**
	(0.005)	(0.027)	(0.019)
1-year after transplant			
Post-Ann	-0.011*	0.081**	-0.027
	(0.006)	(0.038)	(0.017)
Post-Imp	-0.011*	0.106**	-0.029
	(0.006)	(0.041)	(0.018)

Notes: This table relates to the analysis in section 7.3. It presents the estimated effect on post-transplant death, as well as the prescription of tacrolimus and cyclosporine, obtained by estimating equations 4, 5 with the full patient sample. I cluster standard errors at the transplant center level. *$p < 0.05$, **$p < 0.01$, *** $p < 0.001$

Table D.XXV: Transplant centers have perfect foresight

	Post-transplant mortality	Tacrolimus prescription	Cyclosporine prescription
2-weeks after transplant			
Post-Ann	-0.019***	0.111	-0.114
	(0.005)	(0.083)	(0.083)
Post-Imp	-0.025***	0.106	-0.138
	(0.006)	(0.110)	(0.092)
6-months after transplant			
Post-Ann	-0.052***	0.110	-0.067
	(0.012)	(0.075)	(0.067)
Post-Imp	-0.056***	0.127	-0.079
	(0.013)	(0.086)	(0.082)
1-year after transplant			
Post-Ann	-0.050**	-0.059	-0.030
	(0.022)	(0.102)	(0.076)
Post-Imp	-0.039*	-0.046	-0.042
	(0.022)	(0.095)	(0.081)

Notes: This table relates to the analysis in section 7.2. It presents the estimated effect on post-transplant death, as well as the prescription of tacrolimus and cyclosporine, obtained by estimating equations 4 and 5, but replacing ρ_c with the penalty status of the center in 2007h2 when Medicare implemented CoP. I cluster standard errors at the transplant center level. *$p < 0.05$, **$p < 0.01$, *** $p < 0.001$

Table D.XXVI: Transplant centers have time-varying penalty beliefs

	Post-transplant mortality	Tacrolimus prescription	Cyclosporine prescription
2-weeks after transplant			
Post-Ann	-0.001	0.014***	-0.011***
	(0.001)	(0.002)	(0.002)
Post-Imp	-0.000	0.024***	-0.013***
	(0.001)	(0.003)	(0.002)
6-months after transplant			
Post-Ann	-0.005***	0.014***	-0.012***
	(0.002)	(0.003)	(0.002)
Post-Imp	-0.004*	0.017***	-0.011***
	(0.002)	(0.003)	(0.003)
1-year after transplant			
Post-Ann	-0.006***	0.020***	-0.006***
	(0.002)	(0.003)	(0.002)
Post-Imp	-0.003	0.034***	-0.006**
	(0.003)	(0.004)	(0.003)

Notes: This table relates to the analysis in section 7.3 . It presents the estimated effect on post-transplant death, as well as the prescription of tacrolimus and cyclosporine, obtained by estimating equations 4 and 5, but replacing the ρ_c with time-varying penalty beliefs. I use robust standard errors. *$p < 0.05$, **$p < 0.01$, *** $p < 0.001$