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 the effectiveness of a policy designed to terminate 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 research design to estimate the policy's impact on patient outcomes and center behaviors. The policy reduced post-transplant mortality by 25% because centers strategically delayed the initiation of immunosuppressive therapy, preserving patients' immune function immediately after transplantation and reducing infection risks associated with premature immunosuppressant initiation. Furthermore, the policy incentivized centers to refine clinical protocols, reducing redundant testing by 50%. Importantly, centers did not adopt discriminatory or selective transplant practices in response to increased oversight. These findings illustrate how carefully structured regulatory oversight can enhance survival outcomes, clinical effectiveness, and resource efficiency in post-transplant care without compromising equitable access to transplantation.

JEL codes: I11, I18, L38

Keywords: quality regulation, kidney transplant, mortality rates, medicare

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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 flagged 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 dedicating resources to

[1]CMS spent 36 billion USD 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.

early detection and optimizing immunosuppression regimens. 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 can be attributed to improvements in post-transplant care versus 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 seeks to maximize profit by performing as many transplants and providing as much post-transplant care as possible. 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 2012, covering approximately four years before, two years after the 2005 CoP announcement, and five years after the 2007 implementation. The follow-up data tracks each transplant patient's health status and records all prescriptions and medical tests performed during the revisits. The patient-

[3]Source: New York Times

[4]For examples, patient's coinsurance kick in or increasing opportunity cost on the patient's time.

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 flagging status, with key center-level characteristics, offering critical insights into how centers were evaluated under CoP.

The research design exploits two sources of policy-driven variation. First, the announcement and delayed implementation affect centers differentially and create 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 flagging likelihood. This measure incorporates the intensive margin of the penalty incentive, i.e., centers with excellent recent performance expect a lower likelihood of flagging.

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, mitigating measurement error concerns. The instrument is a predicted mortality rate based on patient-kidney factors estimated with transplant samples from 2002-2004. All else equal, centers with a higher proportion of these patients were more likely to be penalized. 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 nonparametric estimates of dynamic effects on all key outcomes.

The baseline IV estimates imply that after Medicare announced CoP, a one-standard deviation increase in center belief caused a 2.7 percentage point (pp) (25%) decrease in post-transplant 1-year mortality. The pattern persisted even after Medicare 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 quantify the role of post-transplant care. I use follow-up data to examine how centers prevent and detect acute kidney rejection. First, centers delayed immunosuppressant prescriptions until follow-up visits rather than administering them immediately after transplant. This preserved the patient's immune function post-transplant and reduced infection risk. Beyond medication adjustments, centers refined their diagnostic testing strategies in response to the policy. Initially, centers increased diagnostic testing after Medicare announced CoP, but they later reduced supplementary testing by 50% after determining it did not improve kidney rejection detection. These findings illustrate a transitory period during which centers adapted their clinical practices to achieve more efficient post-transplant care.

Next, I examine three potential selection channels. First, centers can set acceptable donor criteria to filter kidney offers for their patients. My analysis finds no evidence that centers implemented more stringent donor criteria to restrict kidney offers. Secondly, the absence of filtering does not preclude selection; centers might want to evaluate potential kidney offers before deciding. Indeed, after Medicare announced CoP, centers became 7% less likely to transplant a given patient-kidney pair, though this reduction dissipated once CoP was implemented. Notably, there was no indication of systematic discrimination against riskier transplant profiles. Finally, I investigate whether centers strategically admitted patients with more favorable characteristics or demographics but find no evidence supporting this hypothesis. These results suggest that centers did not actively discriminate against patients or kidneys at any stage of the transplant process, and the initial decrease in transplants was likely due to cautious behavior as centers adapted to the new policy.

To broaden my analysis, I examine the effect of CoP on kidney wastage—a key metric beyond CoP's immediate focus but emphasized in the Biden-Harris administration's "Increasing Organ Transplant Access (IOTA)" model (CMS, 2024). I find evidence that the discard rate for high-risk kidneys increased following Medicare's announcement but diminished post-implementation, suggesting a temporary adjustment period as transplant centers adapted to the policy and learned

how performance would be assessed. This finding demonstrates the importance of understanding transplant centers' responses to policy uncertainty. It underscores the critical role that clear communication and timely guidance can play in preventing short-term unintended consequences, particularly during policy transitions.

1.1 Related literature

This paper contributes to three main strands of literature. First, it engages with the economic debate on centralized quality disclosure [5]. 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 firm responses to such policies in the healthcare contexts, such as 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). In contrast, I analyze how federal oversight influences transplant center behavior, offering an alternative perspective on how policy shapes outcomes through post-transplant care. Related work, such as Bae (2024) and Dickert-Conlin, Elder and Teltser (2019), investigates external factors, including donor service area boundary redrawing and state-level policies affecting allocation and mortality. My analysis shifts the focus to center-level behavioral responses, providing insights into the direct impact of regulatory oversight.

Third, this paper contributes to the literature on the causal effects of CoP by addressing limi-

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

tations in previous studies that rely on cross-sectional variation in center flagging 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 adds to existing work by using novel follow-up data to emphasize CoP's impact on post-transplant care practices. Moreover, the 2.5-year gap between 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 mortality and post-transplant care results. Section 7 presents results on selection effects. Section 8 presents results on non-CoP targeted metrics and robustness checks. Section 9 concludes.

2 Institutional background

A patient diagnosed with end-stage renal disease (ESRD) has two options: dialysis or kidney transplant[6]. 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)[7]. This section describes how patients get on the waitlist, how the centralized

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

[7]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 to a deceased donor kidney transplant.

system allocates kidneys, what post-transplant follow-up care is, and the details of the Conditions of Participation (CoP).

2.1 Getting on the waitlist

The physician refers patients to a local transplant center when they have kidney failure[8]. 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 profile, health conditions, and factors to compute 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 deceased donor kidney allocation process. 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, where the patient lives, 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 patient's health condition and decide if the patient is available or suitable for the transplant. For example, the patient's condition might have deteriorated since the last evaluation, or the patient

[8]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).

might be unavailable due to a family emergency. The transplant center does not contact every compatible patient because of the tight deadline[9]. It usually informs the patient after UNet confirms 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 the patient and donor [10]. 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 where the center affects the type of kidney their patients match 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 above age 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. In Section 7, I leverage the patient's acceptable donor criteria and patient-kidney offer data to explore how CoP affects these two channels.

2.3 Post-transplant care and acute kidney rejection

Centers typically discharge patients within 8-14 days post-transplant. After discharge, patients will visit the center for regular check-ups at defined intervals (6 months, 1 year, 2 years, etc) to monitor 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 [11]. During rejection episodes,

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

[10] The 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 cell and destroy the kidney. Source: https://www.kidney.org/atoz/content/BloodTests-for-Transplant.

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

the patient's immune system-particularly T-cells and antibodies-attacks the foreign kidney, potentially leading to impairment and graft failure (Becker et al., 2022). To mitigate this, centers prescribe immunosuppressant medications during recovery. However, immunosuppressants compromise patients' immune systems, increasing vulnerability to viral infections. Therefore, centers must carefully manage the timing and dosage of immunosuppressant therapy to balance kidney rejection risks against infection susceptibility (Roberts and Fishman, 2020).

In addition to managing immunosuppressants, early detection of kidney rejection through follow-up sessions significantly enhances graft survival and kidney function preservation (Sharaby et al., 2023). Kidney rejection often presents asymptomatically, though patients may occasionally experience symptoms such as fever, localized pain at the transplant site, or reduced urine output. Doctors typically rely on subtle signs through biomarkers or changes in kidney function metrics, such as elevated creatinine levels, to accurately diagnose rejection. Therefore, regular follow-up sessions include targeted medical tests that enable early detection through these biomarkers.

In section 6.2, I leverage the follow-up data that track patient health status, immunosuppressant prescription, and outcomes from diagnostic tests to analyze the impact of CoP on centers' post-transplant care practices for preventing and detecting acute kidney rejection.

2.4 Conditions of Participation (CoP)

Before July 2007, OPTN was the primary organization responsible for monitoring a transplant center's number of post-transplant survival but only twice recommended to the Department of Health and Human Services to remove a transplant center's certification (GAO, 2008). 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[12]. As stated in the Final Rule establishing the increase in CMS oversight:

> " The OPTN generally takes a collegial approach and assists the center in improving their performance, while we generally take a regulatory approach which sometimes

[12]Source: https://www.latimes.com/news/la-me-newtransplant17dec17-story.html

may lead to termination ..." (Federal Register, 2007)

CMS announced CoP in February 2005 and implemented it in July 2007. The policy provides a foundation for improving quality and protecting the health and safety of transplant centers (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) on the first week of every January and July. Figure C.1 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) contains transplants from January 1, 2008, and June 31, 2010 (red line). CMS flags a transplant center for poor performance if all of the following criteria are satisfied:

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 uses extensive patient, donor, and match characteristics, including, but not limited to, age, race, diabetic status, donor cause of death, human leukocyte antigens (HLA) matching, etc. 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). Criteria one states that the center's observed deaths have to exceed expected deaths by 50%. Criteria two states that the difference between observed and expected deaths has to be greater than 3. Finally, criteria three states that if observed deaths are different from expected deaths, the difference has to be statistically significant at 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' 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)[13].

Once CMS flags a center for poor performance, it implements a data-driven quality assessment and performance improvement (QAPI) system. If CMS flags 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.2.

3 Conceptual framework

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 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 regulatory penalties associated with high patient mortality. 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 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 I illustrates the center's timeline and decision-making[14].

[13] I account for transplant volume and unadjusted mortality in Section 5.1 when constructing center flagging beliefs.

[14] 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 bad kidney. Both models have similar results on transplant threshold and post-transplant care.

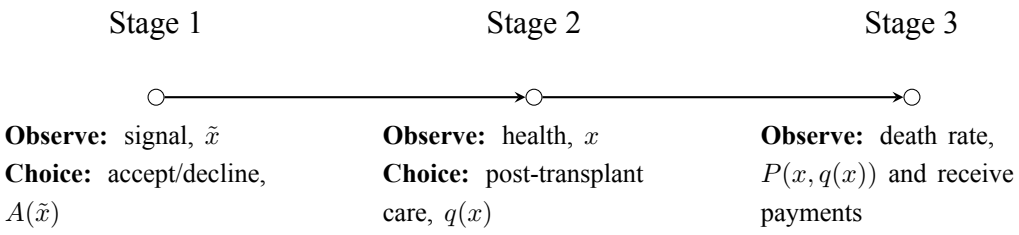

Figure I: Timeline of the center behavior

3.1 Setup

Patient health is denoted as x, where $x \sim N(\mu_x, \sigma_x^2)$ [15]. 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 $P(x, q(x)) = 1 - \Phi\left(\frac{q(x)+x}{\sigma}\right)$, 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). Medicare 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).

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

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 patient's time (Senanayake et al., 2020). The patient receives zero if centers do 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[16]:

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

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

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

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

$A^*(\tilde{x}), q^*(x)$ and present the proofs in Appendix A.

Proposition 1. *The optimal $q^*(x)$ is an implicit solution to the equation A.2. $A^*(\tilde{x})$ takes the form of a cutoff strategy as defined in A.3, 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.

The monotonic relationship between $\mathbb{E}[x|\tilde{x} > t^*]$ and t^* offers a way to evaluate the informativeness of unobserved $\tilde{x}$. If $\mathbb{E}[x|\tilde{x} > t^*]$ does not increase with t, it suggests that $\tilde{x}$ is dominated by noise (i.e., high Var(u)) and contains little information about x. Consequently, selection based

on unobserved $\tilde{x}$ weakly correlates with x, reducing concerns about selection on unobservables threatening identification.

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 includes detailed information on patient and donor characteristics and survival outcomes. Crucially, patients who receive a transplant are also included in follow-up data that tracks their health over time and records medical tests performed during revisits. The PTR dataset includes all kidney offers made by the system and records accept/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 December 31st, 2012, which approximately spans 4 years before and 8 years after the CoP announcement in February 2005. From this set, I exclude patients who needed multiple organ transplants, those who received a living donor kidney, 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 7 uses patient-kidney offers to analyze transplant center accept-decline decisions. Section 8.1 uses kidney-level information to analyze kidney utilization.

4.2 Descriptive analysis

Figure II presents a time-series plot of the post-transplant 1-year mortality rate from 2001 to 2012, showing a steady decline from approximately 12 percent in 2001 to 6 percent in 2012. 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, anything before February 2005 is the pre-CoP period, February 2005 and July 2007 is the post-CoP announcement period, and anything after July 2007 is the post-CoP implementation period. This timeline provides a natural framework for evaluating the impact of CoP on transplant outcomes.

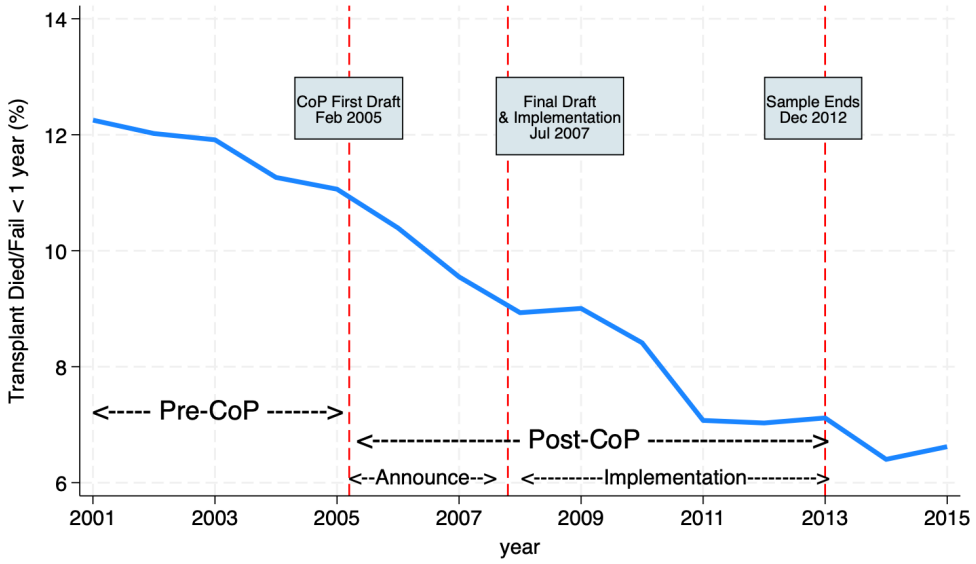

Figure II: Post-transplant mortality decreasing from 2001-2012 (main analysis period)

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 pre-CoP as the period from January 2001 to February 2005, post-CoP announcement as February 2005 to July 2007, and post-CoP implementation as July 2007 to December 2012.

Table I presents summary statistics for the sample, with each row representing different follow-up intervals, while panels group key variables. Between 2001 and 2012, 124,600 patients received a deceased donor kidney transplant, with a 29-26-45 percent split across the three periods. Panel A shows that most transplanted patients attend follow-up appointments, demonstrating high compli-

ance rates. However, attendance declines over time due to post-transplant deaths, which account for 94 percent of non-compliance cases (Table D.1). Panel B indicates that kidney rejection rates at various follow-up intervals have remained stable across all three CoP periods. Panels C and D highlight a post-CoP increase in the prescription of maintenance immunosuppressive drugs and acute kidney rejection testing at different follow-up intervals. However, 6-month testing fell below pre-CoP levels after Medicare implemented CoP. Overall, the patterns in Table I underscore high patient compliance and provide preliminary evidence of evolving center practices to improve the prevention and detection of acute kidney rejection. These trends are further examined in Section 6.2.

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%	98.6%	98.8%
6 month	91.6%	92.8%	94.2%
1 year	87.8%	89.3%	91.5%
Panel B: kidney rejection rates			
2 weeks	3.0%	4.2%	2.2%
6 months	6.6%	6.3%	6.4%
1 year	3.9%	3.5%	3.6%
Panel C: prescription rates (prevent)			
2 weeks	82.4%	82.7%	87.5%
6 months	28.7%	83.9%	77.4%
1 year	38.5%	87.7%	90.4%
Panel D: CMV testing rates (detect)			
2 weeks	92.2%	95.3%	98.9%
6 months	10.8%	18.5%	2.8%
1 years	6.8%	28.0%	38.5%
Number of Observations	36446	32575	55583

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. 94% non-compliers are due to deaths. Panels B, C, and D are conditional on patients attending follow-up appointments. Panel B shows the kidney rejection rates during follow-up. Panel C shows the likelihood of maintenance drug prescription during follow-up. Panel D shows CMV testing rates during follow-up.

Tables D.2 and D.3 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.3 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 total transplants have decreased, there is no clear indication that centers are selecting against specific transplant profiles. These patterns are further examined in Section 7.

5 Research design

The announcement of CoP in February 2005 created both cross-sectional variations in the marginal penalty incentives across centers and within-center temporal variation. This setting lends itself naturally to a differences-in-differences (DiD) research design to quantify the causal effect of the CoP announcement[17]. However, there are three empirical challenges to identifying these effects that the proposed design addresses.

First, CoP flags centers based on past mortality performance and suitable statistical adjustments. Since flagged and non-flagged centers differ in observable characteristics, relying solely on cross-sectional comparisons could introduce bias. To address this, I rely on within-center estimates to control for any time-invariant factors affecting center behavior.

Second, treatment status is not clearly defined because forward-looking center administrators strategize based on their expectations of exceeding CoP limits rather than waiting for the flagging status to be revealed. Simply comparing flagged and non-flagged centers may underestimate the center's response. Instead, I follow Gupta (2021) and model center behavior based on their ex-

[17] In Figure II, I show the post-transplant 1-year mortality from January 2001 to July 2007 and illustrate the pre-CoP and post-CoP in my subsequent analysis.

pectations of exceeding CoP limits, 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:

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

Here, Y_{ickt} represents the outcome of interest, such as patient mortality, the primary performance metric for this discussion. α_c controls for time-invariant center characteristics, while δ_t accounts for common shocks affecting all centers within a six-month window. The key term captures the center's expectation of exceeding the unadjusted mortality cutoff based on its information set at the start of the six-month window. This forward-looking approach differs from prior studies, which focus on post-implementation behavior, and allows me to capture potential anticipatory responses during the 2.5 years between CoP's announcement and implementation[18]. The variable ε_{ickt} captures omitted factors affecting mortality, while X_{ik} accounts for patient and kidney risk factors. The parameter β_s measures the average change in outcomes after the CoP announcement or implementation when beliefs increased by 10 percentage points or one standard deviation.

5.1 Measure of center expectation

Center beliefs provide the key identifying variation across centers but remain unobserved. I make two simplifying assumptions to construct an empirical analog. I assume the center bases its expectation on knowledge of past mortality performance and transplant volume. I also assume that centers have rational expectations conditional on past performance.

I follow Gupta (2021) and non-parametrically predict an empirical analog of each center's expectation of being penalized in the future using a kernel regression of actual penalty status on

[18]The potential penalties for non-compliance, such as system reviews, temporary shutdowns, and CMS decertification, provide strong incentives for the center to adjust its behavior in anticipation of CoP's implementation.

the relevant unadjusted post-transplant mortality and transplant volume, as shown in equation 3.

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

Conceptually, this expectation predicts the probability of a penalty for a center based on the experience of neighboring centers falling within the kernel bandwidth. I denote the predicted value from the kernel regression as $\rho_{c,t+2}$. One problem is that the flagging status released in July 2007 is not exogenous; it consists of transplants that coincide with the post-CoP announcement. To estimate the center's belief, I circumvented this issue using the flagging status in January 2005, July 2005, and January 2006. Their 2.5-year rolling cohort does not overlap with the CoP announcement. I illustrate this in Figure C.3. Hence, I hold the probability of flagging calculated using these penalty statuses and denoted as ρ_c.

In Figure III, the average (median) transplant center has a 10 (7) percent probability of being flagged in the future. Using a similar approach to Gupta (2021), I estimate significantly lower penalty expectations than those observed in the Hospital Readmissions Reduction Program (HRRP), where approximately 50 percent of hospitals receive penalties. This discrepancy arises because Medicare penalizes hospitals in HRRP if their risk-adjusted 30-day readmission rates exceed the national average, creating a higher baseline penalty likelihood. In contrast, under CoP, centers must meet all three conditions outlined in Section 2.4 to be penalized, significantly lowering the expectation of penalties. Figure C.4 plots the density of beliefs, showing how penalty expectations in the CoP setting differ from those in HRRP. Additionally, conditions 2 and 3 of CoP are specifically designed to protect low-volume centers more likely to exceed condition 1 (i.e., an observed-to-expected death ratio exceeding 1.5), further reducing the penalty likelihood.

Although the likelihood of penalties is relatively low, the expected loss for flagged centers is substantial. As discussed in Section 2.4, flagged centers must undergo a system review, and temporary shutdowns pose a significant opportunity cost to their profit margins. Moreover, centers flagged twice within 30 months face the threat of CMS decertification, which could effectively

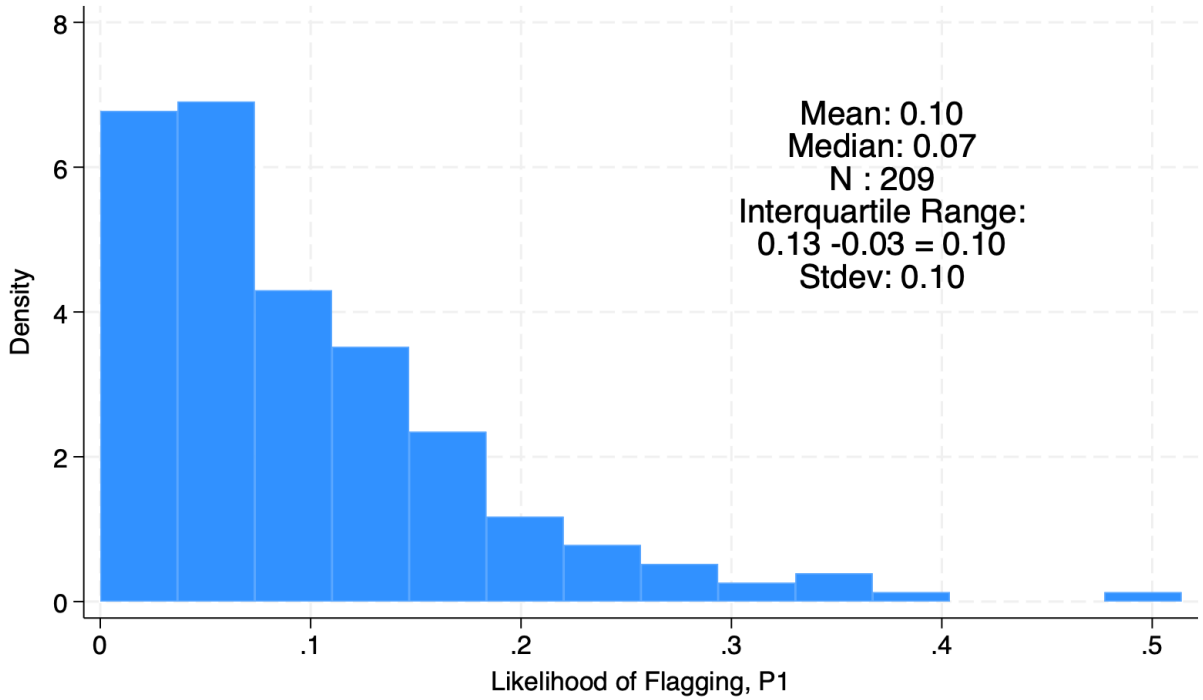

Figure III: Density of center likelihood of future flagging

Note: This figure illustrates the density of the constructed continuously varying measure of a forward-looking center's expectation of being flagged in the future. The expectation, P_c, is predicted by the unadjusted mortality rate and transplant volume from 2005-2006.

shut down their transplant operations. These sizeable potential losses provide strong incentives for centers to adjust their behavior, focusing on improving post-transplant outcomes and avoiding conditions that might trigger penalties.

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 flagging rate was first determined. They adjust their behavior, knowing that their performance will revert to their true, lower-quality self in the future. Hence, an OLS regression on center beliefs will make it seem that the penalty did not motivate

centers to improve. I overcame this concern by using an instrumental variables approach, relying on variation in center quality in 2002-2004, before the CoP announcement, to generate exogenous variation in penalty probability under CoP. This approach assumes that true hospital quality is stable over time and uses historical features to predict flagging probability, eliminating the role of temporary swings. Furthermore, the instrument is exogenous because it is estimated with pre-CoP data and, hence, not influenced by contemporaneous policy changes.

This type of dynamic model has been extensively analyzed (Anderson and Hsiao, 1981; Amemiya and MaCurdy, 1986; Arellano and Bond, 1991) and one solution to obtain a consistent estimate of β is using baseline or "predetermined" characteristics of center c as instruments for ρ_c (Arellano and Bover, 1995; Acemoglu and Finkelstein, 2008; Gupta, 2021). Accordingly, I use a center-level instrument Z_c predicted using baseline covariates. 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 the expectation term with the estimate obtained using equation 3. Equation 4 also contains the first-stage equation:

$$\rho_c \times \mathbb{1}(t=s) = \pi_c + \pi_t + \lambda Z_c \times \mathbb{1}(t=s) + u_{ickt} \quad ; \quad s \in \{ann, imp\} \tag{4}$$

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

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 is an expected mortality rate using data on patient risk factors from 2002 - 2004. This is the earliest year for which I have data available. The predicted value is, therefore, purged of unobserved factors and transient noise. The identifying assumption is that centers with low versus high values of expected mortality rates held constant as in 2005 would evolve along parallel trends in the absence of the CoP announcement. To explore the validity of this assumption, I plot the

coefficients β_s obtained by estimating the following dynamic nonparametric equation:

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

d_{Z_c} is an indicator set to 1 if center c is the top half of all centers, ranked by the instrument Z_c. Recall from previous discussions that the center with the highest historical mortality rate has the highest penalty risk and, therefore, the most incentive to improve.

5.3 Subsample

To identify the causal effect of the CoP policy on post-transplant mortality, I rely on the subsample of patients whose post-transplant mortality timeline do not overlap with the CoP announcement. I exclude patients whose post-transplant timeline overlaps with the CoP announcement due to potential temporal confounding (i.e., post-transplant mortality risk naturally increases as time passes). This approach improves the study's internal validity by ensuring that I make comparisons between patients whose outcomes are exclusively influenced by pre or post-CoP conditions. Figure C.5 illustrates the two kinds of patients discussed above.

The primary threat to the identification strategy is the possibility that the composition of patients across centers changed in significant and potentially unobservable ways. While Table D.3 does not show clear evidence of such changes, this does not entirely rule out the concern. To address this, Section 7 provides additional analysis demonstrating that centers do not appear to systematically discriminate against specific profiles of patients or kidneys at the transplant or admission stages. This combined evidence mitigates concerns about potential selection bias undermining the identification strategy.

6 Effects on post-transplant mortality and care

This section quantifies the effects of the CoP announcement on post-transplant 1-year mortality, the program's targeted metric, to establish its top-line impact. Using follow-up data, I then analyze how CoP influenced post-transplant care, particularly in managing acute kidney rejection.

6.1 Targeted metric

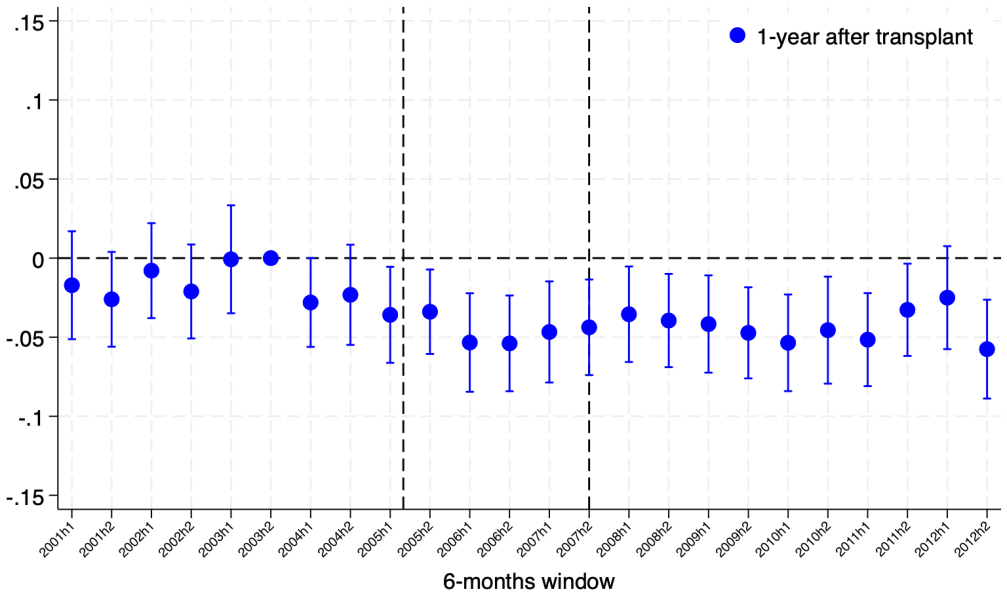

Figure IV: Impact on post-transplant 1-year mortality

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 use robust standard errors. Error bars indicate 95 percent confidence intervals.

Figure IV plots the coefficients β_s of equation 6 for 6-month windows between 2001 and 2012, 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 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, there was a statistically significant and economically meaningful decline in mortality for centers with higher penalty risks.

The pattern persisted even when Medicare 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 2.77 percentage point reduction in post-transplant 1-year mortality for a 1-standard deviation increase in a center's belief after Medicare 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 percent of the 10,370 kidney transplant recipients died within a year. A 2.77 percentage point decline implies that 853 patients died post-transplant, compared to 1,147 previously—a 25 percent decrease. The improvements persisted even after Medicare implemented CoP in July 2007, though at a smaller magnitude (2.21 percentage points).

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

	(1) OLS	(2) IV
Post-Announce	-0.01470***	-0.02777***
	(0.00423)	(0.00582)
Post-Implement	-0.01384***	-0.02212***
	(0.00375)	(0.00524)
Y mean	0.11036	0.11036
F-statistic		14713.68470
Fixed Effects	Center, 6-months	Center, 6-months
Observations	105264	105264

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, 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 use robust standard errors.*$p < 0.1$, **$p < 0.05$, ***$p < 0.01$.

Further analysis in Figure C.6 and Table D.4, using granular time intervals, shows that mortality improvements are most pronounced within the first two weeks and six months of receiving a kidney transplant. This suggests that transplant centers concentrated their mitigation efforts on these critical periods, such as closer monitoring and adjusted immunosuppressant timing. 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 post-transplant care to achieve these gains.

6.2 Post-transplant care

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 marginal benefit to the patient and center. CoP incentivizes centers to increase the optimal post-transplant care to the extent that it decreases the patient's probability of post-transplant deaths 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., $\frac{\partial q^*(x)}{\partial \tau} > 0$).

I use novel follow-up data that track patient health status and outcomes of medical tests performed during revisits to examine how CoP affects post-transplant care. I focus on how centers prevent and detect acute kidney rejection, as mentioned in Section 2.3.

6.2.1 Prevention

The immune system naturally fights against foreign objects in the body, which challenges kidney transplant patients as the immune system often recognizes the new kidney as foreign and attempts to reject it. Immunosuppressants are necessary to prevent acute kidney rejection by suppressing the patient's immune system and reducing the likelihood of an immune attack. Three kinds of immunosuppressants are used at different stages of the transplant process: (i) *induction medicine,* a potent anti-rejection medicine administered intravenously around the time of transplant to prepare the patient's immune system for the new kidney; (ii) *maintenance medicine,* which are taken continuously to manage immune activity and prolong graft viability; (iii) *rejection medicines,* which are used to treat episodes of rejection when they occur[19]. This section focuses on induction and maintenance medicines.

[19]Source: UNOS, types of immunosuppresants

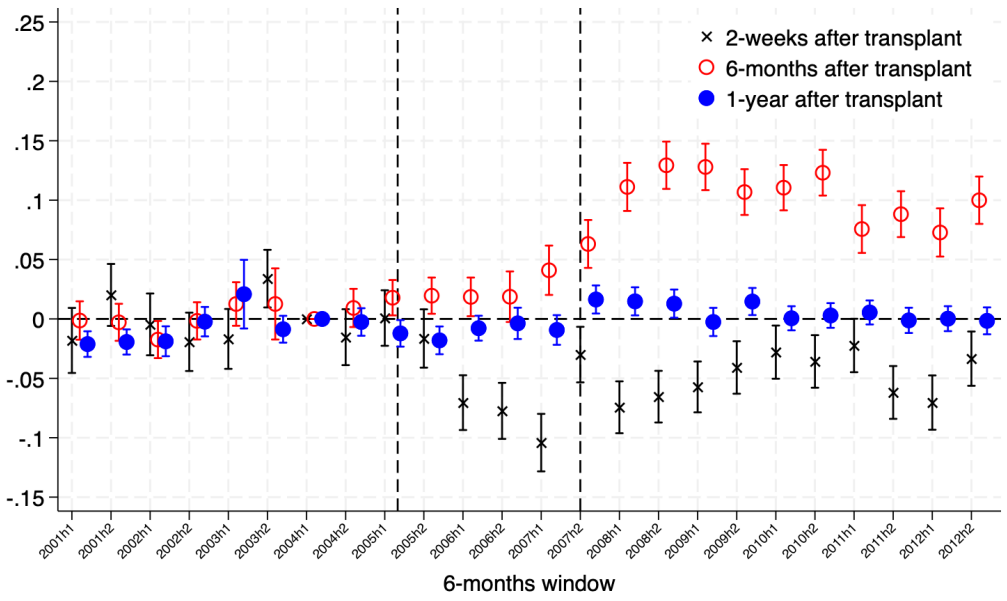

Figure V: Impact on maintenance immunosuppressant prescription at different follow-up periods

Note: The figure presents the estimated effects on the probability of prescribing maintenance immunosuppressant at different follow-up periods, 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 use robust standard errors. Error bars indicate 95 percent confidence intervals.

Figure V presents an event study analysis of maintenance immunosuppressant prescription rates at different follow-up periods (2-week, 6-month, and 1-year) surrounding the CoP policy. The plot suggests that centers adjusted immunosuppressant prescription practices in response to the policy, with notable differences across follow-up periods.

Following the CoP announcement, centers reduced immunosuppressant prescriptions at the 2-week follow-up, a pattern that persisted after implementation. In columns 1 and 2 of Table D.5, centers reduce the induction medicine dosage (5 percent) and the maintenance medicine prescription (2.5 percent) after Medicare announced CoP [20]. One possible explanation is that centers sought to delay immunosuppressant initiation to allow patients to recover post-transplant before introducing immunosuppressant drugs. Early immunosuppressant use presents a tradeoff - it suppresses the immune system and prevents rejection but increases susceptibility to complications such as viral infections (Pilch, Bowman and Taber, 2020). This interpretation aligns with Figure C.7 showing

[20] In the follow-up data, I only observe the number of days patients were given induction medicine leading up to the kidney transplant and use that to proxy my dosage (i.e. longer days is equivalent to higher dosage).

a decline in reported viral infection 3 or 6 months after the initial kidney transplant after Medicare announced and implemented CoP[21].

In contrast, immunosuppressant prescriptions increased at the 6-month follow-up after the CoP announcement and rose further after implementation. This suggests that centers modified their treatment strategies over time, possibly fine-tuning dosages, adjusting immunosuppressant combinations, or tailoring prescriptions based on evolving patient responses. Notably, the range of immunosuppressants remained relatively stable during the analysis period (2001–2012), with no significant breakthroughs in drug efficacy (Cooper, 2020). This stability suggests that the observed prescription changes reflect clinicians optimizing treatment timing and dosages rather than responding to new pharmaceutical developments. Finally, no significant changes were observed in 1-year immunosuppressant prescriptions, indicating that centers primarily focused on short- and medium-term prescription adjustments. The stability in long-term prescription practices suggests that regulatory oversight influenced early post-transplant management rather than long-term treatment protocols.

These findings highlight how transplant centers adapted their post-transplant immunosuppressant strategies in response to regulatory oversight. The decline in early-phase prescriptions reflects a cautious approach to balancing rejection risks with infection prevention, while the increase at 6 months suggests that clinicians refined their protocols over time. The stability at 1-year reinforces the idea that regulatory adjustments primarily shaped immediate post-transplant decision-making. These results suggest that phased policy implementation allowed transplant centers to adapt gradually, experiment with different approaches, and refine patient care protocols in response to evolving clinical and regulatory landscapes.

6.2.2 Detection

Acute kidney rejection is often asymptomatic, making vigilant monitoring essential. Centers typically detect subtle signs through biomarkers or changes in kidney function metrics. Conse-

[21] Due to the delayed nature of taking immunosuppressant drugs, I examine the 3 and 6 months after transplant infection rate, instead of the 2 weeks after transplant infection rate.

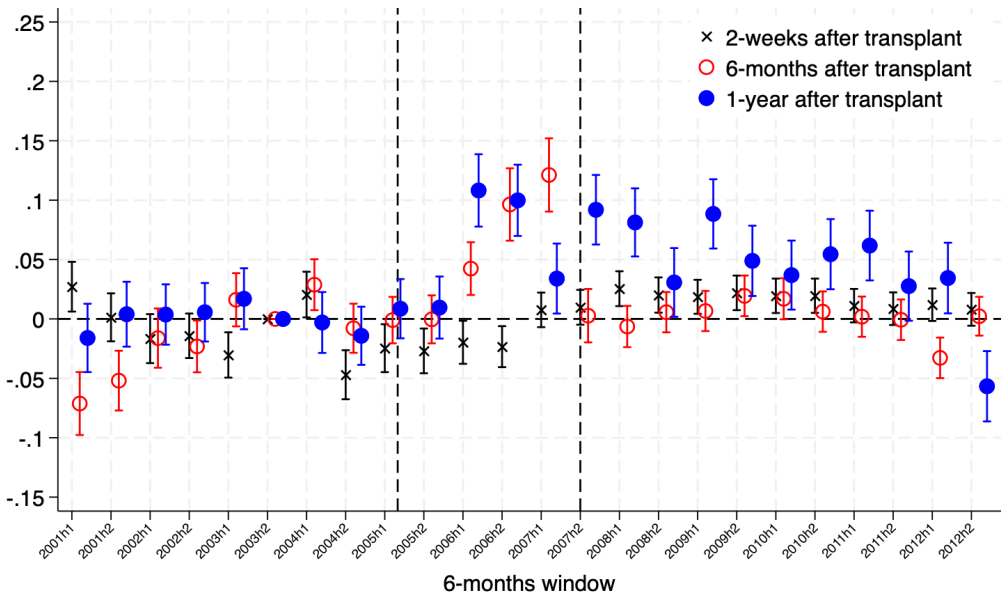

Figure VI: Impact on CMV serology test at different follow-up periods

Note: The figure presents the estimated effects on the probability of performing a CMV serology test at different follow-up periods, 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 use robust standard errors. Error bars indicate 95 percent confidence intervals.

quently, testing for kidney rejection during follow-up visits is critical for preserving graft function (Sharaby et al., 2023). The cytomegalovirus (CMV) test detects the presence of antibodies against CMV, a virus that can reactivate in immunosuppressed kidney transplant recipients, potentially triggering immune activation leading to rejection. Regular CMV testing enables early identification of viral infections, allowing for timely antiviral treatment to prevent immune-mediated damage (Hasanzamani et al., 2016). The CMV test is not a direct diagnostic tool for acute kidney rejection. Still, it plays a vital role in detecting complications that can signal rejection[22].

Figure VI presents an event study analysis of CMV testing rates at different follow-up intervals (2-week, 6-month, and 1-year) surrounding the CoP policy announcement and implementation. The results reveal distinct patterns in testing practices across the three follow-up periods. First, transplant centers did not significantly alter CMV testing protocols at the 2-week follow-up. This

[22] A kidney biopsy remains the gold standard for diagnosing rejection because it provides direct evidence of immune-mediated injury that CMV testing cannot detect. Unfortunately, the follow-up data does not document whether the center performs a kidney biopsy during follow-up.

consistency is expected, as early CMV testing is a standard post-transplant procedure designed to ensure patient stability and detect immediate complications. Since this practice was already well-established, centers had little reason to modify it in response to the CoP announcement or implementation.

In contrast, CMV testing rates increased at the 6-month and 1-year follow-ups immediately after the CoP announcement. This suggests that transplant centers initially responded to the threat of regulatory scrutiny by expanding testing to identify potential kidney rejections. However, following the implementation of CoP, testing at the 6-month follow-up dropped sharply, whereas 1-year testing remained elevated. This divergence indicates a period of fine-tuning in response to the policy. Initially, centers may have increased CMV testing across multiple time points as a precautionary measure. However, as they gained experience, they recognized that CMV testing was an ineffective standalone diagnostic tool for kidney rejection, reducing 6-month testing. This interpretation is supported by Figure C.8 showing that rejection rates at both the 6-month and 1-year follow-ups remained stable, reinforcing that increased CMV testing was uninformative. Since CMV testing is a supplementary rather than a definitive diagnostic tool—unlike the gold-standard kidney biopsy—its limited effectiveness likely prompted centers to reassess its utility. Despite this, transplant centers continued CMV testing at the 1-year follow-up after implementing CoP. One possible explanation is that centers used this testing as a final precautionary check to ensure patient stability before submitting data to Medicare for assessment.

These findings highlight the dynamic nature of transplant center responses to regulatory oversight. While the initial reaction to CoP was to increase testing, subsequent adjustments suggest a more nuanced approach, balancing compliance with clinical efficacy. The persistence of 1-year CMV testing underscores the role of institutional adaptation, where centers selectively retained practices that aligned with regulatory and administrative considerations.

7 Selection mechanisms

Quantifying the role of distortions (if any) in producing the decline in post-transplant 1-year mortality reported above is vital. This section considers three potential selection mechanisms: donor filtering, selection into transplant, and strategic admission.

7.1 Donor filtering

In Section 2.1, centers register patients onto the national waitlist and upload relevant medical and biological information. Simultaneously, centers can proactively filter potential donors for patients in the UNet system (King et al., 2022; Yu et al., 2024). For instance, a center might impose a maximum donor age, thereby preventing offers from donors who exceed this criterion, regardless of biological compatibility. Using patients' waitlist records, I document all modifications to donor criteria made during the patients' waitlist periods to analyze whether these adjustments reflect strategic donor filtering. The hypothesis is that CoP could incentivize centers to avoid risky kidney profiles by imposing stricter donor criteria.

To identify this effect, I leverage patients whose waitlist tenure overlaps with the CoP announcement and subsequent implementation. Using a patient fixed-effects regression, I isolate the impact of CoP on donor filtering by examining within-patient changes in donor criteria[23]. The following equation represents the donor filtering model:

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

Here, Y_{ict} represents different donor criteria, such as age, BMI, human leukocyte mismatches, etc. α_i controls for time-invariant patient characteristics, while δ_t accounts for common shocks affecting all patients within a quarter. Tables D.8 and D.9 present estimates for various modifi-

[23]To minimize potential temporal confounds-specifically, that centers might naturally become less restrictive as patients' waiting time increases-I limit the analysis to patients listed from 2004 to 2012, who had waited fewer than six months and subsequently remained on the waitlist for at least three additional years.

able donor characteristics[24]. The analysis reveals no consistent evidence that centers tighten donor criteria in response to CoP; instead, centers appeared to loosen specific criteria (e.g., accepting higher creatinine levels and longer ischemic times) to broaden their available kidney pool. Nevertheless, the absence of proactive donor filtering does not preclude selection against undesirable kidneys; centers might prefer evaluating all potential kidney offers before deciding on behalf of their patients. I investigate this potential selective behavior further in the following subsection.

7.2 Selection into transplant

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 VII 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 figure shows 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 flattened after Medicare implemented CoP, highlighting a potential stabilization in acceptance behavior. On average, Table III indicates a 1.42 percentage point decline in acceptance probability, a 7 percent decrease given the mean acceptance rate of 20 percentage points after Medicare announced CoP.

To examine whether centers avoided certain patient or kidney profiles to reduce mortality risk, I estimated triple-difference models that interact center penalty risk with predetermined patient and kidney risk measures.[25] These models test whether acceptance probabilities changed more for high-risk profiles than low-risk ones. Columns 3–5 in Table III show that the triple-difference co-

[24] Some criteria (e.g., diabetes, obesity, outside of donor service area and death by cardiac arrest) were omitted from the tables because they were never modified and were often set to the upper bound, further emphasizing the lack of desire to omit these potentially undesirable kidneys.

[25] 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 a high risk if $KDPI \geq 0.5$, and a patient is a high risk if $EPTS \geq 0.5$. A patient-kidney pair is considered high-risk if the patient and kidney are high-risk.

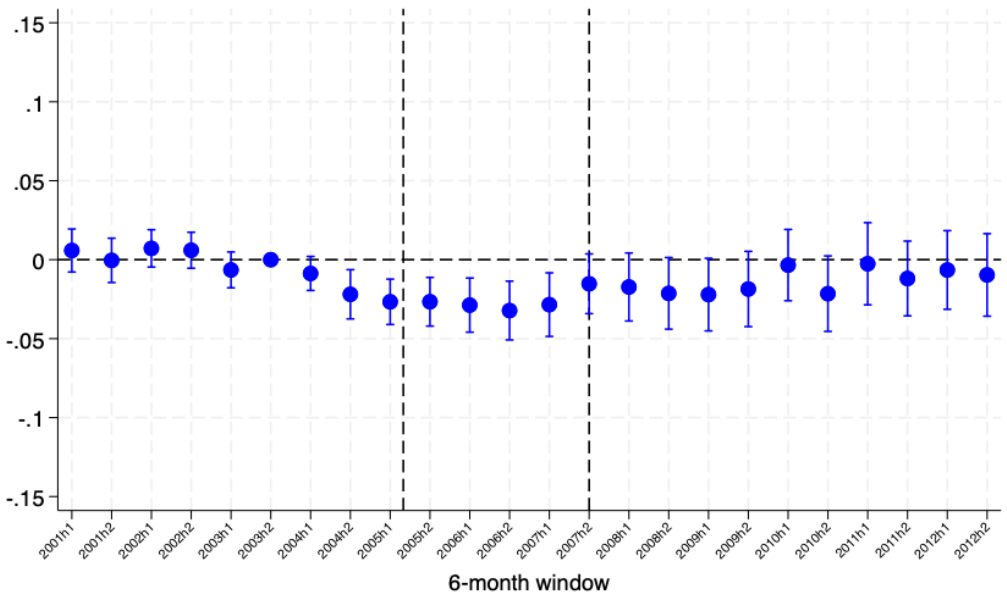

Figure VII: Impact on patient-kidney offer acceptance

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 use robust standard errors. Error bars indicate 95 percent confidence intervals.

efficients are statistically insignificant, suggesting that centers did not systematically avoid riskier profiles.[26].

While the previous finding rules out selection on observable factors, it does not preclude selection on unobservables. To investigate this, I leverage a corollary of Proposition 2, which predicts that if $\tilde{x}$ is correlated with x, the average health of transplanted patients rises as selection on unobserved health measures ($\tilde{x}$) intensifies (i.e., $\frac{\partial \mathbb{E}[x|\tilde{x}>t]}{\partial t} > 0$). Using Equation 4 and 5, I examine two health indicators that are available to centers after the transplant in Table D.12 and find no statistically significant evidence of changes after the CoP announcement. This suggests that the unobserved signal $\tilde{x}$ is not informative about patient health, ruling out the selection of unobservables as a threat to identification.

These findings indicate that while centers reduced the proportion of accepted patient-kidney pairs, this behavior does not align with observed or unobserved risk factors associated with mor-

[26]In the Appendix, I run similar triple-difference regressions interacting with various patient and kidney characteristics and find no statistically significant results in Tables D.10 and D.11.

tality. Thus, these changes in transplant decisions are unlikely to explain the improvements in mortality discussed in Section 6. Instead, the evidence points to enhanced post-transplant care as the primary driver of these outcomes.

Table III: Impact on selection into transplant

	Accept Decision (DiD)		Accept Decision across subgroups (Triple DiD))		
	(1)	(2)	(3)	(4)	(5)
	OLS	IV	Risky Pat.	Risky Kid.	Risky Pat.x Kid.
Post-Ann	-0.00983***	-0.01424***	-0.01125**	-0.01440*	-0.01170**
	(0.00105)	(0.00160)	(0.00412)	(0.00660)	(0.00436)
Post-Imp	-0.00953***	-0.00525***	-0.01152*	-0.01033	-0.01054
	(0.00099)	(0.00159)	(0.00544)	(0.00782)	(0.00580)
Post-Ann (Tri)			0.00509	0.00867	0.00795
			(0.00536)	(0.00553)	(0.00497)
Post-Imp (Tri)			0.00429	0.00256	0.00199
			(0.00437)	(0.00597)	(0.00482)
Y mean	0.19991	0.19991	0.19991	0.19991	0.19991
F-statistic		85350.65540			
Fixed Effects	Center, 6-months	Center, 6-months	Center, 6-months	Center, 6-months	Center, 6-months
Observations	874517	874517	874517	874517	874517

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). Columns 3, 4, and 5 are estimates from triple differences regression interacted with patient, kidney, and patient-kidney risk profiles. I use robust standard errors. *$p < 0.1$, **$p < 0.05$, *** $p < 0.01$

7.3 Strategic admission

Another way centers could influence post-transplant outcomes is by altering the patient population they admit. For example, White et al. (2014) suggests that centers may adopt more conservative admission criteria based on the belief that socioeconomic factors affect patients' ability to comply with post-transplant care instructions[27]. I examine various socio-economic and health indicators among admitted patients to test this hypothesis. As shown in Tables D.13 and D.14, the analysis reveals no significant changes in these characteristics after the CoP announcement, suggesting that centers did not modify their admission strategies significantly based on socioeconomic and health

[27] White et al. (2014) uses within-center temporal variation to analyze flagged centers' patient admission strategies.

factors.

CoP penalizes centers for post-transplant mortality but not waitlist mortality. Consequently, admitting a patient may represent a low-commitment action for centers, as it does not obligate them to proceed with a transplant or directly affect their performance metrics under CoP. This lack of immediate accountability might explain why admission practices remained unchanged despite the CoP announcement and implementation.

8 Discussion and robustness checks

This section discusses non-targeted metrics like kidney discard and tests the sensitivity of the estimates to modeling assumptions.

8.1 Kidney discard

While CoP successfully reduced post-transplant mortality by improving centers' post-transplant care practices, it might not cover all inefficiencies. This subsection examines the broader implications of CoP on kidney wastage, a metric emphasized in the recent Biden-Harris administration's "Increased Organ Transplantation Access (IOTA)" model.

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[28]. 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. Factors such as center preferences, risk aversion, and systemic inefficiencies can influence these decisions, potentially leading to avoidable waste. Analyzing kidney discard patterns is essential to identify whether CoP unintentionally 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

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

a weighted average of center penalty exposure, $Exposure_k$[29]. 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 post-CoP announcement or implementation. Table D.15 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 kidney indicator, suggests that high-risk kidneys were 3.6 percentage points (23.1 percent relative increase) more likely to be discarded than low-risk kidneys following Medicare'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 Medicare's CoP announcement, high-risk kidneys were offered to 35.8 patients (a 13.2 percent increase relative to low-risk kidneys), suggesting greater difficulty in finding an accepting center. However, post-implementation, high-risk kidneys were offered to 95.7 fewer patients (a 35.4 percent relative decrease), indicating that centers became more receptive.

These findings suggest that the initial increase in high-risk kidney discards was driven by uncertainty and unfamiliarity with the CoP policy. In the period immediately following the announcement, transplant centers may have exercised greater caution, leading to increased rejection rates. However, as centers adapted to the policy, the discard rate of high-risk kidneys converged toward that of low-risk kidneys. These results highlight a temporary adjustment period in response to

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

policy change.

8.2 Robustness checks

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.16 reproduces the coefficients for equation 5 using outcome variables: post-transplant mortality, CMV testing, and immunosuppressant prescription. The estimates are similar to the specifications where I omit the overlapping patients.

Second, I use an alternate approach to construct center-flagging beliefs. A useful benchmark is to assume that centers had perfect foresight and accurately predicted their actual flagging status in July 2007 under CoP. This is straightforward to implement by replacing the flagging probabilities with actual first-time flagging status indicators in equation 5. Table D.17 has estimates that remain largely unaffected, except for the columns on the prescription of immunosuppressants.

Third, I also change the main specification, allowing the center flagging 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.18 are considerably smaller. Still, they remain statistically significant for most cases[30].

9 Conclusion

This paper examines a large-scale federal oversight program in the U.S. deceased donor kidney transplant setting. The CoP announcement incentivized transplant centers to reduce post-transplant mortality rates, leading to a 25 percent decline in mortality since the program's announcement. Importantly, there is no evidence of donor filtering, selection into transplant, or strategic admissions. Leveraging novel follow-up data, I demonstrate how centers adapted their post-transplant strate-

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

gies to address acute kidney rejection. Centers delayed immunosuppressant initiation until 6-month follow-up. Furthermore, the gap between announcement and implementation allowed centers to reduce unnecessary diagnostic testing by 50 percent.

While the results highlight some inefficiencies in kidney utilization after the announcement, they show that CoP successfully motivated transplant centers to improve post-transplant care. Three key factors likely contributed to this success. First, the incentive structure of CoP was carefully designed, with sufficiently large penalties to prompt action while protecting vulnerable centers from disproportionate risks. Second, the 2.5-year gap between the program's announcement and implementation gave centers time to learn the policy's nuances and refine their practices to reduce post-transplant mortality. Third, frequent communication between CMS and centers during this period fostered a collaborative environment, emphasizing guidance and improvement rather than punitive measures (Abecassis et al., 2008).

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 organ allocation and post-transplant care aspects could yield valuable insights for improving system-wide outcomes.

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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:

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

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

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:

$$q^*(x) = \frac{\rho\,\alpha + (1-\rho)\,x}{(1-\rho)\,\gamma} \;+\; \frac{\lambda}{(1-\rho)\,\gamma\,\sigma}\,\phi\Big(\tfrac{x+q^*(x)}{\sigma}\Big). \tag{A.2}$$

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:

$$A^*(\tilde{x}) = \begin{cases} 1 & \text{if } \tilde{x} \geq t^* \\ 0 & \text{if } \tilde{x} < t^* \end{cases} \tag{A.3}$$

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.1 illustrates the center's timeline and decision-making.

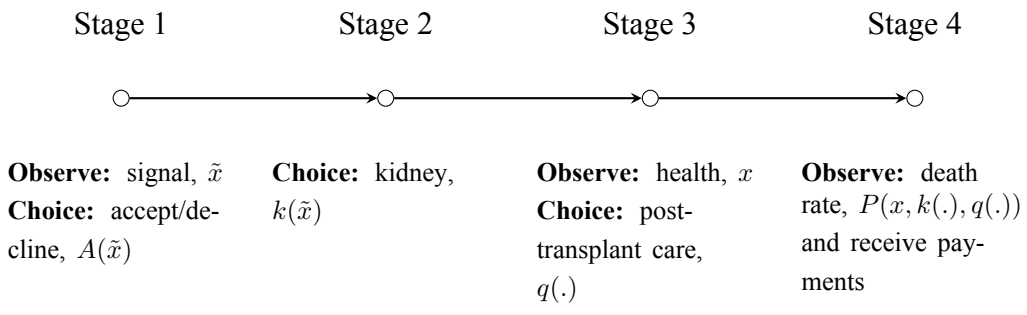


Figure B.1: Timeline of the center behavior

B.1 Setup

Patient health is denoted as x, where $x \sim N(\mu_x, \sigma_x^2)$ [31]. 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). Medicare 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

[31] 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[32]:

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

$$\text{s.t.} \quad \overbrace{\int_{\tilde{x}} A(\tilde{x})}^{\text{"small center discount"}} \quad \overbrace{\int_{x} P(x,k,q(.)) p(x|\tilde{x}) dx \, dF(\tilde{x})}^{\text{"not too many deaths"}} \leq \tau \tag{B.1}$$

τ is the CoP limit, and the rest of the terms in the constraint reflect the CoP conditions in Section 2.4. $\int_x P(x,k,q(x,k)) p(x|\tilde{x}) dx$ is equivalent to condition 1: there cannot be too many post-transplant deaths. However, even if there is, the center can still escape CMS flagging if conditions 2 and 3 are unmet. $\int_{\tilde{x}} A(\tilde{x}) dF(\tilde{x})$ mimics those conditions 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 increase 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.

Proposition B.1. *The optimal $q^*(x,k)$ is an implicit solution to the equation B.3. $A^*(\tilde{x})$ takes the*

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

form of a cutoff strategy B.6 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 B.5.

Because the center cannot observe a patient's true health x and instead relies on the noisy signal $\tilde{x}$, Proposition B.1 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

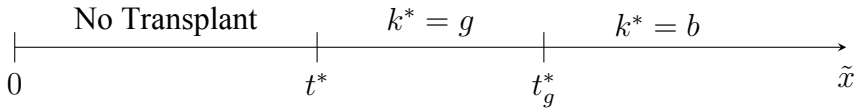
In this stylized model, the pre-CoP announcement reflects $\tau \to \infty$, meaning no effective regulatory constraints on the product of transplants and mortality, allowing centers to optimize without restrictions. The post-CoP announcement reflects $\tau < \infty$, introducing binding regulatory constraints. 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 B.2. *As CMS announces CoP (i.e., τ decreases), the transplant threshold t^* increases*

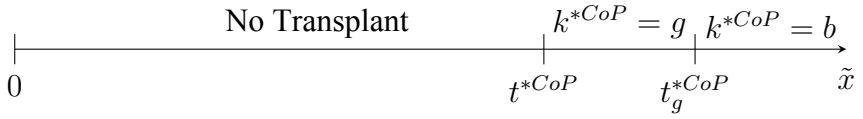
$\left(\frac{\partial t^*}{\partial \tau} < 0\right)$*; post-transplant care* $q^*(x,k)$ *increases* $\left(\frac{\partial q^*(x,k)}{\partial \tau} < 0\right)$*; the good kidney threshold* t_g^* *increases* $\left(\frac{\partial t_g^*}{\partial \tau} < 0\right)$.

Proposition B.2 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 B.2 predicts fewer bad kidney transplants after CMS implements CoP. Using Figure B.2 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.2: 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 B.1 and B.2

From equation B.1, the center's objective function is

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

$$\text{s.t.} \int_{\tilde{x}} A(\tilde{x}) \left[\int_{x} P(x,k,q) p(x|\tilde{x}) dx \right] dF(\tilde{x}) \leq \tau \tag{B.2}$$

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:

$$q^*(x,k) = \frac{\rho\,\alpha + (1-\rho)\, x}{(1-\rho)\,\gamma} \;+\; \frac{\lambda}{(1-\rho)\,\gamma\,\sigma_k}\, \phi\Big(\tfrac{x+q^*(x,k)}{\sigma_k}\Big). \tag{B.3}$$

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:

$$k(\tilde{x}) = \arg\max_{k \in \{g,b\}} \overbrace{\int \left[\Pi(x,k,q^*) \;- \lambda P(x,k,q^*)\right] p(x|\tilde{x})\, dx}^{\widetilde{\Pi}(x,k,q^*)} \tag{B.4}$$

Next, we define:

$$D(\tilde{x}) = \widetilde{\Pi}(x, g, q^*) - \widetilde{\Pi}(x, b, q^*) \tag{B.5}$$

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:

$$A(\tilde{x}) = \begin{cases} 1 & \text{if } \tilde{x} \geq t^* \\ 0 & \text{if } \tilde{x} < t^* \end{cases} \tag{B.6}$$

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

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 B.2.

C Supplementary Figures

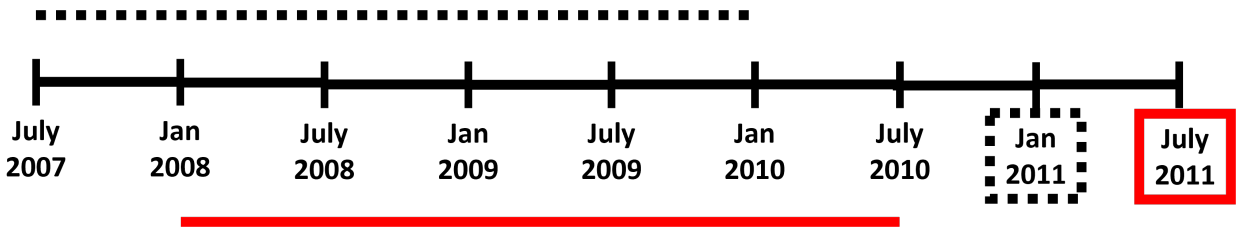


Figure C.1: An illustration of the rolling 2.5-year cohort for CoP

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.2: 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 flagged 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., $Pr(O = E) = 0.469 > 0.05$).

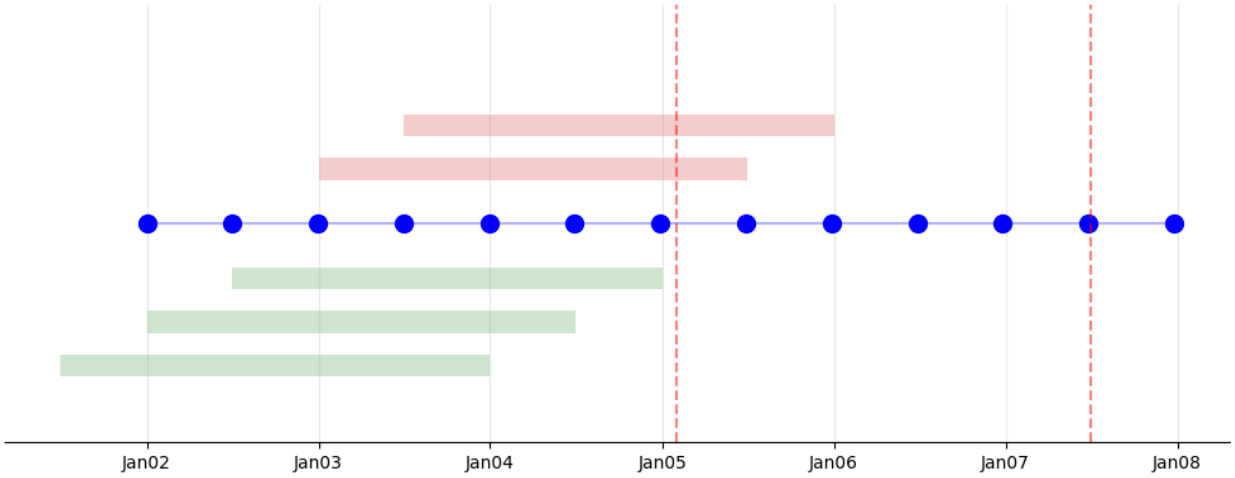


Figure C.3: CoP reports and their 2.5-years rolling cohorts

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

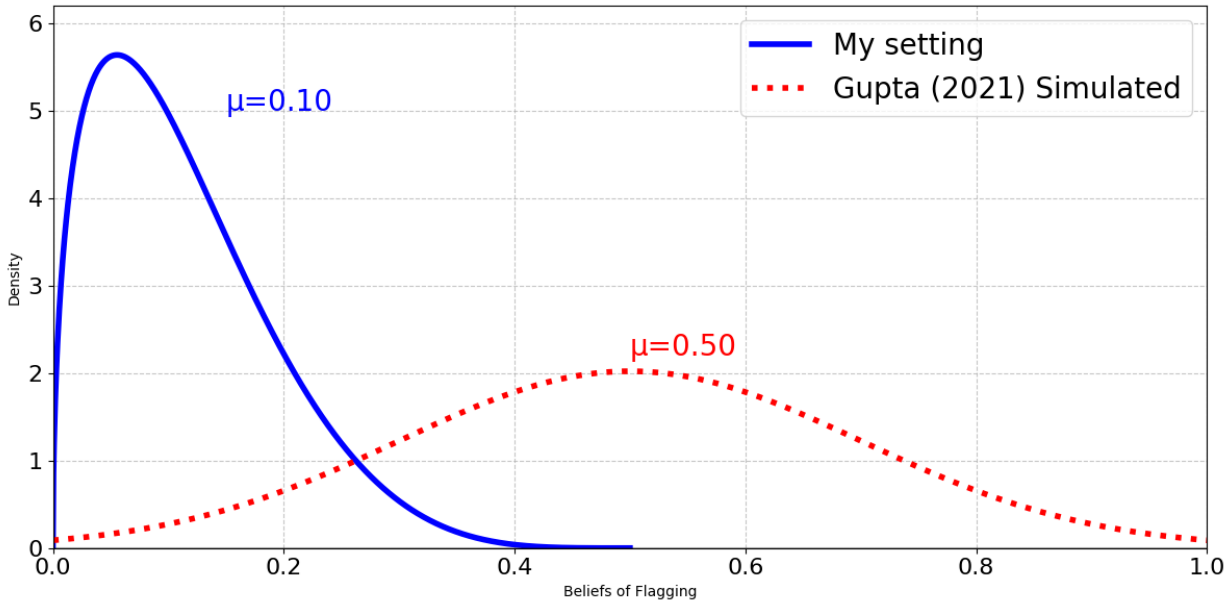


Figure C.4: Density of actual flagging beliefs in my setting versus simulated beliefs in Gupta (2021)

Note: The blue solid lines are the density of my estimated flagging beliefs ρ_c. The density is similar to a beta distribution with parameter $(\alpha = 1.5, \beta = 5)$ 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 $[0, 1]$

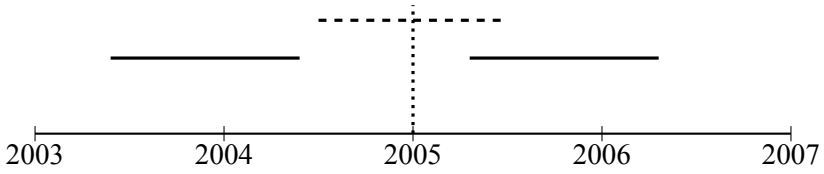
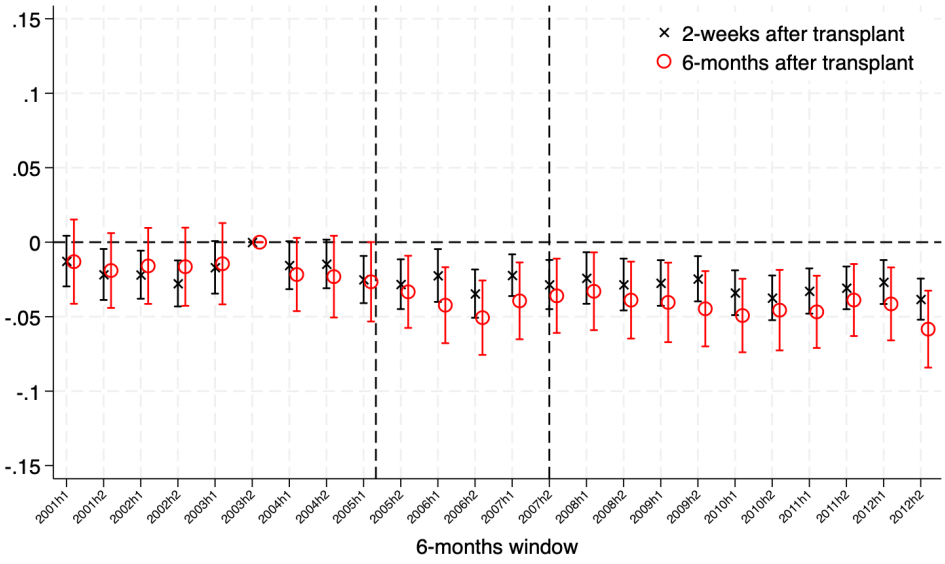


Figure C.5: Non-overlapping (solid) and overlapping (dash) patients

Note: This figure highlights my regression subsample in Sections 6 and 7. 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 do not overlap with CoP announcements have solid lines, and those with dashed lines are excluded from my analysis.

Panel A



Panel B

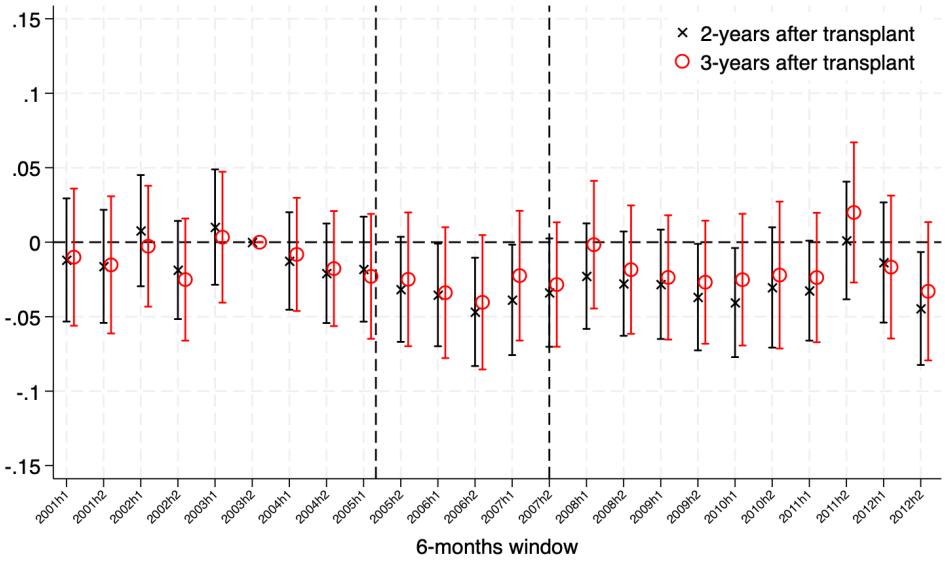


Figure C.6: Impact on post-transplant mortality at different periods

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 use robust standard errors. Error bars indicate 95 percent confidence intervals.

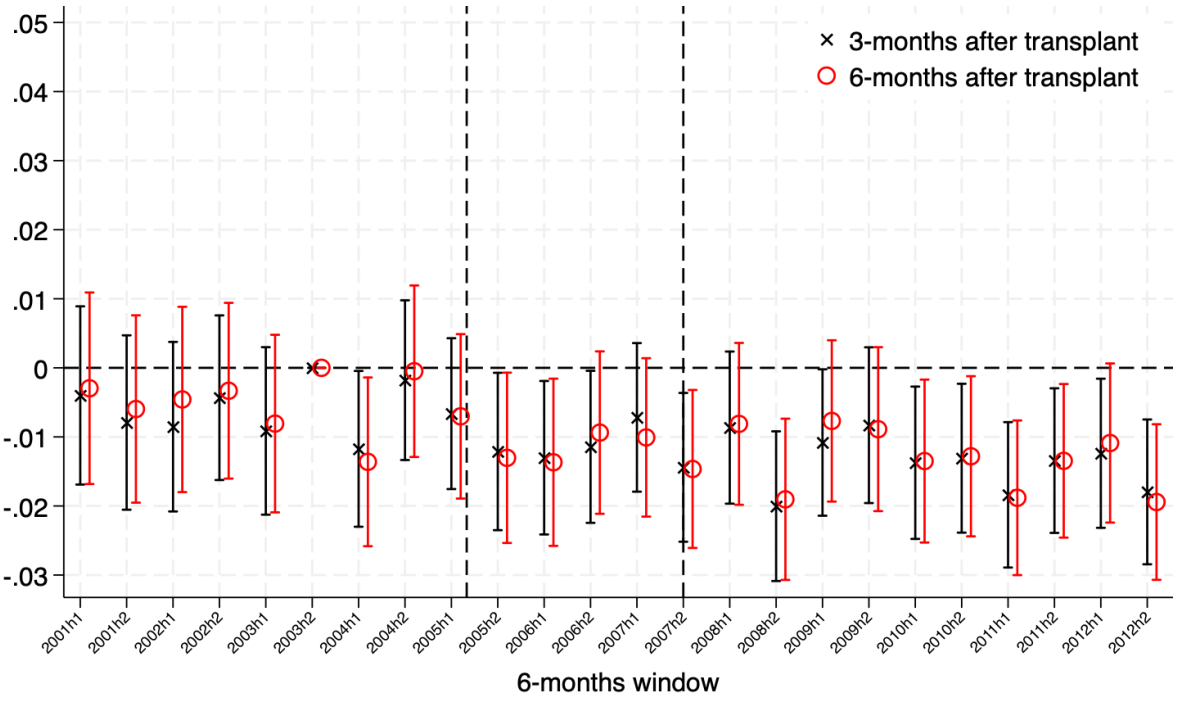


Figure C.7: Impact on viral infections 3-month/6-month after transplant

Note: The figure presents the estimated effect on the probability of getting infection 3 months/6 months after transplant, obtained using equation 6 with the instrument Z_c and 2003h2 as the reference 6-month window. I use robust standard errors. Error bars indicate 95 percent confidence intervals.

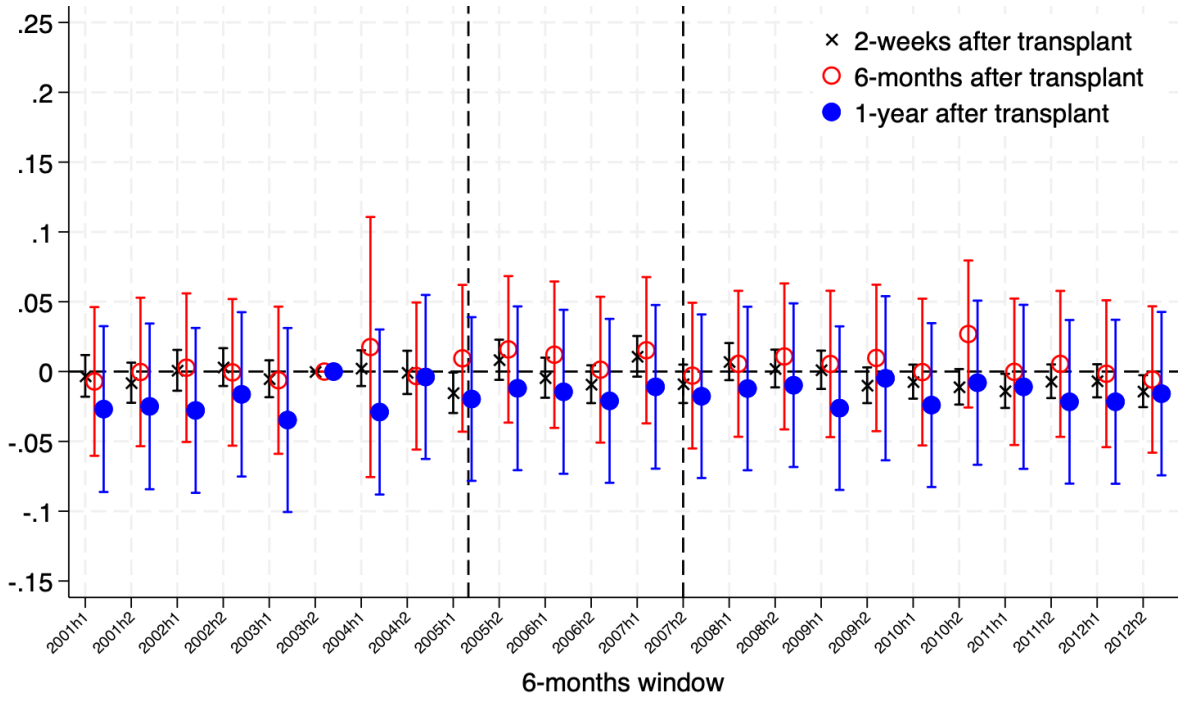


Figure C.8: Impact on acute kidney rejection at different follow-up periods

Note: The figure presents the estimated effect on the probability of acute kidney rejection at different follow-up periods, obtained using equation 6 with the instrument Z_c and 2003h2 as the reference 6-month window. I use robust standard errors. Error bars indicate 95 percent confidence intervals.

D Supplementary Tables

Table D.1: 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=467)	(N=658)
Dead within 6 months	92.0%	93.3%	94.4%
	(N=3078)	(N=2353)	(N=3207)
Dead within 1 year	94.0%	94.9%	96.0%
	(N=4451)	(N=3500)	(N=4699)

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.2: 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	36.7	52.5	53.8	52.1
	(17.3)	(17.1)	(17.0)	(17.0)	(16.5)	(16.1)
Creatinine Levels	1.1	1.1	1.1	1.4	1.5	1.5
	(1.0)	(0.8)	(0.9)	(1.1)	(1.1)	(1.1)
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%	60.7%	60.6%	52.2%	53.2%	52.4%
	(49.1)	(48.8)	(48.9)	(50.0)	(49.9)	(49.9)
White	72.4%	68.5%	67.6%	72.2%	68.9%	68.2%
	(44.7)	(46.4)	(46.8)	(44.8)	(46.3)	(46.6)
Death - Stroke	37.8%	35.9%	32.2%	65.9%	64.8%	56.3%
	(48.5)	(48.0)	(46.7)	(47.4)	(47.8)	(49.6)
Death - Head Trauma	47.2%	44.9%	39.4%	19.8%	17.6%	17.5%
	(49.9)	(49.7)	(48.9)	(39.9)	(38.1)	(38.0)
Hypertension	19.6%	24.1%	25.7%	52.7%	61.1%	61.2%
	(39.7)	(42.8)	(43.7)	(49.9)	(48.7)	(48.7)
Total Offers	95.0	69.9	176.3	796.4	545.0	1334.4
	(505.2)	(300.9)	(701.4)	(2352.7)	(1084.5)	(2430.6)
Observations	37975	33846	58325	5750	6634	12968

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, they would each count as an observation in the transplanted and discarded columns.

Table D.3: Tranplant patient characteristics pre and post-CoP

	Pre-CoP	Post-CoP	
		Announcement	Implementation
Age	47.9	49.4	50.8
	(14.5)	(15.3)	(15.3)
White	51.7%	48.8%	45.7%
	(50.0)	(50.0)	(49.8)
Years on WL	2.2	2.3	2.7
	(1.9)	(2.0)	(2.2)
Completed Univ.	14.2%	16.0%	19.1%
	(34.9)	(36.6)	(39.3)
Medicare	60.5%	61.0%	65.1%
	(48.9)	(48.8)	(47.7)
Diabetic	31.2%	33.8%	36.3%
	(46.4)	(47.3)	(48.1)
On Dialysis	55.9%	75.9%	75.7%
	(49.7)	(42.8)	(42.9)
Total Offers	55.9	69.5	133.3
	(72.3)	(103.0)	(209.7)
Ecpected Post-TX Survival	31.2	35.2	39.3
	(29.7)	(31.0)	(32.4)
Observations	36446	32575	55583

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 - December 2012) periods.

Table D.4: 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	-0.01057***	-0.02696***	-0.02777***	-0.00937	0.01012
	(0.00296)	(0.00480)	(0.00582)	(0.00768)	(0.01111)
Post-Implement	-0.01523***	-0.03123***	-0.02212***	0.00217	0.02823**
	(0.00266)	(0.00431)	(0.00524)	(0.00708)	(0.01058)
Y mean	0.02801	0.07573	0.11036	0.17144	0.23285
F-statistic	15805.65184	15197.02759	14713.68470	13759.31457	12762.85322
Fixed Effects	Center, 6-months	Center, 6-months	Center, 6-months	Center, 6-months	Center, 6-months
Observations	113803	109569	105264	97206	89312

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, 5. Each column uses the subsample of patients whose post-transplant mortality timeline do not overlap with the CoP announcement described in Section 5.3. I use robust standard errors. *$p < 0.1$, **$p < 0.05$, *** $p < 0.01$

Table D.5: Impact on the dosage of induction immunosuppressants/prescription of maintenance immunosuppressants

	Dosage	Prescription			
	(1)	(2)	(3)	(4)	(5)
	Before TX	2-weeks	6-months	1-year	2-years
Post-Announce	-0.13972***	-0.02201***	0.02110***	-0.00179	0.03767***
	(0.03582)	(0.00471)	(0.00401)	(0.00308)	(0.00677)
Post-Implement	-0.26706***	-0.03615***	0.05341***	-0.00613*	0.01145
	(0.03279)	(0.00410)	(0.00407)	(0.00300)	(0.00667)
Y mean	2.67955	0.84904	0.66734	0.79945	0.69618
F-statistic	15809.44128	15747.16465	14059.04112	13392.32853	10959.11957
Fixed Effects	Center, 6-months	Center, 6-months	Center, 6-months	Center, 6-months	Center, 6-months
Observations	114885	114535	102717	95384	83002

Note: This table relates to the analysis in Section 6.2.1. It presents the estimated effect on the dosage of induction immunosuppressants (Column 1) and probability of prescribing maintenance immunosuppressants (Columns 2 - 5) at different follow-up timelines, obtained by jointly estimating equations 4, 5. Each column uses the subsample of patients whose post-transplant mortality timeline do not overlap with the CoP announcement described in Section 5.3. I use robust standard errors. *$p < 0.1$, **$p < 0.05$, ***$p < 0.01$.

Table D.6: Impact on the rate of acute kidney rejection

	(1) 2-weeks	(2) 6-months	(3) 1-year	(4) 2-years
Post-Announce	-0.00756**	-0.00401	0.00878	0.01676**
	(0.00259)	(0.00488)	(0.00456)	(0.00539)
Post-Implement	-0.00605**	-0.00248	0.00434	0.00733
	(0.00234)	(0.00483)	(0.00444)	(0.00516)
Y mean	0.02981	0.06683	0.03720	0.03098
F-statistic	16396.47443	14157.17526	13533.28614	11654.98563
Fixed Effects	Center, 6-months	Center, 6-months	Center, 6-months	Center, 6-months
Observations	114885	95194	88568	77137

Note: This table relates to the analysis in Section 6.2.2. It presents the estimated effect on the probability of acute kidney rejection episodes being reported at different follow-up timelines, obtained by jointly estimating equations 4, 5. Each column uses the subsample of patients whose post-transplant mortality timeline do not overlap with the CoP announcement described in Section 5.3. I use robust standard errors. *$p < 0.1$, **$p < 0.05$, ***$p < 0.01$.

Table D.7: Impact on the rate of CMV testing

	(1) 2-weeks	(2) 6-months	(3) 1-year	(4) 2-years
Post-Announce	-0.01564***	0.03858***	0.04198***	0.06242***
	(0.00377)	(0.00549)	(0.00661)	(0.00779)
Post-Implement	0.00745*	0.02154***	0.03114***	0.01361*
	(0.00290)	(0.00411)	(0.00588)	(0.00653)
Y mean	0.95981	0.09167	0.28389	0.31329
F-statistic	15809.44128	14059.04112	13392.32853	10959.11957
Fixed Effects	Center, 6-months	Center, 6-months	Center, 6-months	Center, 6-months
Observations	114885	102717	95384	83002

Note: This table examines the proposed detection channel in section 6.2.2. 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, 5. Each column uses the subsample of patients whose post-transplant mortality timeline do not overlap with the CoP announcement described in Section 5.3. I use robust standard errors. *$p < 0.1$, **$p < 0.05$, ***$p < 0.01$.

Table D.8: Impact on acceptable donor criteria (i)

	(1) Min. Age	(2) Max. Age	(3) HLA Mismatch	(4) Creatinine
Post-Announce	0.03917	-0.73965*	0.00707	-0.82138
	(0.04781)	(0.32938)	(0.01299)	(1.34205)
Post-Implement	0.04438	0.78721	-0.01107	2.51965
	(0.10207)	(0.58262)	(0.01816)	(2.41131)
Y mean	1.22524	78.86188	5.94687	17.45362
Fixed Effects	Patient, Quarters	Patient, Quarters	Patient, Quarters	Patient, Quarters
Observations	26890	26890	26350	26890

Note: This table relates to the analysis in section 7.1. It presents the estimated effect on centers setting more stringent donor criteria, obtained by estimating equation 7. Each column represents a different modifiable donor criteria. I use robust standard errors. *$p < 0.1$, **$p < 0.05$, *** $p < 0.01$

Table D.9: Impact on acceptable donor criteria (ii)

	(1) Hypertension	(2) Cold Ischemic Time	(3) Warm Ischemic Time	(4) Expanded Criteria
Post-Announce	0.00039	1.23367	-0.09437	0.00127
	(0.00038)	(2.22015)	(1.06507)	(0.01270)
Post-Implement	0.00099	10.63076**	3.25256	-0.03059
	(0.00163)	(3.52796)	(1.87777)	(0.01971)
Y mean	0.99821	58.82800	66.02855	0.34128
Fixed Effects	Patient, Quarters	Patient, Quarters	Patient, Quarters	Patient, Quarters
Observations	26890	26890	23220	26890

Note: This table relates to the analysis in section 7.1. It presents the estimated effect on centers setting more stringent donor criteria, obtained by estimating equation 7. Each column represents a different modifiable donor criteria. I use robust standard errors. *$p < 0.1$, **$p < 0.05$, *** $p < 0.01$

Table D.10: Impact on accept decision across subgroups (i)

	(1) White	(2) Diabetic	(3) Uni.	(4) Medicare	(5) On dialysis
Post-Ann (Tri)	0.00304	-0.00043	0.00399	0.00442	0.00794
	(0.00437)	(0.00433)	(0.00492)	(0.00373)	(0.00432)
Post-Imp (Tri)	-0.00121	-0.00098	0.00192	0.00737	0.00507
	(0.00536)	(0.00393)	(0.00380)	(0.00507)	(0.00509)
Y mean	0.05689	0.05689	0.05689	0.05689	0.05689
Fixed Effects	Center, 6-months	Center, 6-months	Center, 6-months	Center, 6-months	Center, 6-months
Observations	880506	880506	880506	880506	880506

Note: This table relates to the analysis in section 7.2. It presents the estimated effect on the center's acceptance decision across different subgroups, obtained from triple differences regression interacted with different patient characteristics. I use robust standard errors. *$p < 0.1$, **$p < 0.05$, *** $p < 0.01$

Table D.11: Impact on accept decision across subgroups (ii)

	(1) White	(2) Diabetic	(3) Hypertension	(4) Death-Stroke	(5) Death-Head Trauma
Post-Ann (Tri)	0.00297	0.00683	0.00677	0.00045	-0.00105
	(0.00329)	(0.00437)	(0.00457)	(0.00324)	(0.00376)
Post-Imp (Tri)	-0.00236	0.00701	0.00350	-0.00021	-0.00284
	(0.00359)	(0.00422)	(0.00405)	(0.00330)	(0.00381)
Y mean	0.05689	0.05689	0.05689	0.05689	0.05689
Fixed Effects	Center, 6-months	Center, 6-months	Center, 6-months	Center, 6-months	Center, 6-months
Observations	880506	880506	880506	880506	880506

Note: This table relates to the analysis in section 7.2. It presents the estimated effect on the center's acceptance decision across different subgroups, obtained from triple differences regression interacted with different kidney characteristics. I use robust standard errors. *$p < 0.1$, **$p < 0.05$, *** $p < 0.01$

Table D.12: Impact on transplanted patient health

	(1) EPTS	(2) Creatinine
Post-Announce	-0.29275	-0.06437
	(0.24930)	(0.05155)
Post-Implement	-1.01252***	0.04067
	(0.22771)	(0.04788)
Y mean	36.42286	8.10584
F-statistic	14845.59951	14712.22787
Fixed Effects	Center, 6-months	Center, 6-months
Observations	107717	105265

Note: This table relates to the analysis in section 7.2. It presents the estimated effect on transplanted patient characteristics, obtained by estimating equation 5 I use robust standard errors. *$p < 0.1$, **$p < 0.05$, *** $p < 0.01$

Table D.13: Impact on admitted patient characteristics (i)

	(1) Age	(2) White	(3) No education	(4) Working	(5) Medicare
Post-Announce	-0.12504	-0.00304	-0.00187	-0.00836***	0.01617***
	(0.09173)	(0.00290)	(0.00220)	(0.00237)	(0.00317)
Post-Implement	-0.12850	-0.00152	-0.02350***	0.00202	-0.00072
	(0.07889)	(0.00249)	(0.00182)	(0.00207)	(0.00274)
Y mean	49.64180	0.47940	0.12864	0.19979	0.49139
Fixed Effects	Centers, 6-months	Centers, 6-months	Centers, 6-months	Centers, 6-months	Centers, 6-months
Observations	364083	364083	364083	364083	364083

Note: This table relates to the analysis in section 7.3. It presents the estimated effect on admitted patient characteristics, obtained by estimating equation 5. I use robust standard errors. *$p < 0.1$, **$p < 0.05$, *** $p < 0.01$

Table D.14: Impact on admitted patient characteristics (ii)

	(1) BMI	(2) Diabetic	(3) On dialysis	(4) Blood type O
Post-Announce	-0.14220***	-0.00001	-0.00246	0.13240
	(0.03880)	(0.00318)	(0.00258)	(0.32871)
Post-Implement	-0.15560***	0.00001	0.00029	0.14281
	(0.03303)	(0.00273)	(0.00221)	(0.28093)
Y mean	27.88551	0.40196	0.76968	48.53371
Fixed Effects	Centers, 6-months	Centers, 6-months	Centers, 6-months	Centers, 6-months
Observations	356297	364083	364083	364083

Note: This table relates to the analysis in section 7.3. It presents the estimated effect on admitted patient characteristics, obtained by estimating equation 5. I use robust standard errors. *$p < 0.1$, **$p < 0.05$, *** $p < 0.01$

Table D.15: Impact on kidney discard

	Kidney Discards		No. of Patients Offered	
	(1)	(2)	(3)	(4)
	Baseline	Risky Kidney	Baseline	Risky Kidney
Post-Announce	0.00720	-0.00780*	17.97013***	4.07020
	(0.00425)	(0.00329)	(4.85904)	(4.53587)
Post-Implement	-0.00513	-0.00867**	-61.44860***	-23.73487***
	(0.00350)	(0.00289)	(5.42300)	(4.91193)
Post-Announce (Tri)		0.03596***		35.81557***
		(0.00959)		(9.57540)
Post-Implement (Tri)		0.00522		-95.73517***
		(0.00791)		(11.08671)
Y mean	0.15538	0.15538	270.16646	270.16646
Fixed Effects	DSA, 6-months	DSA, 6-months	DSA, 6-months	DSA, 6-months
Observations	107444	107444	107444	107444

Note: This table relates to the analysis in section 8.1. It presents the estimated effect on kidney discard, obtained by estimating equation 8. I use robust standard errors. *$p < 0.1$, **$p < 0.05$, *** $p < 0.01$

Table D.16: Full patient sample

	Post-Transplant Death	CMV Testing	Immunosuppression
2-weeks after transplant			
Post-Ann	-0.004*	-0.013***	-0.040***
	(0.002)	(0.003)	(0.003)
Post-Imp	-0.007***	0.033***	-0.047***
	(0.002)	(0.002)	(0.003)
6-months after transplant			
Post-Ann	-0.011***	0.022***	0.005**
	(0.003)	(0.004)	(0.002)
Post-Imp	-0.014***	0.036***	0.013***
	(0.003)	(0.004)	(0.002)
1-year after transplant			
Post-Ann	-0.011***	0.031***	0.046***
	(0.004)	(0.004)	(0.004)
Post-Imp	-0.010***	0.017***	0.062***
	(0.003)	(0.004)	(0.003)

Notes: This table relates to the analysis in section 8.2. It presents the estimated effect on post-transplant death, CMV testing, and immunosuppressant prescription, obtained by estimating equation 5 with the full patient sample. I use robust standard errors. *$p < 0.1$, **$p < 0.05$, *** $p < 0.01$

Table D.17: Transplant centers have perfect foresight

	Post-Transplant Death	CMV Testing	Immunosuppression
2-weeks after transplant			
Post-Ann	-0.019**	0.006	0.041***
	(0.007)	(0.010)	(0.010)
Post-Imp	-0.027***	0.058***	0.010
	(0.006)	(0.007)	(0.009)
6-months after transplant			
Post-Ann	-0.051***	0.074***	0.016**
	(0.012)	(0.013)	(0.007)
Post-Imp	-0.051***	0.059***	0.009
	(0.011)	(0.012)	(0.006)
1-year after transplant			
Post-Ann	-0.048***	0.043***	-0.094***
	(0.014)	(0.015)	(0.014)
Post-Imp	-0.026**	0.018	-0.093***
	(0.013)	(0.012)	(0.014)

Notes: This table relates to the analysis in section 8.2. It presents the estimated effect on post-transplant death, CMV testing, and immunosuppressant prescription, obtained by estimating equation 5, but replacing the ρ_c with the flagged status of the center in 2007h2 when Medicare implemented CoP. I use robust standard errors. *$p < 0.1$, **$p < 0.05$, *** $p < 0.01$

Table D.18: Transplant centers have time-varying flagging beliefs

	Post-Transplant Death	CMV Testing	Immunosuppression
2-weeks after transplant			
Post-Ann	-0.000	-0.005***	-0.004*
	(0.001)	(0.002)	(0.002)
Post-Imp	-0.001	0.016***	-0.012***
	(0.001)	(0.001)	(0.002)
6-months after transplant			
Post-Ann	-0.004**	0.013***	0.002
	(0.002)	(0.002)	(0.001)
Post-Imp	-0.003*	0.001	0.000
	(0.002)	(0.002)	(0.001)
1-year after transplant			
Post-Ann	-0.005**	0.013***	0.013***
	(0.002)	(0.003)	(0.002)
Post-Imp	-0.002	0.002	0.030***
	(0.002)	(0.002)	(0.002)

Notes: This table relates to the analysis in section 8.2 . It presents the estimated effect on post-transplant death, CMV testing, and immunosuppressant prescription, obtained by estimating equation 5, but replacing the ρ_c with time-varying flagging beliefs. I use robust standard errors. *$p < 0.1$, **$p < 0.05$, *** $p < 0.01$