Performance Scores and Strategic Choices of Kidney Transplant Centers *

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Abstract

Kidney transplant centers are critical to patient survival, yet there is little oversight over their performance and behavior. This study examines the impact of a policy that terminates centers if risk-adjusted death rates exceed a limit. Using variation in policy exposure across centers and over time, combined with adjustments for statistical trends, I implement a difference-in-differences design to identify causal effects. The policy reduced post-transplant death rates by 19 percent, with the improvements coming from better detection of complications like acute kidney rejections during follow-up visits. While the policy did not cause discriminatory practices at the transplant or admission stages, nor did it increase waiting times or waiting-list deaths, it had unintended consequences. Notably, a 28 percent drop in risky kidney offerings by adjustments in the centralized allocation system led to an 18 percent rise in the discard rate. These findings challenge existing assumptions in the medical literature and demonstrate that regulatory oversight can enhance patient outcomes while maintaining equity and access. The study offers valuable lessons for regulators on balancing quality improvement initiatives with minimizing unintended consequences, such as kidney discards.

JEL codes: I11, I18, L38

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

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 their importance and 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 performance scores by leveraging 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,

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[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.

the most common post-transplant complication (Gjertson et al., 2002), can be mitigated by dedicating resources to early detection and optimizing immunosuppression regimens. Secondly, centers may engage in selection 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 performance scores 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 patient health and sets a transplant eligibility threshold, determining how many patients it expects to 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 source is administrative follow-up records for all transplant patients, comprehensive patient-kidney offers data, and CMS's CoP report. The dataset spans from

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[3]Source: New York Times

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

2001 to 2007, covering approximately four years before and two years after the 2005 CoP announcement. The follow-up data tracks each transplant patient's health and the outcomes of medical tests performed during the revisits. The patient-kidney offer dataset records all kidney offers, including information on the final decision, offer dates, reasons for declining, and detailed patient and kidney characteristics. The CoP report documents the center's flagging status in 2007, 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., hospitals 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

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assumption, I present nonparametric estimates of dynamic effects on all key outcomes.

The baseline IV estimates imply that after the policy announcement, a one standard deviation increase in center belief caused a 2.3 percentage point (pp) (19 percent) decrease in post-transplant 1-year mortality on average; OLS estimates are substantially larger, consistent with upward bias due to mean reversion. This estimate will understate the aggregate effects of the penalty.

Applying the same research design, I test and quantify the role of both response mechanisms, beginning with post-transplant care. Using follow-up data, I examine how centers prevent, detect, and treat acute kidney rejection. First, I find no evidence that centers increased their prescription or dosage of immunosuppressant medicines following the CoP announcement. Secondly, the rate of acute kidney rejection rose by 61%, driven by increased diagnostic testing to detect potential rejections rather than by centers matching patients with unsuitable kidneys. Finally, I find no evidence that centers altered their treatment strategies, such as increased hospitalization or immunosuppressant prescription, for patients diagnosed with rejection. These findings suggest that centers shifted their focus toward detection rather than prevention or treatment, likely reflecting a strategy to mitigate post-transplant mortality risks through early diagnosis.

Next, I examine the role of three potential selection channels. First, centers can set acceptable donor criteria to filter kidney offers for their patients. However, my analysis finds no evidence that centers implemented more stringent donor criteria to restrict kidney offers. Secondly, while there is no evidence of filtering, this does not preclude the possibility of selection; centers might want to review all potential kidney offers before deciding. Using comprehensive data on patient-kidney offers, I find no evidence that centers discriminate against patients or kidneys with high-risk scores. Finally, I investigate whether centers admit patients with more favorable characteristics or demographics and find no evidence supporting this hypothesis. These findings suggest that centers do not actively discriminate against patients or kidneys at any stage of the transplant process.

To broaden my analysis, I examine the effect of CoP on transplant wait time and kidney wastage-two critical inefficiencies beyond CoP's focus but emphasized in the Biden-Harris administration's "Increasing Organ Transplant Access (IOTA)" model (CMS, 2024). I find no evidence that the CoP announcement led to longer waitlist times or increased waitlist mortality. However, the discard rate for high-risk score kidneys did rise, driven not by changes in center behavior but by adjustments in the centralized allocation system that reduced the number of patients receiving offers for these kidneys. These findings highlight CoP's success in addressing post-transplant outcomes while leaving other systemic inefficiencies unaddressed, underscoring the need for complementary policies like IOTA that target these broader challenges.

1.1 Related literature

This paper contributes to three main strands of literature. First, it engages with the economic debate on the merit of performance scores, which primarily focuses on how report cards alleviate consumer information asymmetry and incentivize firms to improve quality (Jin and Sorensen, 2006; Kolstad, 2013; Atal, Cuesta and Sæthre, 2022; Barahona, Otero and Otero, 2023; Vatter, 2023). Unlike prior work, this study examines a setting where information asymmetry, though present, plays a limited role in patient choice of transplant centers due to logistical constraints[5]. Instead, I focus on how performance scores influence centers' selection and quality improvement decisions. Closely related studies, such as Dranove et al. (2003) and Gupta (2021), explore the effects of performance scores and pay-for-performance policies in other healthcare contexts, such as coronary artery bypass grafts and hospital readmissions. My use of follow-up data provides a unique advantage, allowing the identification of specific channels through which centers improve quality in response to penalties.

Second, this paper contributes to economic research on deceased donor organ transplants,

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[5]Patients often cannot afford to travel to distant transplant centers due to the frequent dialysis treatments required, which restricts choice (Schaffhausen et al., 2019).

which predominantly examines the design of allocation systems (Su and Zenios, 2005; Bloch and Cantala, 2017; Zhang, 2010; 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. 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 limitations 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 approach complements existing work using novel follow-up data to assess 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 and research design. Section 5 presents mortality and post-transplant care results. Section 6 presents results on selection effects. Section 7 highlights the effects of CoP on non-targeted metrics. Section 8 discusses the results and 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 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. 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

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[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.

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 might be unavailable due to a family emergency. The transplant center does not contact every compatible patient because of the tight deadline[8]. It usually informs the patient after UNet confirms the center's acceptance (Husain et al., 2019; King et al., 2023)

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 [9]. 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

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

[9]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.

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 6, 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 discharge transplant patients within 8-14 days. During this period, patients take immunosuppression drugs to prevent kidney rejection. After discharge, patients will visit the transplant center for regular check-ups at 6 months, 1 year, 2 years, etc.

Some degree of kidney rejection is to be expected; about $15\% - 20\%$ of transplanted patients will experience some rejection[10]. Acute kidney rejection is an immune response within the first 12 months of a transplant. T-cells and antibodies attack the foreign kidney, leading to impairment and graft failure (Becker et al., 2022). Symptoms, if present, might include fever, pain over the transplant site, and reduced urine output, but kidney rejection is often asymptomatic. Doctors typically detect subtle signs through biomarkers or changes in kidney function metrics, such as creatinine levels. Hence, early detection during follow-up sessions plays a crucial role in preserving kidney function (Sharaby et al., 2023).

In section 5.2, I leverage the follow-up data that track patient health status and the outcome of all medical tests performed to study how CoP announcement affects how the center prevents, detects, and treats 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

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[10]Source:Cleveland Clinic

concerned that the lack of severe penalties for poor performance may have led to a decline in the quality of kidney transplants[11]. 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 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 B.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

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

(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 states that the center cannot have too many observed deaths; criteria two and 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)[12].

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.

3   Stylized model of center behavior

In this section, I formalize the transplant center's incentives and how CoP affects decision-making. I present a stylized model where the center observes patient health status. Then, the center chooses the transplant eligibility threshold and the amount of post-transplant care. In choosing the threshold and post-transplant care, the center trades off the additional revenue from more transplants and post-transplant care with the cost of these transplants - the likelihood of exceeding the CoP limit if too many patients die. More post-transplant

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[12]I account for transplant volume and unadjusted mortality in Section 4.1 when constructing center flagging beliefs.

	Stage 1	Stage 2	Stage 3

Stage 1

Observe: health, x
Choice: threshold, t

Stage 2

Choice: post-transplant care, $q(x)$

Stage 3

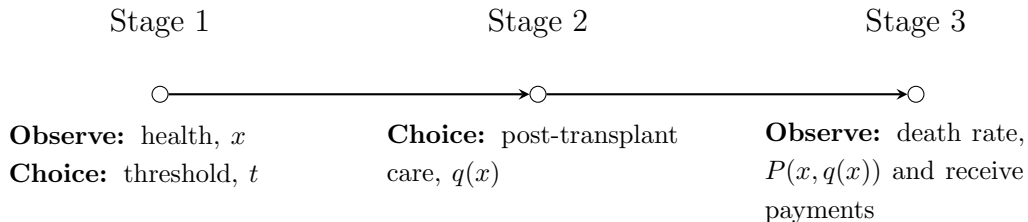**Observe:** death rate, $P(x, q(x))$ and receive payments

Figure 1: Timeline of the center behavior

care potentially improves patient survival but is costly to the patient. The model delivers two predictions about how centers respond to CoP implementation. First, by raising the marginal cost of each transplant, CoP leads centers to reduce transplants. Second, by raising the marginal benefit of care, CoP incentivizes centers to increase post-transplant care. In subsequent analysis, I model patient death in my setting. Then, I describe the center's objective function and characterize the optimal transplant threshold and post-transplant care. Finally, I provide comparative statics on key parameters and present proofs in Appendix A. Figure 1 illustrates the center's timeline and decision-making.

3.1   Setup

I model patient death after receiving a kidney transplant and post-transplant care. I characterize patient health status as x, where $x \in \mathbb{R}_0^+$ is drawn from a known distribution $F(.)$ [13]. After receiving a transplant, centers decide on post-transplant care $q(x)$. Transplant patients die if the latent variable $y > 0$, where $y = \varepsilon - x - q(x)$ and $\varepsilon \sim N(0, \sigma^2)$. ε is a normally distributed idiosyncratic shocks with mean 0 and variance σ^2. Let the likelihood that a patient with health status x and post-transplant care $q(x)$ die 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 threshold t, the center expects

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

$\int_t^\infty P(x, q(x))dF(x)$ patients to die[14].

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).

The patient's utility from post-transplant care is concave in $q(x)$, with healthier patients (higher x) benefiting more from kidney transplant (Senanayake et al., 2020). The key assumption is that patients have a bliss point in $q(x)$. Excess care does not necessarily become physically harmful, but it can be interpreted as the patient's coinsurance kicking in or the opportunity cost of their time. The patient receives zero if centers do not perform a transplant. The center maximizes utility and chooses $t,q(x)$ to maximize a weighted average of their profit and the patient's utility from transplant[15]:

$$\max_{t,q(x)} E[U(t, q(x)] = \int_t^\infty \left[\rho \overbrace{[\pi + \alpha q(x)]}^{\text{center profit}} + (1-\rho) \overbrace{\left[xq(x) - \frac{\gamma}{2}q^2(x) \right]}^{\text{patient utility}} \right] dF(x) \tag{1}$$

$$\text{s.t. } \rho \int_t^\infty P(x, q(x))dF(x) \leq \tau$$

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[14]For brevity, I model the patient death as a function of post-transplant care and health status and abstract from the quality of the kidney.

[15]The center's optimal transplant strategy is a cutoff strategy (i.e., transplant if $x \geq t$; otherwise not). Intuitively, if the center optimally avoids transplant with a positive probability for some $x > t$, then there must exist $\tilde{t} > t$, and the center chooses transplant if and only if $x > \tilde{t}$, such that the overall probability of transplant remains the same. However, the center has a higher expected utility due to the higher expected x. Threshold $\tilde{t}$ then constitutes a profitable deviation. I provide the proofs in Appendix A.

τ is the CoP limit, and the rest of the terms in the constraint reflect the CoP conditions in Section 2.4. $\int_t^\infty P(x, q(x))dF(x) \leq \tau$ is equivalent to CoP condition 1 (i.e., death ratio cannot exceed a limit). However, even if it does, the center can still escape CMS flagging if conditions 2 and 3 are unmet. In this setting, ρ mimics CoP conditions 2,3 and is a scaling factor that makes it less likely to exceed the CoP limit, τ.

Intuitively, the center wants to transplant as many patients and provide as much care as possible to maximize profits but is constrained by performance concerns and patient preferences. Transplanting too many patients exposes the center to the CoP limit τ, and patients dislike too much post-transplant care due to the increasing marginal cost of care, $\gamma > 0$. The center optimizes the tradeoffs between these countervailing incentives when choosing the optimal transplant threshold rule, t, and post-transplant care regimen, $q(x)$. Next, I characterize t, $q(x)$ and present the proofs in Appendix A.

Proposition 1. *All patients with $x \in [t, \infty)$ receive a transplant and post-transplant care. t, $q(x)$ are defined implicitly by the following system of equations:*

$$\gamma q(x) = x + \frac{\rho}{1-\rho}\left[\alpha + \frac{\lambda}{\sigma}\phi\left(\frac{-x - q(x)}{\sigma}\right)\right];$$

$$\lambda\Phi\left(\frac{-t - q(t)}{\sigma}\right) = \rho\left[\alpha q(t)\right] + (1 - \rho)\left[tq(t) - \frac{\gamma}{2}q(t)^2\right];$$

$$0 = \lambda\left(\rho\int_t^\infty \Phi\left(\frac{-x - q(x)}{\sigma}\right)\partial F(x)\right)$$

where λ is the Lagrange multiplier and $\phi(.)$ is the standard normal density function. Patients with $x \in [0, t)$ do not receive a transplant, and the quality of care, $q(x) = 0$

3.2   Comparative statics

In this stylized model, the pre-CoP announcement reflects $\tau \to \infty$, and death rate rules are not stringent; the post-CoP announcement is when τ is finite. The following result illustrates the comparative statics for transplant threshold τ and post-transplant care $q(x)$

as CMS announces CoP (i.e., τ decreases) or when the center's likelihood of punishment increases (i.e., ρ increases).

Proposition 2. *As CMS announces CoP (i.e. τ decreases) or likelihood of punishment increases (i.e. ρ increases), transplant threshold t increases $\left(\frac{\partial t}{\partial \tau} < 0, \frac{\partial t}{\partial \rho} > 0\right)$; post-transplant care $q(x)$ increases $\left(\frac{\partial q(x)}{\partial \tau} < 0, \frac{\partial q(x)}{\partial \rho} > 0\right)$.*

Intuitively, as τ decreases or ρ increases, the CoP limit becomes more stringent, and the likelihood of exceeding the CoP limit increases. Centers reduce transplants (t increases) because of the drop in the marginal benefit of transplant and increase post-transplant care ($q(x)$ increases) to avoid exceeding the CoP limit. I present the proofs in Appendix A.

4 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. 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 expectations of exceeding CoP limits, conditional on information available at the end of the prior six-month window. The linear equation below represents a

16

static version of this economic model:

$$Y_{ickt} = \alpha_c + \delta_t + \beta\mathbb{E}[\mathbf{1}(\mathbf{CoP_{ct}} > \mathbf{\bar{CoP}})|I_{t-1}] \times \mathbf{1}(t \geq 2005)_t + X'_{ik}\gamma + \varepsilon_{ickt} \qquad (2)$$

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[16]. The variable ε_{ickt} captures omitted factors affecting mortality, while X_{ik} accounts for patient and kidney risk factors. The parameter β measures the average change in outcomes between 2005 and 2007 for centers with a 10% probability of being flagged.

4.1   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, conditional on past performance, centers have rational expectations.

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

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[16]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.

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

$$\widehat{\mathbb{E}}[\mathbf{1}(\mathbf{CoP_{ct}} > \bar{\mathbf{CoP}})|I_{t-1}] = \widehat{f}(R_{c,t-1}, TX_{c,t-1})$$

(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 B.3. Hence, I hold the probability of flagging calculated using these penalty statuses and denoted as ρ_c.

In Figure 2, the average (median) transplant center has a 10%(7%) likelihood 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% 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 B.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,
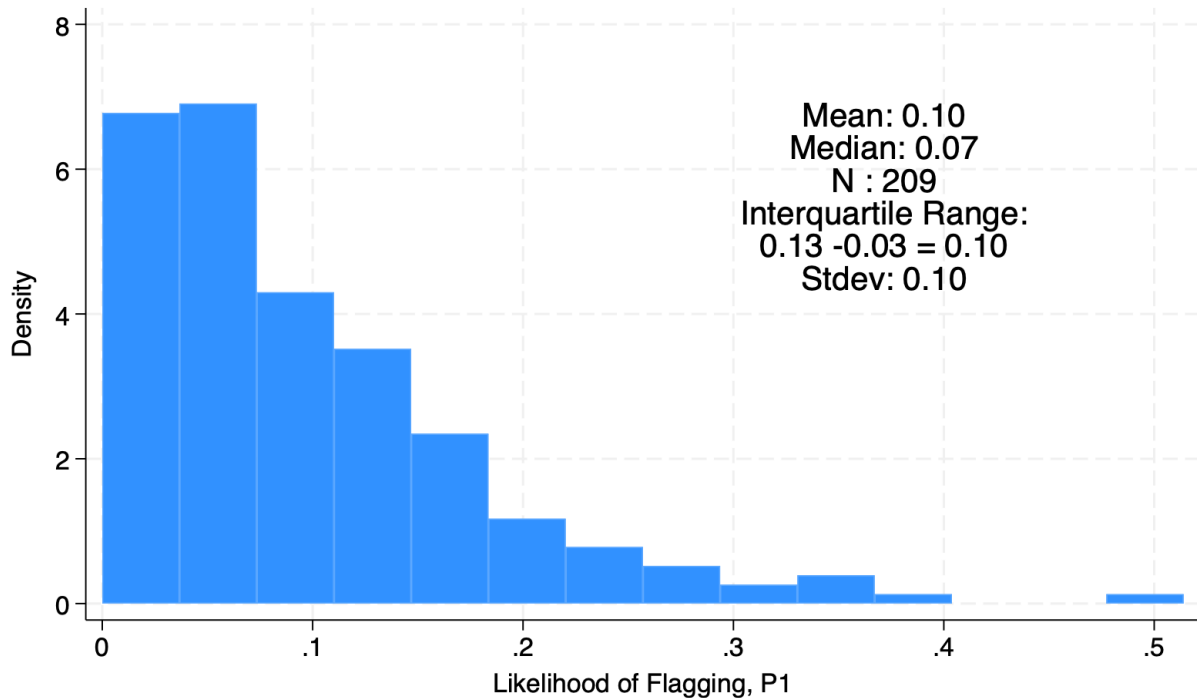
18

Figure 2: 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.

centers flagged twice within 30 months face the threat of CMS decertification, which could effectively 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.

4.2   Mean Reversion

The OLS regression in the previous subsection could overestimate the effect of the CoP announcement due to the possibility of mean reversion Chay, McEwan and Urquiola (2005). 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). When these centers revert to their true, higher mean in the future, it will appear that the penalty

did not motivate them to improve. I overcame this concern by using an instrumental variables approach where I relied on variation in center quality in 2004-2005, 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. This approach also has the benefit of eliminating other possible measurement error sources in computing center expectations.

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 hospital-level instrument Z_c predicted using baseline covariates. The IV approach also mitigates concerns of measurement error in constructing hospital 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:

$$\rho_c \times \mathbb{1}(t \geq 2005) = \pi_c + \pi_t + \lambda Z_c \times \mathbb{1}(t \geq 2005) + u_{ickt} \tag{4}$$

$$Y_{ickt} = \alpha_c + \delta_t + \beta \rho_c \times \mathbb{1}(t \geq 2005) + X'_{ik}\gamma + \varepsilon_{ickt} \tag{5}$$

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 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:

$$Y_{ickt} = \alpha_h + \delta_t + \sum_{s \neq 2004h2} \beta_s \mathbf{1}(d_{Z_h=1}) \times \mathbf{1}(t=s) + \varepsilon_{ickt} \tag{6}$$

where d_{Z_h} is an indicator set to 1 if center c is the top half of all centers, ranked by the instrument Z_h. Recall from the discussion in Section 1 that centers with the highest historical mortality rate have the highest penalty risk and, therefore, the most incentive to improve.

4.3 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 B.5 illustrates the two kinds of patients discussed above.

The primary threat to the identification of this approach is that the composition of patients across centers may have changed in essential and potentially unobservable ways. I address this concern in Section 6 by showing that centers do not discriminate against different profiles of patients or kidneys at the transplant or admission stages.

5 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.

5.1 Targeted metric
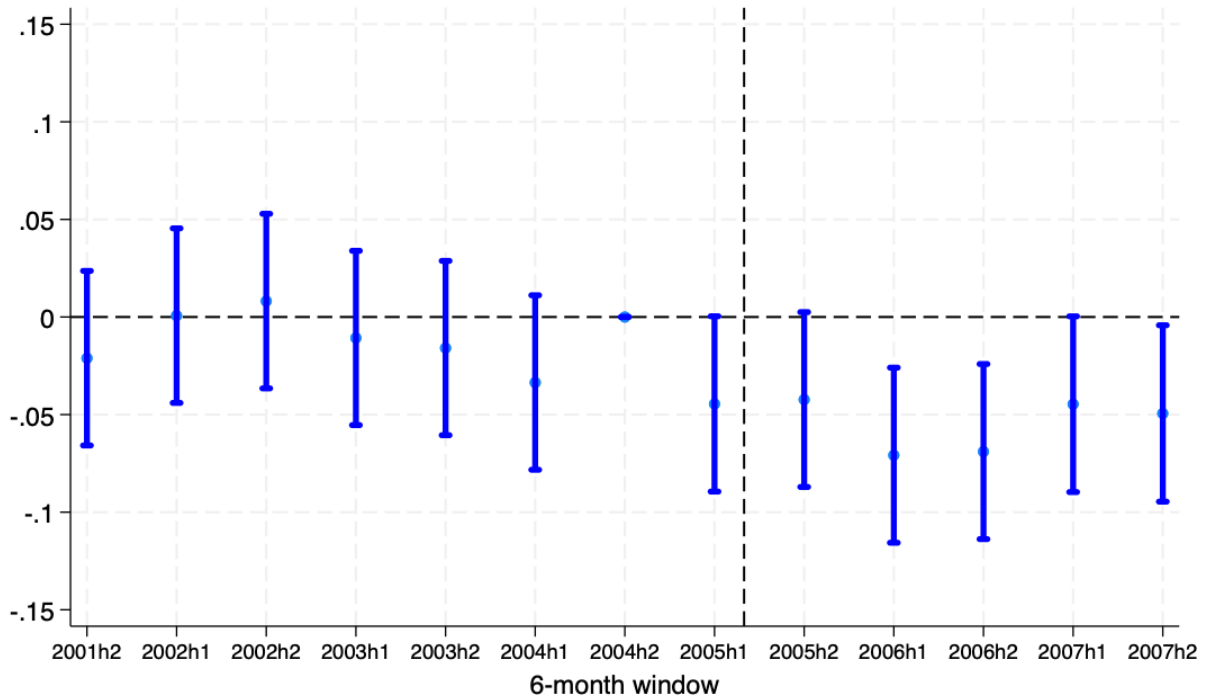
Figure 3: Impact on post-transplant 1-year mortality

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 2004h2 as the reference 6-month window. I use robust standard errors. Error bars indicate 95 percent confidence intervals.

Figure 3 plots the coefficients β_s for 6-month windows between 2001 and 2007, with 2004h2 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

22

exist between centers with low and high values of Z_h, indicating that the parallel trends assumption holds. 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. This suggests that the no-anticipatory assumption in prior studies, which focus solely on post-2007 behavior, may overlook essential center responses during the announcement period.

Table 1 presents OLS (Column 1) and IV (Column 2) estimates, showing a 2.37 percentage point reduction in post-transplant 1-year mortality for a 1-standard deviation increase in a center's belief of being flagged. The IV estimates are larger than the OLS estimates, consistent with concerns that mean reversion may underestimate the CoP response. Further analysis in Table C.1, using granular time intervals, shows that improvements are most pronounced within the first two weeks (46%) and six months (53%) post-transplant, indicating that mitigation efforts are concentrated in these critical periods.

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

	(1) OLS	(2) IV
DiD estimates	-0.01227**	-0.02371***
	(0.00406)	(0.00555)
Y mean	0.12496	0.12496
F-statistic		25811.09616
Fixed Effects	Center, 6-months	Center, 6-months
Observations	55432	55432

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 4.3. I use robust standard errors.

Interpreting these results, the 19% reduction in post-transplant mortality represents a substantial improvement. For context, in 2004, 11% of the 10,370 kidney transplant recipients died within a year. A 2.37 percentage point decline implies that 895 patients died

post-transplant, compared to 1,147 previously—a 22% decrease. These findings underscore the significant impact of CoP in improving transplant outcomes and suggest that centers prioritize immediate and intermediate post-transplant care to achieve these gains.

5.2 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 weakly increase post-transplant care on average, with more significant responses by centers that believe they are more likely to be penalized (i.e., $\frac{\partial q(x)}{\partial \rho} > 0$).

I use novel follow-up data that track patient health status and outcomes of all medical tests performed during revisits to examine how CoP affects post-transplant care. I focus on the most common post-transplant complication, acute kidney rejection (Gjertson et al., 2002), as mentioned in Section 2.3. This section carefully considers and chronologically analyzes how centers prevent, detect, and treat acute kidney rejection.

5.2.1 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[17]. This section focuses on induction and maintenance medicines, with rejection medicines discussed in Section 5.2.3.

Table 2 examines centers' use of induction medicines at the start of the transplant process. The analysis indicates that centers have reduced the number of medicines and duration of induction therapy, as reflected in the number of days of drug induction. For maintenance medicines, Table C.2 explores prescriptions during follow-up revisits. While maintenance medicines are critical for long-term graft survival, I find no consistent evidence that centers increased their likelihood of prescribing them during the analysis period[18]. These findings suggest that centers have not increased their reliance on immunosuppressants since the CoP announcement.

Table 2: Impact on immunosuppressants after transplant

	(1) Number of Drugs	(2) Days of Induction
DiD estimates	-0.01413**	-0.14079***
	(0.00434)	(0.03405)
Y mean	0.82468	3.46250
F-statistic	57566.45023	57566.45023
Fixed Effects	Center, 6-months	Center, 6-months
Observations	65066	65066

Note: This table presents an estimated effect on the use of immunosuppressants after the transplant, obtained by jointly estimating equations 4, 5 on patient-level data. I use robust standard errors.

One potential explanation for these patterns in Tables 2 and C.2 is that centers aim to balance reducing rejection rates with minimizing the risks associated with excessive immunosuppressive therapy. While these drugs effectively prevent rejection, they suppress the immune system, increasing susceptibility to complications such as viral infections (Pilch,

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[17]Source: UNOS, types of immunosuppresants

[18]Unfortunately, the data does not include dosage information, limiting further interpretation.

Bowman and Taber, 2020). This trade-off necessitates careful decision-making to optimize patient outcomes while managing treatment risks. Additionally, the range of available immunosuppressants remained relatively stable during the analysis period (2001-2007), with no significant breakthroughs in drug efficacy (Cooper, 2020). This stability suggests that the observed prescription reduction reflects clinicians refining their prescribing strategies by optimizing dosages and tailoring treatments to individual patients rather than improvements in drug efficacy.

5.2.2 Detection

Acute kidney rejection is often asymptomatic. Centers typically detect subtle signs through biomarkers or changes in kidney function metrics such as creatinine levels. Consequently, early detection during follow-up visits is critical for preserving graft function (Sharaby et al., 2023). Table C.3 demonstrates that acute kidney rejection increased by 1.26 percentage points (61%) during 1-year follow-up visits after CMS announced CoP.

Two potential channels may explain this increase in rejections. The first is the detection channel: after the CoP announcement, transplant centers became more diligent in performing medical checks for rejections, resulting in higher detection rates. The second is the matching channel: less effective matching of patients with suitable kidneys may have led to an increased likelihood of rejections during follow-up visits. I leverage follow-up data to investigate the relative contributions of these channels to the observed rise in rejection cases.

The cytomegalovirus (CMV) test, while not a direct diagnostic tool for acute kidney rejection, plays a vital role in detecting complications that can signal or contribute to rejection[19]. CMV infection or reactivation is a common issue in kidney transplant recipients due to immunosuppression and can trigger immune activation, potentially leading to graft rejection. Regular CMV testing helps identify viral infections early, allowing for timely an-

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[19]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.

tiviral treatment to prevent immune-mediated damage (Hasanzamani et al., 2016). Table C.4 shows CMV testing increased by 3.1 pp (22%) during 1-year follow-up visits after the CoP announcement, suggesting greater diligence in rejection detection.

While centers increased testing, the analysis of the matching channel did not yield evidence supporting its role in the observed increase in acute kidney rejection. Table C.5 examines various patient health outcomes during 1-year follow-up revisits and finds no significant indications of poor matching contributing to rejection rates.

The analysis reveals that the observed increase in acute kidney rejection incidence following the CoP announcement is primarily driven by heightened detection efforts rather than poor matching practices. The significant rise in CMV testing underscores the role of improved diligence in post-transplant monitoring, allowing centers to identify potential rejection cases more effectively. While the matching channel was hypothesized as a potential contributor, no evidence supports this claim, indicating that centers maintained their standards for patient-kidney compatibility. These findings highlight the impact of regulatory oversight in incentivizing more robust follow-up care and detection protocols, ultimately improving transplant outcomes.

5.2.3 Treatment

While acute kidney rejection is a serious complication, it is not an irreversible outcome; timely and effective treatment can often save the failing kidney and restore graft function (Vitak, 2014). After detecting rejection, CoP could incentivize centers to adopt more aggressive interventions, such as increased hospitalization or enhanced immunosuppressive regimens, to stabilize the graft and prevent further complications. I examine these possibilities in Table C.6 and C.7 but find no consistent evidence that centers increased hospitalization rates or immunosuppressant prescriptions for patients diagnosed with rejection.

One potential explanation is that, as discussed in Section 5.2.1, centers strive to balance the need to treat rejection with minimizing the risks of excessive immunosuppression, which

can expose patients to other complications, such as infections. Additionally, the treatment protocols for acute kidney rejection are highly standardized, leaving limited room for significant variation in approach (Eckardt, Kasiske and Zeier, 2009). This suggests that the most impactful gains in managing rejection arise from early detection rather than adjustments in prevention or treatment strategies.

6   Alternate Mechanisms

Quantifying the role of distortions (if any) in producing the average 2.37 decline in post-transplant 1-year mortality reported above is vital. This section carefully considers and chronologically analyzes three potential selection mechanisms: donor filtering, selection into transplant, and strategic admission.

6.1   Donor filtering

In Section 2.1, the center registers patients onto the national waitlist and uploads their medical and biological information. Simultaneously, the center can filter donors for patients in the UNet system (King et al., 2022; Yu et al., 2024). For instance, a center might set a maximum donor age of 80 for one of its patients, ensuring donors above 80 would not be offered, even if biologically compatible. Using patients' waitlist records, I document all modifications to donor criteria made by centers during the patient's time on the waitlist and analyze whether these adjustments reflect an attempt to filter kidney offers. The rationale is that CoP incentivizes centers to avoid risky kidney profiles, potentially by restricting the arrival of such kidneys through stricter donor criteria.

To identify this effect, I leverage patients whose time on the waitlist overlaps with the CoP announcement. Using a patient fixed-effects regression, I isolate the effect of CoP on donor filtering by examining within-patient changes in donor criteria[20]. Tables C.8 and C.9

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[20]To minimize potential temporal confounds (i.e., centers become less stringent with donor criteria as patients' wait time increases), I restrict my period of analysis to 2004 - 2006 and patients that had waited

present estimates for various donor characteristics that centers can modify[21]. The analysis finds no consistent evidence that centers tighten donor criteria to exclude kidneys with undesirable characteristics. However, the absence of donor filtering does not preclude the possibility of selection against risky kidney profiles. Centers may want to evaluate all potential kidney offers before deciding on behalf of their patients. I investigate this potential selection behavior further in the following subsection.

6.2 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 4 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 flattens by 2007, highlighting a potential stabilization in acceptance behavior. On average, Table 3 indicates a 0.5 percentage point decline in acceptance probability, a relatively small change given the mean acceptance rate of 16 percentage points.

To examine whether centers avoided certain patient or kidney profiles to reduce mortality risk, I estimated triple-difference models that interacted center penalty risk with predetermined patient and kidney risk measures[22]. These models test whether acceptance probabilities changed more for high-risk profiles than low-risk ones. Columns 3–5 in Table 3 show

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less than 6 months and have at least 2 more years on the waitlist.

[21]Some criteria (e.g., diabetes, hypertension, obesity, 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

[22]I use kidney donor profile index (KDPI) and estimated post-transplant survival (EPTS) as my kidney and patient risks measurement respectively. A kidney is high-risk if $KDPI \geq 0.5$, and a patient is high-risk if $EPTS \geq 0.5$. A patient-kidney pair is high-risk if both patient and kidney are high-risk.
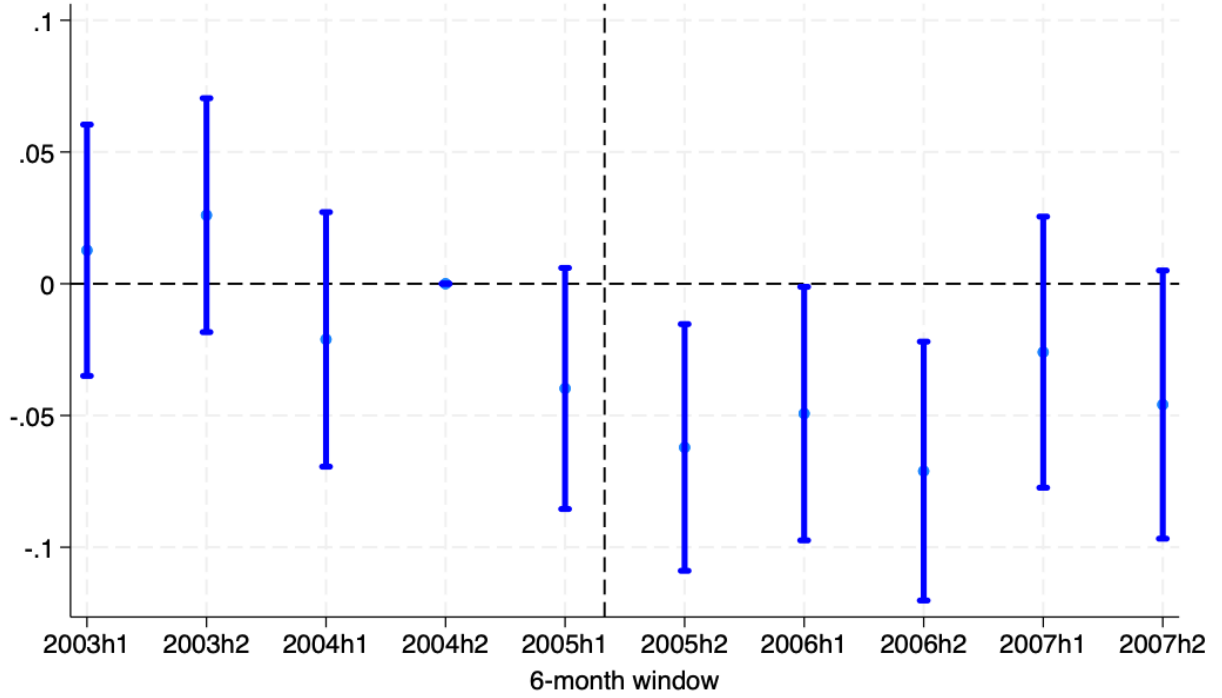
Figure 4: Impact on patient-kidney offer acceptance

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 2004h2 as the reference 6-month window. I use robust standard errors. Error bars indicate 95 percent confidence intervals.

that the triple-difference coefficients are statistically insignificant, suggesting that centers did not systematically avoid riskier profiles.

These findings indicate that while centers accept a slightly lower proportion of patient-kidney pairs, this behavior does not correlate with observed health risk factors that influence mortality risk. Assuming unobserved factors align with observed risk, these changes in transplant decisions are unlikely to account for the improvements in mortality and quality discussed in Section 5.

Table 3: Impact on selection into transplant

	Accept Decision (DiD)		Accept Decision across subgroups (Triple DiD))		
	(1)	(2)	(3)	(4)	(5)
	OLS	IV	Patient	Kidney	Patient x Kidney
DiD estimates	-0.00536***	-0.00578**	-0.00591***	-0.00671***	-0.00633***
	(0.00118)	(0.00179)	(0.00127)	(0.00197)	(0.00127)
Triple DiD estimates			0.00135	0.00236	0.00200
			(0.00303)	(0.00233)	(0.00337)
Y mean	0.16750	0.16750	0.16750	0.16750	0.16750
F-statistic		124056.00081			
Fixed Effects	Center, 6-months	Center, 6-months	Center, 6-months	Center, 6-months	Center, 6-months
Observations	364758	367272	364758	364758	364758

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.

6.3 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[23]. To test this hypothesis, I examine various socio-economic and health indicators among admitted patients. As shown in Tables C.10 and C.11, 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 factors.

One potential explanation for these patterns is that 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.

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

7 Effects on non-CoP metric

While CoP successfully reduced post-transplant mortality by improving centers' post-transplant care practices, it does not cover all inefficiencies. This section examines the broader implications of CoP for transplant wait time and kidney wastage, two metrics emphasized in the recent Biden-Harris administration's "Increased Organ Transplantation Access (IOTA)" model.

7.1 Wait time and waitlist mortality

Approximately 100,000 people are on the kidney transplant waitlist, with average wait times of three years or more Stolberg (2023). Transplant wait times are a critical measure of the system's efficiency and fairness, as prolonged waits increase the risk of complications and reduce the likelihood of successful transplantation. A potential concern is that the Conditions of Participation (CoP) policy may incentivize centers to modify their decision-making processes to avoid penalties, potentially altering patients' likelihood of receiving a transplant or remaining on the waitlist for extended periods. Table C.12 examines this concern and finds no statistically significant evidence that patient wait times increased after the CoP announcement. Column 1 shows no overall increase in wait times, while Column 2 compares transplanted and non-transplanted patients, revealing no significant differences between the two groups. Column 3 assesses waitlist mortality and finds no evidence of increased patient mortality off the waitlist after the CoP announcement.

These results support the findings in section 6.2, which indicate that centers do not use selective practices when determining which patients receive transplants. These findings suggest that the CoP policy did not negatively impact patient wait times or waitlist mortality, further emphasizing the policy's neutrality in pre-transplant patient management.

7.2 Kidney discard

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[24]. 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 collapse data on all patient-kidney offers to the kidney level and construct a weighted average of center penalty exposure, $Exposure_k$[25]. The following equation represents the discard model:

$$D_{kdt} = \alpha_d + \delta_t + \beta Exposure_k \times CoP_t + X_k'\gamma + \varepsilon_{kt} \tag{7}$$

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, captures whether kidneys offered to more exposed centers are more likely to be discarded. Table C.13 shows the results. Column 1 finds no evidence that overall kidney discard increased after CoP implementation. However, Column 2 reveals that high-risk kidneys are 2.71 percentage points (18%) more likely to be discarded than low-risk kidneys.

I further examine the factors driving the higher discard rate for high-risk kidneys. Column 4 shows that UNOS offered high-risk kidneys to an average of 50 fewer patients than low-risk kidneys despite their viability for up to 24–48 hours. This disparity could reflect logistical bottlenecks, such as delays in center response times, which limit the number of

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[24]Nearly 30% of recovered kidneys are discarded each year (McKenney et al., 2024).
[25]I use the proportion of patients from the same transplant center as weights.

subsequent patient offers. However, using time-stamp data, I find no evidence that centers take longer to respond to high-risk kidneys. Section 6.1 also finds no evidence that centers set more stringent donor criteria for their patients. These findings suggest that center behavior is not the primary driver of kidney discard, pointing to other inefficiencies in the allocation process.

8 Discussion and Conclusion

This paper examines one of the first large-scale federal oversight programs in the U.S. deceased donor kidney transplant setting. The CoP announcement incentivized transplant centers to reduce post-transplant mortality rates, leading to a 19% 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 strategies to address acute kidney rejection. The findings indicate that centers prioritized diligent detection of rejection over prevention or treatment, reflecting a shift towards early diagnosis.

While the results highlight inefficiencies in kidney utilization, 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. First, further investigation is needed to understand the mechanisms behind the increased discard of high-risk kidneys

following the CoP announcement. This is critical given the kidney shortage and the significant number of patients on the transplant waiting list. Second, 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 Model setup and Predictions

The center chooses its transplant threshold t and quality of care $q(x)$ to maximize its expected payoff:

$$\max_{t,q(x)} \int_t^\infty \rho\left[\pi + \alpha q(x)\right] + (1-\rho)\left[xq(x) - \frac{\gamma}{2}q(x)^2\right] \partial F(x)$$

$$(8)$$

$$\text{s.t. } \rho \int_\tau^\infty P(x, q(x)) \partial F(x) \leq \tau$$

Where $P(x, q(x)) = 1 - \Phi\left(\frac{q(x)+x}{\sigma}\right) = \Phi\left(\frac{-x-q(x)}{\sigma}\right)$ and $\Phi(.)$ is the standard normal cdf.

I introduce a Lagrange multiplier $\lambda \geq 0$ for the constraint. The Lagrangian is:

$$\mathcal{L} = \int_t^\infty \left(\rho[\pi + \alpha q(x)] + (1-\rho)\left[xq(x) - \frac{\gamma}{2}q(x)^2\right]\right) dF(x)$$
$$+ \lambda\left[\tau - \rho \int_t^\infty \Phi\left(\frac{-x - q(x)}{\sigma}\right) dF(x)\right].$$

A.1 Optimal $q(x)$

Differentiating w.r.t. $q(x)$:

$$\frac{\partial \mathcal{L}}{\partial q(x)} = (\rho\alpha + (1-\rho)\left[x - \gamma q(x)\right])f(x) - \lambda\rho f(x)\frac{d}{dq(x)}\Phi\left(\frac{-x - q(x)}{\sigma}\right).$$

Since

$$\frac{d}{dq(x)}\Phi\left(\frac{-x - q(x)}{\sigma}\right) = \phi\left(\frac{-x - q(x)}{\sigma}\right)\left(-\frac{1}{\sigma}\right),$$

we have:

$$\frac{\partial \mathcal{L}}{\partial q(x)} = (\rho\alpha + (1-\rho)\left[x - \gamma q(x)\right])f(x) + \lambda\rho f(x)\frac{\phi((-x - q(x))/\sigma)}{\sigma}.$$

44

Set this equal to zero for optimality:

$$\rho\alpha + (1-\rho)\left[x - \gamma q(x)\right] = -\frac{\lambda\rho}{\sigma}\phi\left(\frac{-x - q(x)}{\sigma}\right).$$

If the constraint is not binding ($\lambda = 0$), we get the unconstrained solution:

$$q(x) = \frac{x}{\gamma} + \frac{\rho}{1-\rho}\left[\frac{\alpha}{\gamma}\right]$$

If $\lambda > 0$, $q(x)$ is determined implicitly by:

$$\gamma q(x) = x + \frac{\rho}{1-\rho}\left[\alpha + \frac{\lambda}{\sigma}\phi\left(\frac{-x - q(x)}{\sigma}\right)\right] \tag{9}$$

Note that when the constraint binds, the presence of the λ term generally lowers $q(x)$ relative to the unconstrained optimum.

A.2 Optimal τ

Now consider the FOC with respect to t. Differentiating $\mathcal{L}$ with respect to t requires applying Leibniz rules to the integrals.

$$\frac{\partial\mathcal{L}}{\partial t} = \left(\rho\left[\pi + \alpha q(t)\right] + (1-\rho)\left[tq(t) - \tfrac{\gamma}{2}q(t)^2\right]\right)f(t) - \lambda\rho\left[\Phi\left(\frac{-t - q(t)}{\sigma}\right)f(t)\right]$$

Set this equal to zero for optimality:

$$\rho\left[\pi + \alpha q(t)\right] + (1-\rho)\left[tq(t) - \frac{\gamma}{2}q(t)^2\right] = \lambda\Phi\left(\frac{-t - q(t)}{\sigma}\right) \tag{10}$$

A.3 Complementary Slackness

The constraint:

$$\rho \int_t^\infty \Phi\left(\frac{-x - q(x)}{\sigma}\right) dF(x) \le \tau$$

is associated with the multiplier λ. Complementary slackness implies:

$$\lambda\left(\rho \int_\tau^\infty \Phi\left(\frac{-x - q(x)}{\sigma}\right) f(x)\, dx - \tau\right) = 0.$$

- If the constraint is not binding, $\lambda = 0$ and:

$$q(x) = \frac{x}{\gamma} + \frac{\rho}{1-\rho}\left[\frac{\alpha}{\gamma}\right]$$

The optimal t would then be determined by a marginal condition on profits if such an interior solution exists.

- If the constraint binds, $\lambda > 0$ and:

$$\rho \int_t^\infty P(x, q(x)) f(x)\, dx = \tau.$$

Combined with:

$$\gamma q(x) = x + \frac{\rho}{1-\rho}\left[\alpha + \frac{\lambda}{\sigma}\phi\left(\frac{-x - q(x)}{\sigma}\right)\right], \quad \rho[\pi + \alpha q(t)] + (1-\rho)\left[tq(t) - \frac{\gamma}{2}q(t)^2\right] = \lambda\Phi\left(\frac{-t - q(t)}{\sigma}\right)$$

and the integral equation, these determine λ, t, and the function $q(x)$. The solution is characterized by the system of equations derived from the first-order conditions.

A.4 Comparative statics

As τ increases, $\int_t^\infty \Phi\left(\frac{-x - q(x)}{\sigma}\right) dF(x)$ can be larger without violating the constraint. A looser constraint implies a lower λ. Since $q(x)$ is increasing in λ in equation 9, $q(x)$ decreases

as λ decreases $\left(\frac{dq(x)}{d\tau} < 0\right)$

Centers only transplant a patient if $\rho\left[\pi + \alpha q(x)\right] + (1 - \rho)\left[xq(x) - \frac{\gamma}{2}q(x)^2\right] > 0$. The integrand is positive for all $x \leq t$. Thus, as τ increases, the constraint relaxes, making it easier to expand t. With a less binding constraint, there is more incentive to include less healthier patients and increase the center's utility, thus t decreases $\left(\frac{dt}{d\tau} < 0\right)$.

A.5   Decomposing death rate

Applying *Leibniz* rule and differentiating the center's total death rate by τ, we have:

$$\frac{\partial}{\partial\tau}\left[\int_{t(\tau)}^{\infty} P(x, q(x, \tau))dF(x)\right]$$

$$= -P(t, q(t, \tau))f(t)\frac{\partial t}{\partial\tau} + \int_{t(\tau)}^{\infty}\frac{\partial}{\partial\tau}\left[P(x, q(x, \tau))f(x)\right]dx$$

$$= -P(t, q(t, \tau))f(t)\frac{\partial t}{\partial\tau} \tag{11}$$

$$+ \int_{t(\tau)}^{\infty}\left[\frac{\partial P}{\partial x}\frac{\partial x}{\partial\tau}f(x) + \frac{\partial P}{\partial q}\left[\frac{\partial q}{\partial x}\frac{\partial x}{\partial\tau} + \frac{\partial q}{\partial\tau}\right]f(x) + \frac{\partial f}{\partial x}\frac{\partial x}{\partial\tau}P(x, q(x, \tau))\right]dx$$

$$= -P(t, q(t, \tau))f(t)\frac{\partial t}{\partial\tau} + \int_{t(\tau)}^{\infty}\frac{\partial P}{\partial q}\frac{\partial q}{\partial\tau}f(x)dx$$

$\frac{\partial x}{\partial\tau} = 0$. Thus, we obtain:

$$\frac{\partial}{\partial\tau}\left[\int_{t(\tau)}^{\infty} P(x, q(x, \tau))dF(x)\right] = \underbrace{\overbrace{-P(t, q(t, \tau))}^{<0} f(t)\overbrace{\frac{\partial t}{\partial\tau}}^{<0}}_{\text{Selection Channel}} + \underbrace{\int_{t(\tau)}^{\infty}\overbrace{\frac{\partial P}{\partial q}}^{<0}\overbrace{\frac{\partial q}{\partial\tau}}^{<0}f(x)dx}_{\text{Quality Channel}} > 0 \tag{12}$$
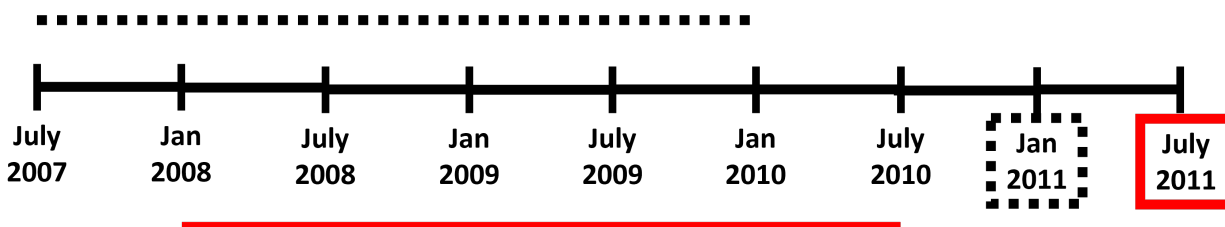
47

B   Supplementary Figures

Figure B.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 B.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., $Pr(O = E) = 0.469 > 0.05$).
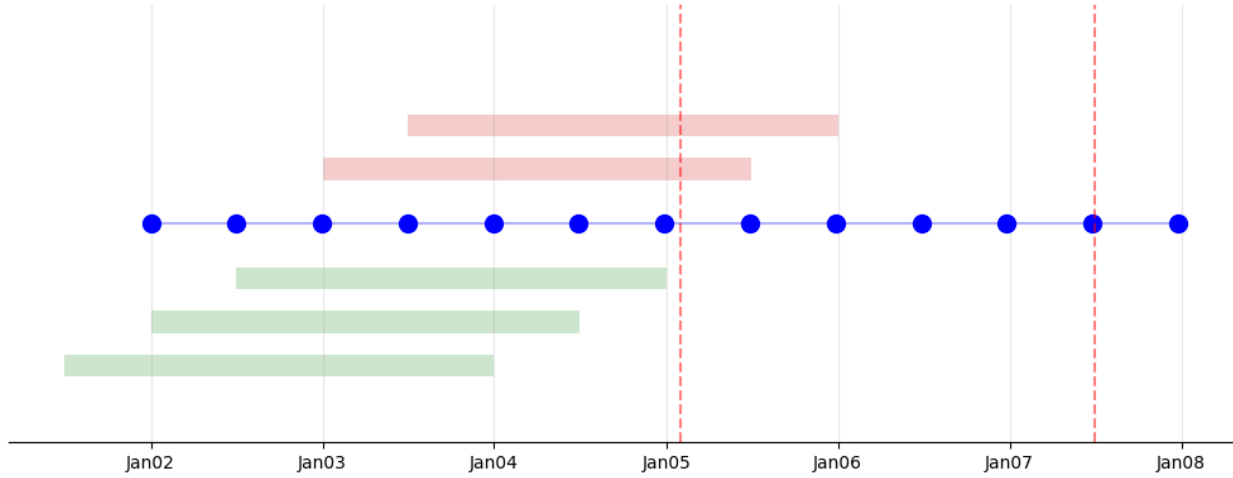
Figure B.3: CoP reports and their 2.5-years rolling cohorts

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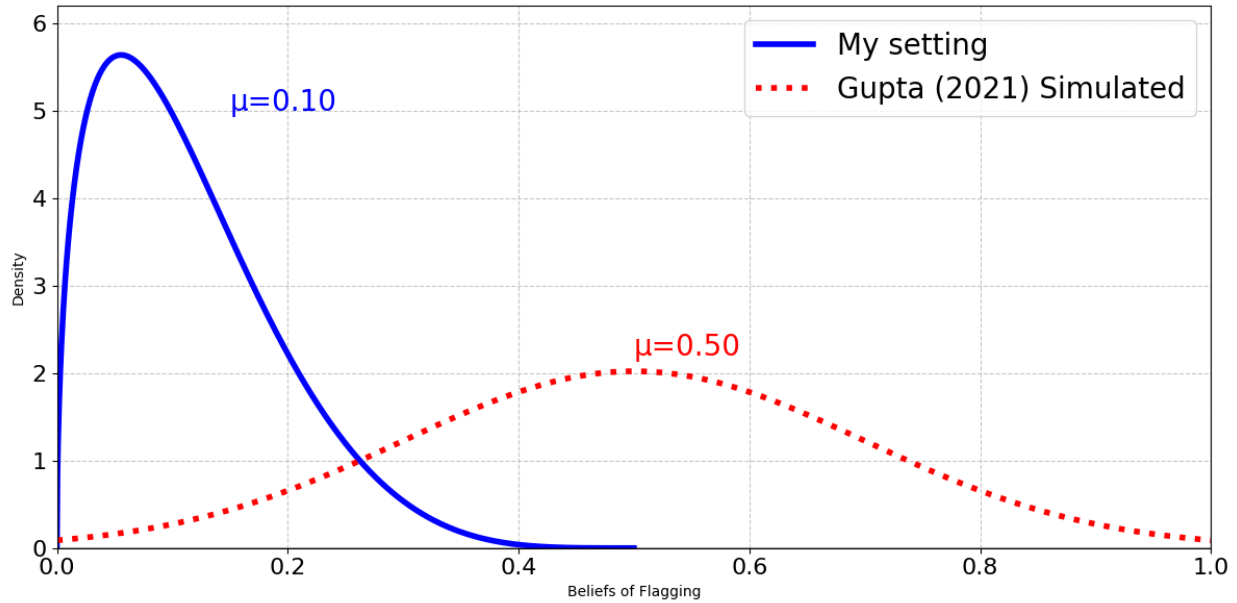
Figure B.4: Density of actual flagging beliefs in my setting versus simulated beliefs in Gupta (2021)

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 $[0, 1]$
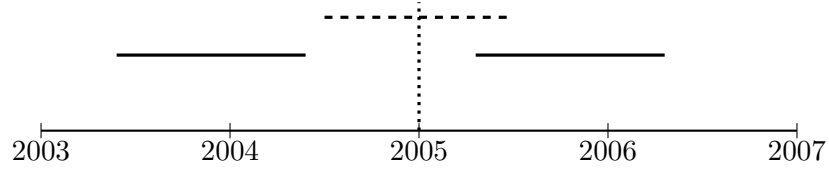
Figure B.5: Non-overlapping (solid) and overlapping (dash) patients

Note: This figure highlights my regression subsample in Sections 5 and 6. 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 who do have dashed lines are excluded from my analysis.

C Supplementary Tables

Table C.1: Impact on different post-transplant timeline mortality

	Post-transplant ≤ 1-year			Post-transplant > 1-year	
	(1)	(2)	(3)	(4)	(5)
	14-days	6-months	1-year	2-years	3-years
DiD estimates	-0.01101***	-0.02380***	-0.02371***	-0.00696	0.01377
	(0.00283)	(0.00457)	(0.00555)	(0.00736)	(0.01062)
Y mean	0.03175	0.08555	0.12496	0.19266	0.25765
F-statistic	28775.11307	28622.21959	25811.09616	17825.83114	8009.39088
Fixed Effects	Center, 6-months	Center, 6-months	Center, 6-months	Center, 6-months	Center, 6-months
Observations	64305	59944	55432	46978	38674

Note: This table relates to the analysis in Section 5.1. 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 4.3. I use robust standard errors.

Table C.2: Impact on the prescription of maintenance immunosuppressants

	(1)	(2)	(3)	(4)
	6-months	1-year	2-years	3-years
DiD estimates	0.00477	-0.00507	0.02293***	-0.00098
	(0.00425)	(0.00447)	(0.00668)	(0.01114)
Y mean	0.51526	0.57224	0.50875	0.54521
F-statistic	28128.49544	25524.97166	17564.19735	7910.55895
Fixed Effects	Center, 6-months	Center, 6-months	Center, 6-months	Center, 6-months
Observations	59332	54872	46496	38299

Note: This table relates to the analysis in Section 5.2.1. It presents the estimated effect on the probability of prescribing maintenance immunosuppression medicines 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 4.3. I use robust standard errors.

Table C.3: Impact on the rate of acute kidney rejection

	(1) 6-months	(2) 1-year	(3) 2-years	(4) 3-years
DiD estimates	0.01328***	0.01257***	0.01039***	0.00495
	(0.00304)	(0.00258)	(0.00278)	(0.00268)
Y mean	0.03816	0.02156	0.02011	0.01661
F-statistic	28128.49544	25524.97166	17564.19735	7910.55895
Fixed Effects	Center, 6-months	Center, 6-months	Center, 6-months	Center, 6-months
Observations	59332	54872	46496	38299

Note: This table relates to the analysis in Section 5.2.2. 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 4.3. I use robust standard errors.

Table C.4: Impact on the rate of CMV testing

	(1) 6-months	(2) 1-year	(3) 2-years	(4) 3-years
DiD estimates	0.02272***	0.03072***	0.02424***	0.00176*
	(0.00463)	(0.00523)	(0.00560)	(0.00083)
Y mean	0.13917	0.16871	0.20829	0.00056
F-statistic	28128.49544	25524.97166	17564.19735	7910.55895
Fixed Effects	Center, 6-months	Center, 6-months	Center, 6-months	Center, 6-months
Observations	59332	54872	46496	38299

Note: This table examines the proposed detection channel in section 5.2.2. 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 4.3. I use robust standard errors.

Table C.5: Impact on different health indicators during 1-year follow-up

	(1) Creatinine	(2) Dialysis	(3) Diabetes
DiD estimates	-0.01643	-0.00099	0.00266
	(0.01067)	(0.00160)	(0.00452)
Y mean	1.52505	0.01091	0.10790
F-statistic	23611.24276	28128.49544	28128.49544
Fixed Effects	Center, 6-months	Center, 6-months	Center, 6-months
Observations	52647	59332	59332

Note: This table examines the proposed matching channel in section 5.2.2. It presents the estimated effect on various health indicators, 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 4.3. I use robust standard errors.

Table C.6: Impact on hospitalizations for patients diagnosed with rejections

	(1) 6-months	(2) 1-year	(3) 2-years	(4) 3-years
DiD estimates	0.03676	0.03994	0.10253	0.49081**
	(0.03412)	(0.03813)	(0.06114)	(0.18141)
Y mean	0.55176	0.37875	0.30428	0.67481
F-statistic	936.58269	788.62918	139.61306	26.89142
Fixed Effects	Center, 6-months	Center, 6-months	Center, 6-months	Center, 6-months
Observations	5904	4727	4405	639

Note: This table relates to the analysis in section 5.2.3. It presents the estimated effect on the probability of centers hospitalizing patients diagnosed with rejections 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 4.3. I use robust standard errors.

Table C.7: Impact on immunosuppressant prescription for patients diagnosed with rejections

	(1) 6-months	(2) 1-year	(3) 2-years	(4) 3-years
DiD estimates	0.01333	-0.00605	0.07472	-0.01481
	(0.01598)	(0.01530)	(0.04982)	(0.03823)
Y mean	0.68378	0.84490	0.73718	0.97710
F-statistic	936.58269	788.62918	139.61306	26.89142
Fixed Effects	Center, 6-months	Center, 6-months	Center, 6-months	Center, 6-months
Observations	5904	4727	4405	639

Note: This table relates to the analysis in section 5.2.3. It presents the estimated effect on the probability of immunosuppressant prescription for patients diagnosed with rejections 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 4.3. I use robust standard errors.

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

	(1) Min. Age	(2) Max. Age	(3) HLA Mismatch	(4) Creatinine
DiD estimates	-0.00195	0.02120	0.00406	-0.93453***
	(0.00355)	(0.02061)	(0.00363)	(0.10013)
Y mean	1.03382	85.92787	5.94825	39.03037
Fixed Effects	Patient, Quarters	Patient, Quarters	Patient, Quarters	Patient, Quarters
Observations	38777	38777	38040	38777

Note: This table relates to the analysis in section 6.1. It presents the estimated effect on centers setting more stringent donor criteria, obtained by estimating equation 5. I use a patient-fixed effects regression here and replace center-fixed effects, α_c, with patient-fixed effects, α_i. I use robust standard errors.

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

	(1)	(2)	(3)	(4)
	Outside DSA	Cold Ischemic Time	Warm Ischemic Time	Expanded Criteria
DiD estimates	0.00004	-2.65025***	-0.65335***	0.00491*
	(0.00013)	(0.25749)	(0.08736)	(0.00220)
Y mean	0.99977	98.90750	66.15076	0.42237
Fixed Effects	Patient, Quarters	Patient, Quarters	Patient, Quarters	Patient, Quarters
Observations	38777	38777	24524	38777

Note: This table relates to the analysis in section 6.1. It presents the estimated effect on centers setting more stringent donor criteria, obtained by estimating equation 5. I use a patient-fixed effects regression here and replace center-fixed effects, α_c, with patient-fixed effects, α_i. I use robust standard errors.

Table C.10: Impact on admitted patient characteristics (i)

	(1)	(2)	(3)	(4)	(5)
	Age	White	No education	Working	Medicare
DiD estimates	-0.13499	-0.00688*	0.00027	-0.00753**	0.01064**
	(0.10543)	(0.00332)	(0.00245)	(0.00272)	(0.00363)
Y mean	49.19511	0.48571	0.15769	0.18320	0.47689
Fixed Effects	Centers, 6-months	Centers, 6-months	Centers, 6-months	Centers, 6-months	Centers, 6-months
Observations	148104	148104	148104	148104	148104

Note: This table relates to the analysis in section 6.3. It presents the estimated effect on admitted patient characteristics, obtained by estimating equation 5. I use robust standard errors.

Table C.11: Impact on admitted patient characteristics (ii)

	(1)	(2)	(3)	(4)
	BMI	Diabetic	On dialysis	Blood type O
DiD estimates	-0.12782**	-0.00088	-0.00158	0.34568
	(0.04461)	(0.00365)	(0.00295)	(0.37660)
Y mean	27.59877	0.39058	0.76605	48.63407
Fixed Effects	Centers, 6-months	Centers, 6-months	Centers, 6-months	Centers, 6-months
Observations	144159	148104	148104	148104

Note: This table relates to the analysis in section 6.3. It presents the estimated effect on admitted patient characteristics, obtained by estimating equation 5. I use robust standard errors.

Table C.12: Impact on patient waitlist experience

	Time on Waitlist		Likelihood of Dying off Waitlist
	(1)	(2)	(3)
	Baseline DiD	Triple DiD	Baseline DiD
DiD estimates	0.11736	-0.29467	0.06200
	(0.15195)	(0.28071)	(0.03459)
Triple DiD estimates		0.46485	
		(0.32699)	
Y mean	3.73455	3.73455	0.08451
Fixed Effects	Center, 6-months	Center, 6-months	Center, 6-months
Observations	127434	127434	127439

Note: This table relates to the analysis in section 7.1. It presents the estimated effect on transplant wait time and waitlist mortality, obtained by estimating equation 5. I use robust standard errors.

Table C.13: Impact on kidney discard

	Kidney Discards		No. of Patients Offered		Minutes for centers to reply	
	(1)	(2)	(3)	(4)	(5)	(6)
	Baseline	Risky Kidney	Baseline	Risky Kidney	Baseline	Risky Kidney
DiD estimates	0.00210	0.02882***	19.87703***	55.00605**	-6.47184	20.43389
	(0.00580)	(0.00777)	(4.93789)	(20.11251)	(18.94257)	(24.80549)
Triple DiD estimates		0.02715***		-49.99623**		-63.22164
		(0.00716)		(17.81905)		(37.23333)
Y mean	0.15097	0.15097	182.67227	182.67227	388.87112	388.87112
Fixed Effects	DSA, 6-months	DSA, 6-months	DSA, 6-months	DSA, 6-months	DSA, 6-months	DSA, 6-months
Observations	42987	42987	42987	42987	42987	42987

Note: This table relates to the analysis in section 7.2. It presents the estimated effect on kidney discard, obtained by estimating equation 7. I use robust standard errors.