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A Time-Series Analysis of U.S. Kidney Transplantation and the Waiting List: Donor Substitution Effects and "Dirty Altruism"

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A Time-Series Analysis of U.S. Kidney Transplantation and the Waiting List: Donor Substitution Effects and "Dirty Altruism"

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Abstract

This paper provides an econometric analysis of the relationship between live and deceased (cadaveric) kidney donations for the United States for the period 1992:IV through 2006:II. We find strong evidence for deceased donor kidneys "crowding out" living donations, potentially undermining conventional efforts to reduce the shortage. We also find evidence for the "dirty altruism" hypothesis of Osterkamp (2006), in which heavy reliance on living donors undermines cadaveric donor transplants.

Keywords: Kidney Transplantations, Donor Substitution Effects, Dirty Altruism, Cointegration, Vector Error Correction Model

JEL Specification: I18, I19

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1 Introduction

The medical miracles represented by organ transplantation and immune-suppression therapy have saved hundreds of thousands of lives over the last thirty years.[1] While many patients have enjoyed life-altering improvements in health and longevity, hundreds of thousands more have been unable to take advantage of these procedures due to a chronic shortage of organs for transplantation. While most organs exhibit some degree of shortage, the case of kidneys is by far the most severe in the U.S. and abroad. Public funding of hemodialysis treatment for virtually all patients suffering from End-Stage Renal Disease (ESRD) in the U.S., Europe, and in most other high-income countries has allowed the waiting-lists for kidney transplants to grow year after year.[2] As of March, 2010, the official U.S. kidney waiting list, maintained by the Organ Procurement and Transplantation Network (OPTN), had reached about $84,100$ patients, while an additional 2,200 were awaiting kidney-pancreas transplants.[3] These figures vastly understate the significance of the shortage, however; $6,417$ patients were removed from the official list in 2009 due to reasons of death $(4,476)$ or deteriorating health $(1,941)$. Beard, Jackson, and Kaserman (2008) estimate that cumulative deaths on the U.S. list alone, since 1982, will exceed deaths from the Hiroshima bomb of WWII by 2010.[4] Further, dialysis itself is incredibly expensive, running around $72,000 per year in direct care costs per patient, and over 300,000 patients are currently receiving dialysis therapy under the Medicare program in the U.S. Long-term dialysis care has numerous severe medical consequences, and dialysis reduces the patient's prospects for a successful transplant.[5]

The organ procurement systems of virtually all countries share a few common features which have resulted in the current severe shortage of organs.[6] First, almost all nations prohibit compensation for organ donors, whether those donors are living (as can be done in the case of kidneys, for example), or

[1]Extensive reviews of the U.S. experience are available in Kaserman and Barnett (2002) and Goodwin (2006).

[2]Most countries have national authorities that maintain the "official" waiting list statistics. In the U.S., OPTN does this task. Supernational organizations such as Eurotransplant and Scandiatransplant maintain waiting lists that combine different national populations. OECD health statistics include census counts of dialysis populations and kidney transplantation. Data on U.S. dialysis programs is compiled by the United States Renal Disease System (USRDS).

[3]There are many more kidney dialysis patients than there are patients on the kidney transplant waiting list. This is because admission to the waiting list requires a variety of medical and social criteria be met. However, the waiting list itself does reflect, to some extent, the severity of the shortage, although on medical grounds it clearly understates it.

[4]U.S. deaths on the list vastly understate deaths from the shortage due to sickness removals.

[5]Steinbuch (2009) provides a concise summary of the main negative consequences of dialysis.

[6]Iran pays living donors under a state-administered system, and it has no waiting list for kidneys. See Ghods (2002).

deceased ("cadaveric"). Second, living donors of kidneys (who surpassed cadaver donors for the first time in the U.S. in 2001) are generally required to have a long-term, private relationship with the recipient. Donations "to the waiting list" are prohibited, or else made quite difficult. In contrast, donations from cadavers must be "anonymous", and the families of deceased donors are not allowed to decide who can receive harvested organs. Neither living nor deceased donors, nor their families, can receive compensation, and there is some evidence that living donors in the U.S. are not even fully compensated for their direct costs.[7]

The evolution of the kidney transplant waiting list, and the complex roles played by living and deceased donors in this process, is of great public interest. In broad terms, the waiting list for renal grafts is composed of individuals admitted to the list by transplant centers using both medical and non-medical criteria. Not all ESRD patients are viable candidates for transplantation.[8] Kidneys for transplant are obtained from deceased donors who meet relatively strict medical criteria. Such removals require family consent, and that consent is often not given.[9] Alternately, living kidney donation is feasible and low-risk, although it is generally necessary for patients to find a willing and compatible donor from among their family and closest friends.[10] The waiting list is primarily composed of persons waiting for a cadaveric organ from a stranger, since most patients needing kidney transplants are urged to find a willing living donor at their initial diagnosis.

Many programs have been implemented over the years in an attempt to increase the numbers of transplants performed.[11] Since the families of brain-dead potential donors cannot legally be offered compensation for donation, they must be persuaded by other means. Similarly, potential living donors, when faced with the critical medical need of a relative or close acquaintance, must decide whether the personal costs of donation are outweighed by the benefits.[12] Because these decisions are, in principle,

[7] See Reilly *et al.* (1997) and Clarke *et al.* (2006).

[8] Estimates vary, but perhaps about 40% of dialysis patients in the U.S. are, on purely medical grounds, viable candidates. This number exceeds the waiting list by a substantial amount. See Barnett and Kaserman (2002), Ch. 2, for a discussion of the demand and supply for transplants.

[9] In the U.S., the "conversion rate" hovers around 50%. See Matas (2004).

[10] See Matas (2004) for statistics on living donation. Around 98% of donors give to relatives, close friends, or do so indirectly through paired exchanges.

[11] Beard and Kaserman (2006) provide a list. These innovations include mandated request, public service advertising, donor cards, paired exchange systems, presumed consent laws, and so on.

[12] It is quite likely that many living donors face "nonmarket" incentives to donate, and payments made within the family setting are quite beyond the ability of the law to regulate.

non-monetary, they are likely affected by considerations related to the prospects of the patients involved, the degree of the shortage, and the extent to which the potential donor decision-makers view the current system as honest and fair.

Our goal here is to examine two phenomena, discussed by those in the transplant community, which are potentially important for any reform effort within the current altruistic paradigm. First, because living donor and deceased donor kidneys are fairly good (but not perfect) substitutes, the question naturally arises as to whether increases in the cadaveric supply will displace living donor organs and, if so, to what degree?

A second issue, only recently introduced into the literature on the organ shortage by German economist Rigmar Osterkamp, refers to the effects of living donations on the willingness of bereaved families to allow deceased donations.[13] Numerous press reports in the U.S. and abroad have described illegal "kidney deals", in which living donors are recruited from poor countries and paid to masquerade as willing, altruistic relatives. The existence of such schemes is believed to undermine the efforts of legitimate national organ procurement agencies. Families of potential deceased donors come to believe that they are asked to make a gift, while other donors are paid huge sums to provide organs for wealthy patients. Osterkamp termed the resulting negative effect on cadaveric donation "dirty altruism."

In this paper, we analyze time series data for U.S. kidney transplantation activities and the kidney waiting list to isolate and measure these two effects. We are particularly interested in the substitution effect between living and cadaver donor transplants, because their existence would affect the likelihood of relieving the shortage using increased numbers of cadaver donors. In particular, the Organ Donation Breakthrough Collaborative (ODBC), a recent U.S. effort aimed at increasing the success rate for obtaining deceased donor organs, presupposes that an additional deceased donor kidney transplant is simply a one unit net increase in the number of transplants performed.[14] However, if such an increase leads to a significant reduction in living donor transplant levels, the ability of the ODBC to reduce the shortage will be undermined to some degree. Similarly, the existence of dirty altruism would also present serious

[13] Osterkamp (2006) introduces this concept, and relates it to the donor analysis of Barnett, Beard, and Kaserman (1993). Osterkamp remarks, "However, there seems to be a growing awareness in the medical community, at least in Germany, that living donation may have only a small or even adverse net effect on total donation." (Osterkamp, p.3, note 1)

[14] This conclusion is implicit in the discussion, but this sentiment is more explicit in other, related discussions, such as Gjertson and Cecka (2000).

constraints on reform efforts, since the percentage of patients receiving living donor transplants has grown enormously throughout the world as the shortages have worsened in most countries.

This paper is organized as follows. Section 2 provides a concise background description. Section 3 presents our econometric model and estimation results. A concluding section describes the import of the results for organ supply reform efforts, and suggests further issues.

2 Background: Kidney Procurement and Transplantation in the U.S.

About eighteen patients die each day in the U.S. as a direct result of the shortage of kidneys for transplantation.[15] Hundreds of thousands endure hemodialysis, spending typically 14-20 hours per week hooked up to dialysis machines, suffering the aftereffects of dialysis therapy, or remaining at home, unable to work due to hospitalization.[16] Over twenty billion dollars are spent annually by the U.S. federal authorities on direct payments for dialysis treatment, transplantation, and related activities (Health & Human Services, 2008). Numerous studies have shown that a kidney transplant is the best treatment for many patients with ESRD, and that such transplants are cost-effective for public health budgets, often "paying for themselves" in as little as 9-18 months.[17] No one argues that a large increase in the rate of renal transplantation is undesirable: debate focuses solely on the means, and whether donor compensation should be used.

In the U.S., the legal procurement of organs for transplantation is a public function. The United Network for Organ Sharing (UNOS) operates the Organ Procurement and Transplantation Network (OPTN), created by Congress in the National Organ Transplant Act (NOTA) of 1984. Regional Organ Procurement Organizations (OPOs) have monopoly rights to collect organs within their regions. UNOS operates as a central clearinghouse, using a complicated algorithm to match donated organs with those seeking transplants. Unlike many European countries, OPOs and large transplant centers in the U.S. have some autonomy, and control admissions to the waiting lists at their facilities. In principle, organs

[15] This is probably a large underestimate since very sick persons are never even placed on the list. As mentioned earlier, though, there were about 6,238 removals from the U.S. Kidney list in 2008 dues to death or sickness. There were many more deaths of dialysis patients, but of course not all of those persons would be valid candidates for transplantation.

[16] A vivid description of the lives of dialysis patients is in Perez-Pena (2006).

[17] See Karlberg and Nyberg (2006) for a review.

are matched to patients primarily on medical criteria, although time on the waiting list is also considered. Any hospital wishing to perform transplants and receive Medicare or Medicaid funding must comply with UNOS regulations.

Kidneys constitute the primary solid organ transplanted. About 80% of all patients on the U.S. waiting lists are ESRD patients needing kidney graphs.[18] Kidneys are unique among organs transplanted for two primary reasons. First, living donation is possible, since people ordinarily have two kidneys, but fare fairly well with only one. Surgery to remove a healthy kidney is relatively safe.[19] Second, the dialyzer, a machine that mimics the function of the kidney, exists and allows ESRD patients to survive for years while awaiting transplant. In the cases of many other solid organs, such as hearts, livers, or lungs, patient survival expectations on the waiting lists are quite short, and therefore the waiting lists for those organs are only a fraction of that for kidneys. Thus, the shortage of kidneys can be regarded as the defining problem for organ transplantation policy.

Although poorly understood by many people, very few hospital deaths (generally perhaps 1% or less) occur in conditions that allow transplantation of solid organs. In general, the donor must be "brain-dead", a physiological state characterized by the cessation of higher brain activity. The donor must be generally healthy, free of infections, less than 60 years of age, and so on. As a result of these traditional requirements, most such donors are victims of suicide, car/motorcycle wrecks, or strokes. Persons dying of heart attacks, for example, are generally not good sources of organs, and few are used.[20] Many families (around 50%) refuse donation, even in the presence of a donor card, trained requesters, physician support, and the terrible need for organs.[21] Efforts by hospital staff can affect the success of donation requests, and thus the numbers of deceased donor transplants.

Transplants from living donors are medically fairly simple. Transplant success rates are higher,

[18] As of September, 2009, OPTN reported total waiting list of 103,247, or which 80,975 needed kidneys, 15,928 were waiting for livers, and the balance needed other organs or multiple organ transplants.

[19] See Ibrahim *et al.* (2009).

[20] The "best" donors are termed "standard criteria donors" (SCD's), while those donating after cardiac death (non-heart beating donors, or NHB's) or with other undesirable traits such as infection or advanced age (so-called "expanded criteria donors", or ECD's) are associated with poorer medical outcomes and higher rates of rejection and infection. Rudich *et al.* (2002) provide statistics on the consequences of using NHB donors. In 2005, the U.S. UNOS hospitals transplanted 5294 SCD kidneys, 2000 ECD organs, and 793 from NHB donors.

[21] Some hospitals have conversions rates around 10%, while others achieve rates over 80%. These variations are the basis for the ODBC program of sharing "best practices" among member hospitals. See Shafer *et al.* (2006).

ischemic time is extremely short, and the expected lifetime of the graft is greater, when compared to deceased donor transplants. Surgeons, from the purely technical viewpoint, always prefer living donor transplants. The problem, of course, is that, in contrast to a cadaver, the donor in the living case is a second patient, needing care, post-operative evaluation, and so on. Such a donor has rights unlike a cadaver.[22]

Living kidney donation is a highly significant and irreversible act. Although such donation is low risk (statistics suggest a death rate of less than .05%), there are consequences. First, surgical evaluation, recovery, and so on takes weeks or months. The law prohibits compensation, even for costs such as these. General anesthesia is used. After surgery, the donor has only one kidney, which prohibits them from joining the military, playing NCAA contact sports, buying certain forms of insurance, and so on.[23] Long-term follow-up studies of donors have thus far found no deleterious effects from donation, but some physicians express concerns. Finally, living donation requires a physician to remove a healthy organ from a healthy patient, which is not a treatment for any disease, and is sometimes claimed to be mutilation in violation of the Hippocratic Oath.

Properly speaking, kidney transplants of either type involve a combination of stochastic events, combined with choices by both patients and medical officials. Accidents provide deceased donor organs. Fortunate genetic matching makes living donation possible. Yet, combined with these events, we also observe conscious choices made by patients, families, medical staff, and various public officials. Given the "correct" stochastic events, donors (or their families) must make the next, necessary step and agree to the donation. Since payment is prohibited, the decision calculus is likely to include factors that, in a monetized system, would be much less noticeable. In the case of the families of deceased potential donors, the urgency of the need for organs (which can be communicated to them), and the degree to which they believe the organ procurement system is fair and humane, may affect their decision. The efforts of doctors, although complicated by financial interests, also reflect such motives. The term "dirty altruism" has been coined to describe the effect that suspect living donations might have on the willing-

[22] The Common Law provides for the protection of cadavers from abuse and mutilation. For a legal treatment, see Goodwin (2006) and Steinbuch (2009).

[23] The system in Iran, where paid living donation is the rule, provides for insurance benefits and care for donors precisely due to these sorts of effects. See Ghods (2002).

ness of families to provide cadaveric organs. Sensational press accounts of the sale of kidneys by living donors, which have become a staple of the popular media show that some living donors receive very large cash payments.

The problem facing the potential living donor is more difficult. Such a person may well feel pressure from family members and friends. If a donor cares about the patient, then a relevant question would be, "if I do not donate, what is the likely fate of the patient?". The answer to this question depends primarily on the probability that a cadaver organ will become available. That, in turn, depends on the numbers of cadaver transplants and the size of the waiting list.

Thus, we describe two phenomena that may affect the evolution of the kidney waiting list and organ donations: the "substitution effect", and the "dirty altruism" effect. In the substitution case, one would expect to see a negative impact of cadaver transplants on living donations, controlling for the "need" for transplants, as proxied by the waiting list. The size of this effect would be important, since it would determine the degree to which increases in cadaver organs will translated into more total transplants over time.[24]

For the "dirty altruism" effect, one envisions a positive innovation in living donations reducing cadaver donations. Following Osterkamp (2006), this effect is assumed to arise due to the suspicions of potential deceased-donor families that some proportion of living donors are, in fact, paid for their organs. Extensive coverage of such illegal "kidney deals" in the popular media bolster such a conjecture. Because the temporal structures of these phenomena are under *ex ante*, we adopt an approach based on cointegration in what follows.

3 The Econometric Model

Let $\mathbf{y}_t = [l_t \;\; c_t \;\; w_t]'$ be a vector of difference stationary random variables where l_t, c_t, and w_t denote the number of live donor kidney transplants, the number of deceased donor kidney transplants, and the waiting list for kidney transplants at time t, respectively, measured in natural logarithms.

Assume that there is a nonzero vector of real numbers $\gamma = [1 \;\; -\beta']'$, where $\beta = [\beta_1 \;\; \beta_2]'$, such that $\gamma'\mathbf{y}_t$

[24] Abadie and Gay (2006) remark, "$\cdots$ it seems likely that an increase in the supply of cadaveric organs would be followed by a reduction in the supply of organs from living donors." (p.612)

is stationary, that is, $\mathbf{y}_t$ is cointegrated with a (normalized) cointegrating vector γ. Then, the triangular representation (Phillips, 1991) of such a cointegrated vector process is,

$$l_t = \alpha + \beta_1 c_t + \beta_2 w_t + \varepsilon_t \tag{1}$$

$$\Delta \mathbf{x}_t = \delta + \mathbf{u}_t, \tag{2}$$

where $\Delta \mathbf{x}_t = [\Delta c_t \ \ \Delta w_t]'$ is a 2×1 vector of differenced variables, α denotes a constant, δ is a vector of constants (drifts), ε_t is zero-mean stationary for β_1 and β_2, and 2×1 vector $\mathbf{u}_t$ is zero-mean stationary. We assume that the cointegrating vector γ eliminates both the stochastic and deterministic trends, thus a time trend is not included in the cointegrating regression (1).[25]

The ordinary least squares (LS) estimator $\hat{\beta}_{LS}$ for the cointegrating regression (1) is super-consistent as $\hat{\beta}_{LS}$ converges to the true value at the rate of T (sample size) even when $\mathbf{x}_t$ is correlated with ε_t. However, $\hat{\beta}_{LS}$ is asymptotically biased and inefficient, and its asymptotic distribution is non-normal.[26] Consequently, statistical inference based on the usual LS standard errors is not reliable. Fortunately, there is an array of alternative methods, such as Johansen's (1988) Maximum Likelihood (ML) Estimation method, the Fully Modified Ordinary Least Squares (FMOLS) method by Phillips and Hansen (1990), the Canonical Cointegrating Regression (CCR) method by Park (1992), and the Dynamic Ordinary Least Squares (DOLS) estimator by Stock and Watson (1993), which have better asymptotic properties.

It turns out that Park's (1992) CCR method to estimate the cointegrating vector has a number of advantages for our problem. The main idea of CCR is to implement LS estimation via transformed variables using the long-run covariance matrix of $\eta_t = [\varepsilon_t \ \ \mathbf{u}_t]'$, so that the LS estimator is asymptotically efficient. CCR is as efficient as the ML procedure of Johansen, but is robust to distributional assumptions because it is nonparametric. CCR is applicable to more general cases of cointegrating regression models than FMOLS.[27] DOLS is easy to implement but the results may be sensitive to the choice of numbers of leads and lags. Thus, we view CCR as most appropriate to this problem.

Given the cointegrating vector β, we construct a trivariate vector error correction model (VECM)

[25] This is the case when the deterministic cointegration restriction is satisfied. When the cointegrating vector eliminates the stochastic trend only, $\gamma' \mathbf{y}_t$ is trend stationary. See Ogaki and Park (1998) for details.

[26] See Stock (1987) and Phillips (1991) for details.

[27] For example, the FMOLS can not deal with stochastically cointegrated models with deterministic trend in the repressors.

to investigate the donor substitution effects and dirty altruism in both short- and long-run dynamic perspectives.[28] Abstracting from deterministic components,

$$\Delta \mathbf{y}_t = \rho\gamma' \mathbf{y}_{t-1} + \sum_{j=1}^{k} \mathbf{B}_j \Delta \mathbf{y}_{t-j} + \mathbf{C}\varepsilon_t, \tag{3}$$

where $\rho = [\rho_1 \;\; \rho_2 \;\; \rho_3]'$ denotes a vector of speeds of adjustment coefficients, $\mathbf{C}$ is a matrix that defines the contemporaneous structural relationship among the three variables, and $\varepsilon_t = [\varepsilon_{l,t} \;\; \varepsilon_{c,t} \;\; \varepsilon_{w,t}]'$ is a vector of mutually orthogonal structural shocks. That is, $\mathbb{E}\varepsilon_t\varepsilon_t' = \mathbf{I}$, where $\mathbf{I}$ is a 3×3 identity matrix, and $\mathbb{E}\mathbf{u}_t\mathbf{u}_t' = \mathbb{E}\mathbf{C}\varepsilon_t\varepsilon_t'\mathbf{C}' = \mathbf{C}\mathbf{C}' = \mathbf{\Sigma}$, where $\mathbf{\Sigma}$ is the LS variance-covariance matrix. We identify $\mathbf{C}$ recursively with the order $[c_t \;\; w_t \;\; l_t]$. Put in economic terms, we assume that deceased donors are not contemporaneously affected by live donors and the size of the waiting list, while the size of waiting list is not contemporaneously affected by live donors.[29,30]

To estimate response functions to each structural shock, we rewrite (3) as the following VAR($k+1$) system.

$$\mathbf{y}_t = \sum_{j=1}^{k+1} \mathbf{\Gamma}_j \mathbf{y}_{t-j} + \mathbf{C}\varepsilon_t, \tag{4}$$

where

$$\begin{aligned} \mathbf{\Gamma}_1 &= \mathbf{I}_2 + \rho\gamma' + \mathbf{B}_1 \\ \mathbf{\Gamma}_j &= \mathbf{B}_{j+1} - \mathbf{B}_j, \; j = 2, \cdots, k \\ \mathbf{\Gamma}_{k+1} &= -\mathbf{B}_k \end{aligned}$$

[28] Johansen's trace test and the maximum eigenvalue test both indicated 1 cointegrating equation at the 5% significance level. So we consider a VECM with a single cointegrating vector. Test results are available upon request.

[29] With this ordering $\tilde{\mathbf{y}}_t = [c_t \;\; w_t \;\; l_t]'$, its associated contemporaneous matrix $\tilde{\mathbf{C}}$ is a lower-triangular matrix that can be obtained by the Choleski decomposition of the LS variance-covariance matrix $\tilde{\mathbf{\Sigma}}$.

[30] Alternatively, one may use the generalized impulse response analysis proposed by Pesaran and Shin (1998), which is an ordering free method. Kim (2009) shows, however, that it yields response functions based on contradictory assumptions that may lead to misleading inferences. So we do not consider it. Furthermore, our empirical results were robust to alternative orderings.

Finally, we use the state-space representation for (4) as follows.

$$\mathbf{z}_t = \mathbf{F}\mathbf{z}_{t-1} + \zeta_t, \tag{5}$$

where

$$\mathbf{z}_t = \begin{bmatrix} \mathbf{y}_t \\ \mathbf{y}_{t-1} \\ \vdots \\ \mathbf{y}_{t-k} \end{bmatrix}, \ \mathbf{F} = \begin{bmatrix} \mathbf{\Gamma}_1 & \mathbf{\Gamma}_2 & \cdots & \mathbf{\Gamma}_{k+1} \\ \searrow & & \swarrow & \mathbf{0} \\ & \mathbf{I}_{3k} & & \vdots \\ \nearrow & & \nwarrow & \mathbf{0} \end{bmatrix}, \ \zeta_t = \begin{bmatrix} \mathbf{C}\varepsilon_t \\ \mathbf{0} \\ \vdots \\ \mathbf{0} \end{bmatrix}$$

so that the r^{th} period impulse-response functions are obtained by

$$\mathbf{S}'\mathbf{F}^r\mathbf{S},$$

where $\mathbf{S} = [\mathbf{I}_3 \ \ \mathbf{0} \ \ \cdots \ \ \mathbf{0}]'$ is a $3(k+1) \times 3$ selection matrix.

4 Empirical Results

Our data are comprised of quarterly reports of UNOS/OPTN for End-Stage Renal Disease (ESRD) patients admitted to the UNOS kidney transplant waiting list, and renal grafts performed in the US by centers reporting to UNOS. Observations span 1992:IV to 2006:II. We noticed seasonality in l_t and c_t (see Figure 1), so we will implement our empirical analysis with the seasonally adjusted data.[31] All variables are measured in natural logarithms.

As a preliminary analysis, we implement a unit-root test for the level variables and differenced variables. The conventional Phillips-Perron t test results are reported in Table 1. We find that all level variables seem nonstationary, while all differenced variables are stationary at the 10% significance level. So, we conclude that all level variables are integrated of order one.

Insert Table 1 around here

[31] We use the Census $X12$ method for seasonal adjustment. The source of this seasonality is unknown.

Next, we estimate the cointegrating vector for our baseline model (1), testing the null of cointegration by Park's (1990, 1992) $H(p,q)$ test at the same time.[32,33,34] Results are reported in the lower panel of Table 2. We test the null of cointegration instead of the null of no cointegration (e.g., Phillips-Ouliaris test). We employ this strategy because we want to control the probability (size) of rejecting the model we wish to test, which implies cointegrating relations. The $H(0,1)$ test accepts the null of deterministic cointegration with p-values of 0.460. That is, the cointegrating vector eliminates not only the stochastic trend but also deterministic time trend. Other $H(p,q)$ tests, weaker version cointegration tests without the deterministic cointegration restriction, similarly accept the null of cointegration. These results, therefore, provide strong empirical evidence of a stable relation between c_t, l_t, and w_t in the long-run.

Next, we turn to the CCR estimate of the cointegrating vector reported in the upper panel of Table 2. The coefficient estimate for c_t is negative (-1.045), as expected, and significant at the 5% significance level. The variable w_t has a strong positive (1.215) long-run relation with l_t. It should be noted, however, that the negative coefficient on c_t may be due to either the substitution effect or the dirty altruism or both, since cointegrating relations do not imply causality, this result simply means that c_t and l_t are negatively associated with each other in the long-run. The donor substitution effect, in our view, is a decrease in l_t caused by a sudden (exogenous) increase in c_t. Similarly, dirty altruism should be the case of a decrease in c_t in response to an exogenous increase in l_t. In what follows, we employ a structural VECM to separately identify the substitution effect and dirty altruism.

Insert Table 2 around here

To estimate our VECM (3), we chose $k = 1$ by the Bayesian Information Criteria, which corresponds to a VAR(2) system.[35] The 95% confidence intervals were obtained by $10,000$ nonparametric bootstrap simulations from empirical distributions.

[32] We use the quadratic spectral kernel with automatic bandwidth selection method to obtain the long-run variance matrix. We also use the VAR prewhitening method as recommended by Andrews and Monahan (1992).

[33] The $H(p,q)$ test statistic is obtained from CCR estimation of $l_t = \sum_{i=0}^{p} \alpha_i t^i + \sum_{j=p+1}^{q} \psi_j t^j + \beta' \mathbf{x}_t + \varepsilon_t$, where time polynomials up to the order of p represents maintained trends, while higher order time polynomials are "spurious" trends.

[34] Under the null hypothesis, the $H(p,q)$ test has asymptotic χ^2 distributions with $q-p$ degrees of freedom. However, the test statistic diverges to infinity under the alternative hypothesis of no cointegration. Therefore, the test is consistent.

[35] Estimating the system by allowing up to $k = 3$ yields qualitatively similar results.

We first note the substantial roles of l_t and c_t for the short-run adjustment when there is a deviation from the long-run equilibrium (see Table 3). The speed of adjustment for l_t was quantitatively largest and significant even at the 1% level, while c_t also plays an important role in short-run adjustment, of which error correction term is significant at the 5% level. w_t plays virtually no role as expected. It is also interesting to see error-correction adjustments occur fairly quickly when there is either a live donor transplant shock or a deceased donor transplant shock (Figure 2). When there is a waiting list shock, however, adjustment is slow and exhibit high persistence.

Insert Table 3 and Figure 2 around here

Table 4 reports the short-run (unnormalized) response of each variable to each structural shock. The results show that the donor substitution effect exists even in the short-run, though it is insignificant at the 5% level.[36] A 1% increase in c_t lead to a 0.188% decrease ($= -0.006/0.032$) in l_t on impact. However, the long-run substitution effect is substantial as l_t decreases even more, leading to a 0.703% decrease in the long-run, which is significant at the 5% level (see Table 5 and Figure 4).[37] "Dirty altruism" is absent in the short-run by construction. However, a 1% increase in l_t leads to 0.335% decrease in c_t in the long-run, though this is marginally significant at the 95% level (see Table 5 and Figure 5). In a nutshell, we find strong empirical evidence in favor of the donor substitution effect. The long-run evidence for the dirty altruism is also far from being negligible. We also see that a 1% increase in w_t leads to 3.716% increase in l_t in the long-run (see Table 5 and Figure 6), which corroborates the conjecture regarding the effect of "desperation" on l_t. However, given the relatively low variability of w_t, this is not policy relevant.[38]

Insert Tables 4 and 5 and Figures 3, 4, 5, and 6 around here

[36] It is significant at the 10% significance level. See the note on Table 4. Also, Figure 3 provides other impulse-response functions.

[37] The effect is significant even at the 1% significance level. Its 99% confidence interval was [-1.366,-0.167].

[38] The standard deviation of w_t is only 0.004, while those of c_t and l_t are 0.032 and 0.025, respectively. See Table 4.

5 Discussion and Conclusion

For some time members of the transplantation community have discussed the possible existence of interrelationships between living donations, cadaveric donations, and the waiting list. A variety of mechanisms have been suggested to explain the presence of these relationships. The analysis presented in this paper sought to identify and quantify two such phenomena, the "crowding out" of living donor kidneys by deceased donor organs (termed here the "substitution effect"), and the displacement of deceased donor organs by living donations (termed "dirty altruism"). In a bid to credibly and accurately assess the effects, we postulated a cointegrating relationship between these three series, and examined the speeds of adjustment, long-run (equilibrium) responses, and the impulse responses, utilizing quarterly data for the U.S. for the period 1994:IV to 2006:II . Our findings are potentially important for current and future efforts to increase kidney transplantation, and the news is not very good.

Analysis of the long-run responses to various shocks to the underlying series strongly suggests that increases in deceased donor transplants will significantly reduce living donor transplants. In particular, a 1% increase in cadaver organ grafts will result in a .7% permanent decrease in equilibrium living donor transplants. In terms of organ numbers, this suggests an increase of 106 deceased donor transplants (representing around 70 additional cadaver donors using historical values) will result in a decrease in equilibrium living donors of around 42, for a net increase in equilibrium transplants of only 62 (using 2008 transplant figures from OPTN). This finding is both statistically strong and discouraging. All current efforts to expand donor numbers, with the sole (but important) exception of pairwise exchanges, seek to increase cadaver donor organ supply, whether by more effectively convincing families to donate, or by improving protocols for use and handling of organs from substandard donors. Evidently, such efforts are going to prove far more difficult than was hoped. We reluctantly conclude that the substitution effect is real and consequential.

We emphasize that the econometric approach taken here, relying as it does on cointegrating relationships, identifies long-run equilibrium values for the series of interest. Thus, the intervening dynamics must be separately evaluated, and the impacts of shocks in one variable on others can exhibit considerable differentiation. In the case of the substitution effect, the level of living donor transplants adjusts relatively quickly, so that within two years nearly the entire effect has been realized.

Our findings for the "dirty altruism" effect are less strong, but we do have significant evidence of this phenomenon as well. A 1% increase in the level of living donor transplants reduces equilibrium deceased donor numbers by around 1/3 of a percent, although the result is only marginally significant. Still, this effect is discouraging, especially in light of the much higher numbers of deceased donor transplants. (There are sometimes more living than deceased donors, but living donors provide only a single kidney, while cadavers provide about 1.5 each on average.) Again, the adjustment path is quite rapid, with nearly the entire effect manifesting itself within two years.

Finally, our study supports the commonplace, common-sense interpretation of the relationship between the waiting list and living donation. Until the extent of pairwise exchange programs grows sufficiently, it is realistic to attribute living donation to desperation associated with, and measured by, the size of the waiting list for renal transplants. Although few "shocks" to the waiting list have occurred, impulse-response analysis points to a closely related adjustment in living donation. In total, one may say that the waiting list appears largely exogenous to the other series, and that the magnitude of the list drives and determines the level of living donation in an intuitive manner.

The implications of these findings for programs aimed at increasing transplantation are not comforting. Although most discussions of such efforts within the medical/transplantation communities implicitly assume that an additional cadaver kidney, or an additional living donor kidney, represents an additional kidney, this is not generally so. The behavior of those decision makers involved- families of brain-dead potential donors, relatives and friends of ESRD patients needing grafts, physicians and critical-care medicine specialists- respond to the circumstances of the kidney shortage in an unfortunate manner. It seems plausible that these phenomena owe their existence primarily to the ban on compensation for kidney donation. In a system without monetary incentives, factors that might otherwise be quite unimportant can become dominant. It is a testament to the desperate state of transplantation that this appears to be the case. Efforts at reform which fail to take account of these linkages are unlikely to meet the expectations of their creators, to the detriment of ESRD patients and their families.

Table 1. Unit Root Test Results for the Null of Nonstationarity

Level	Z_c	p-value	Z_t	p-value
c_t	0.3095	0.9768	-2.3937	0.3786
w_t	-1.5791	0.4862	-2.6082	0.2785
l_t	-1.7400	0.4058	-2.3987	0.3761
Differenced	Z_c	p-value	Z_t	p-value
Δc_t	-11.046	0.0000	-9.6652	0.0000
Δw_t	-2.6174	0.0959	-5.1902	0.0005
Δl_t	-9.0992	0.0000	-9.0272	0.0000

Note: i) Z_c and Z_t denote the Phillips-Perron test statistics when an intercept and when an intercept and linear time trend are included, respectively. ii) All variables are measured in natural logarithms.

Table 2. Canonical Cointegrating Regressions and Cointegration Test Results

CCR	Coefficients	Standard Error
Constant	1.7137	1.1836
c_t	-1.0452	0.2626
w_t	1.2145	0.0806
$H(p,q)$ Test	Statistics	p-value
$H(0,1)$	0.5472	0.4595
$H(1,2)$	0.1003	0.7515
$H(1,3)$	0.3556	0.8371

Note: i) p represents the order of maintained trends, while q ($\geq p+1$)is the order of spurious trends. ii) The $H(0,1)$ test has the null of deterministic cointegration while the $H(1,q)$ test has the null of stochastic cointegration without the determinstic cointegration restriction. iii) The quadratic spectral kernel with automatic bandwidth selection was used to obtain the long-run variance matrix.

Table 3. Speed of Adjustment Coefficient Estimates

Variable	ρ_i	95% CI
c_t	-0.195	$[-0.502, -0.006]$
w_t	0.005	$[-0.030, \ 0.045]$
l_t	-0.245	$[-0.495, -0.097]$

Note: i) The number of lags is 2 ($k = 1$). We obtained similar results when we allow up to 4 lags. ii) The 95% confidence intervals were obtained by 10,000 nonparametric bootstrap simulations from empirical distributions.

Table 4. Choleski Decomposition of the Covariance Matrix

$$u_{c,t} = \underset{[0.021,0.037]}{0.032}\varepsilon_{c,t}$$

$$u_{w,t} = \underset{[-0.002,0.000]}{-0.001}\varepsilon_{c,t} + \underset{[0.003,0.005]}{0.004}\varepsilon_{w,t}$$

$$u_{l,t} = \underset{[-0.012,0.001]}{-0.006}\varepsilon_{c,t} + \underset{[-0.004,0.010]}{0.003}\varepsilon_{w,t} + \underset{[0.019,0.027]}{0.025}\varepsilon_{l,t}$$

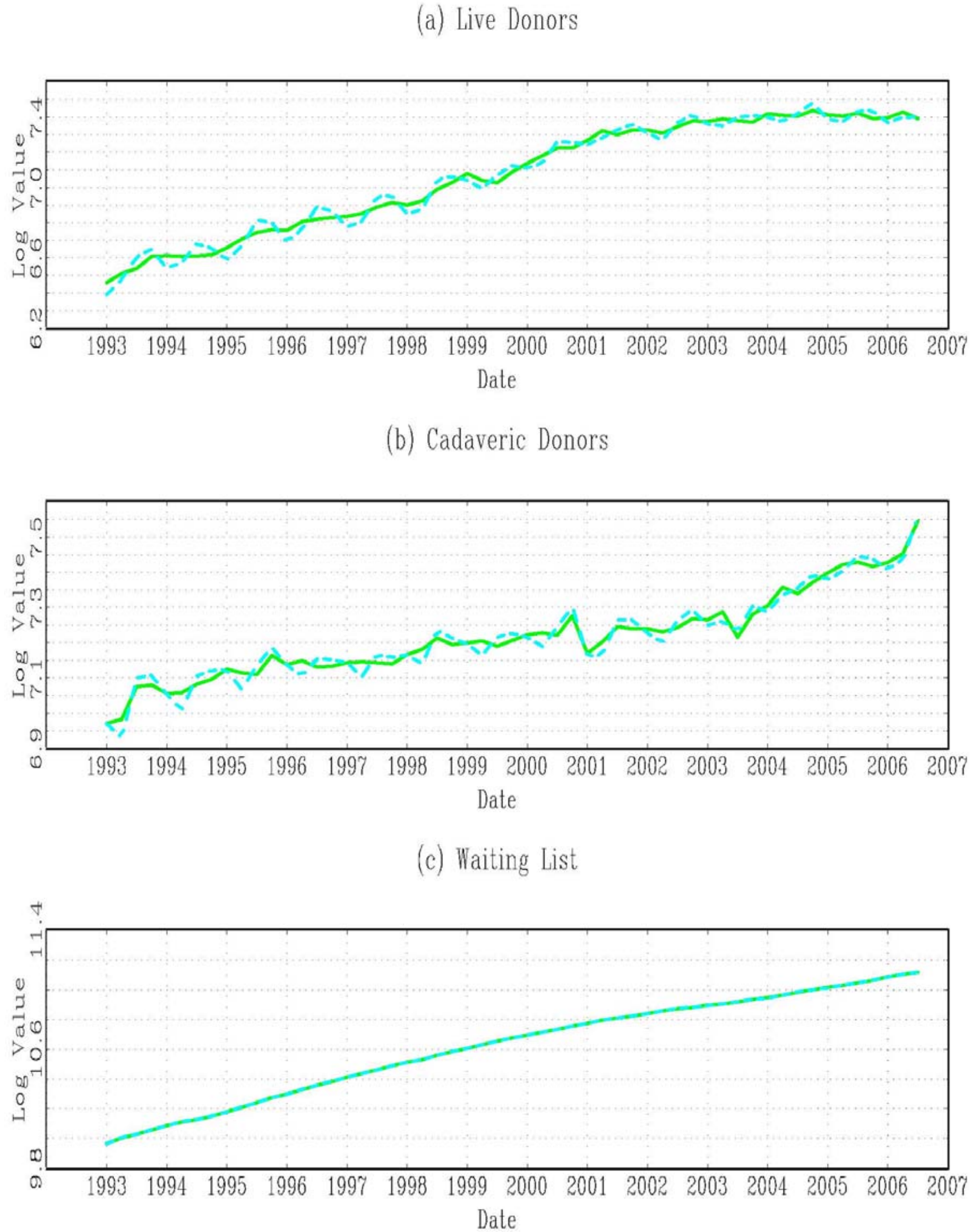
Note: i) The coefficients on the innovation terms correspond to short-run responses to each structural shock without normalization. ii) The 95% confidence intervals were obtained by 10,000 nonparametric bootstrap simulations from empirical distributions. iii) The 90% confidence interval of the contemporaneous effect of $\varepsilon_{c,t}$ on $u_{l,t}$ is [-0.011,0.000], which is marginally significant.

Table 5. Long-Run Response Estimates

Variable	$\varepsilon_{c,t}$	95% CI	$\varepsilon_{w,t}$	95% CI	$\varepsilon_{l,t}$	95% CI
c_t	0.476	$[\ 0.228, \ 0.710]$	0.134	$[-2.316, 2.493]$	-0.335	$[-0.652, -0.009]$
w_t	-0.169	$[-0.408, \ 0.042]$	3.175	$[\ 1.497, 5.309]$	0.078	$[-0.189, \ 0.351]$
l_t	-0.703	$[-1.136, -0.302]$	3.716	$[\ 0.102, 7.873]$	0.445	$[-0.091, \ 0.975]$

Note: i) The long-run responses to 1% structural shocks are reported. ii) The 95% confidence intervals were obtained by 10,000 nonparametric bootstrap simulations from empirical distributions. iii) The 99% confidence interval for the long-run response of l_t to $\varepsilon_{c,t}$ was [-1.366,-0.167]. iv) The 90% confidence interval for the long-run response of l_t to its own shock was [0.010,0.869].

Figure 1. Kidney Transplant Data



Note: We use the Census X12 method for seasonal adjustment. Dashed lines are raw data and solid lines are seasonally adjusted data.

Figure 2. Error Correction ($l_t - \beta_1 c_t - \beta_2 w_t$) Adjustments

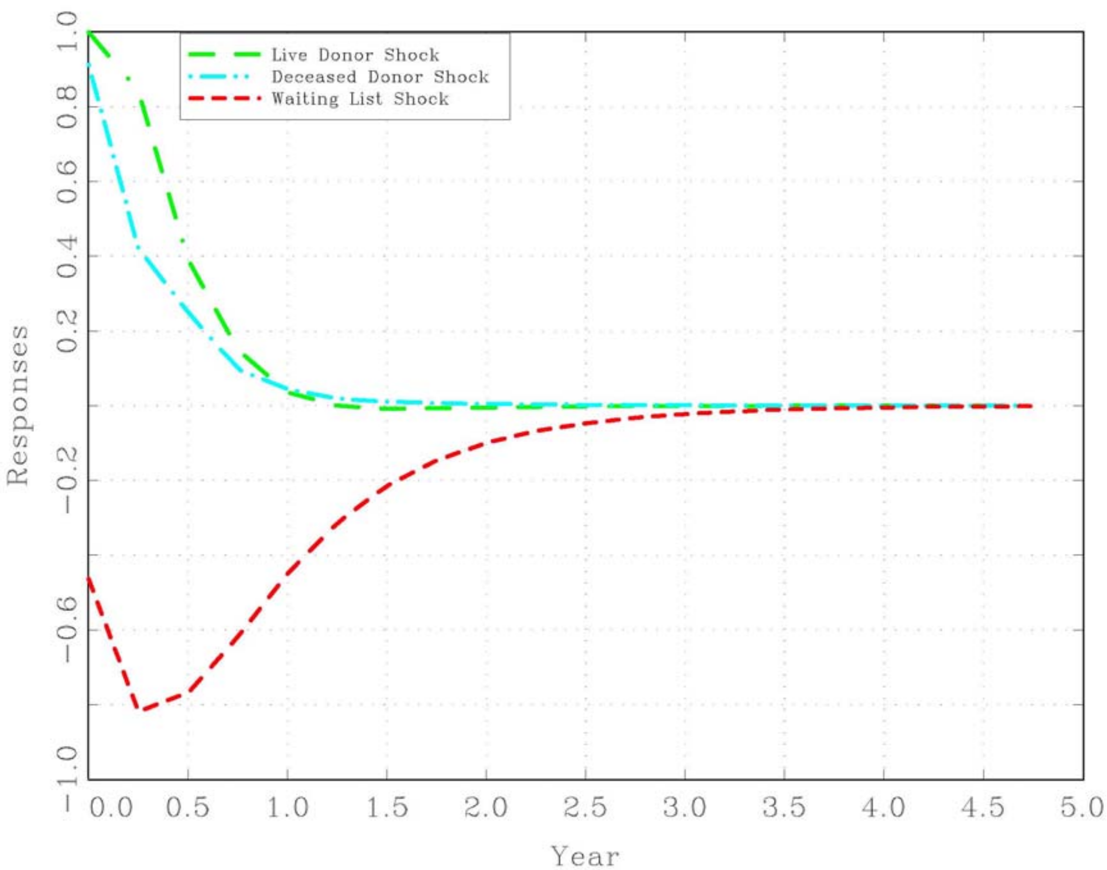
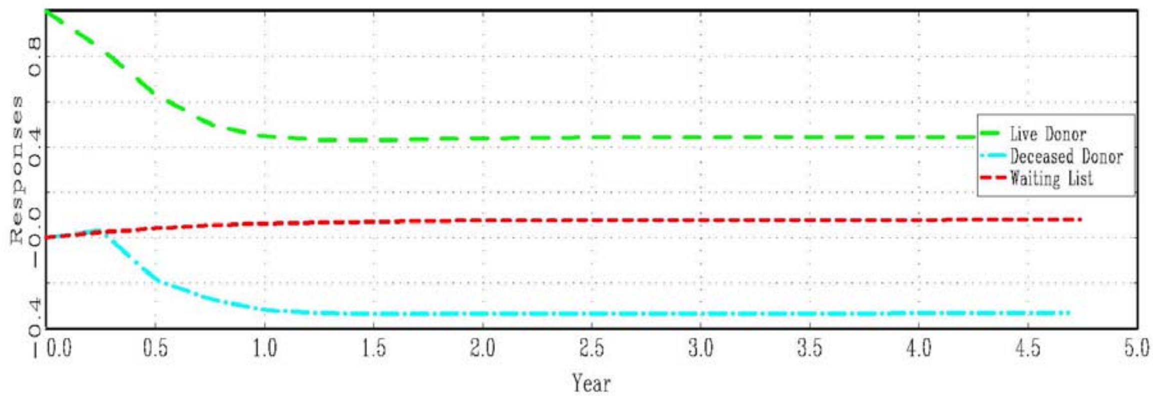
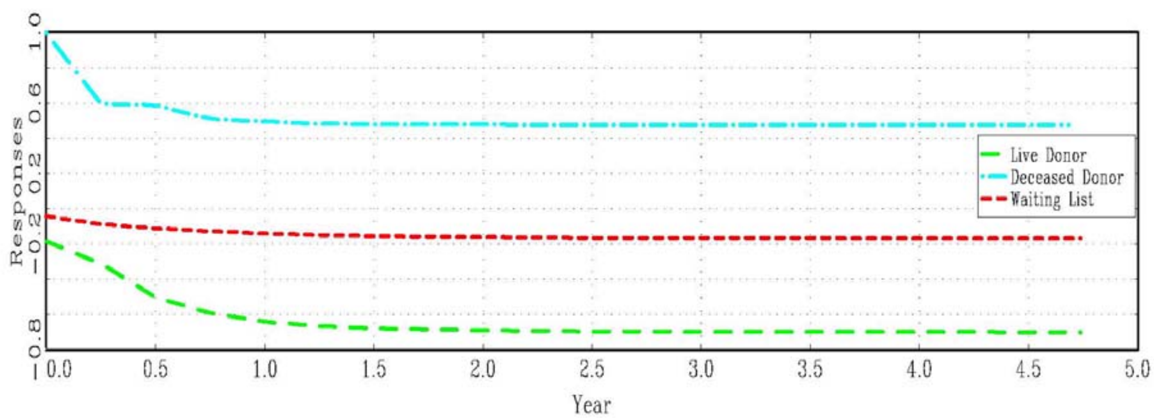


Figure 3. Impulse-Response Functions

(a) Responses to a Live Donor Shock



(b) Responses to a Deceased Donor Shock



(c) Responses to a Waiting List Shock

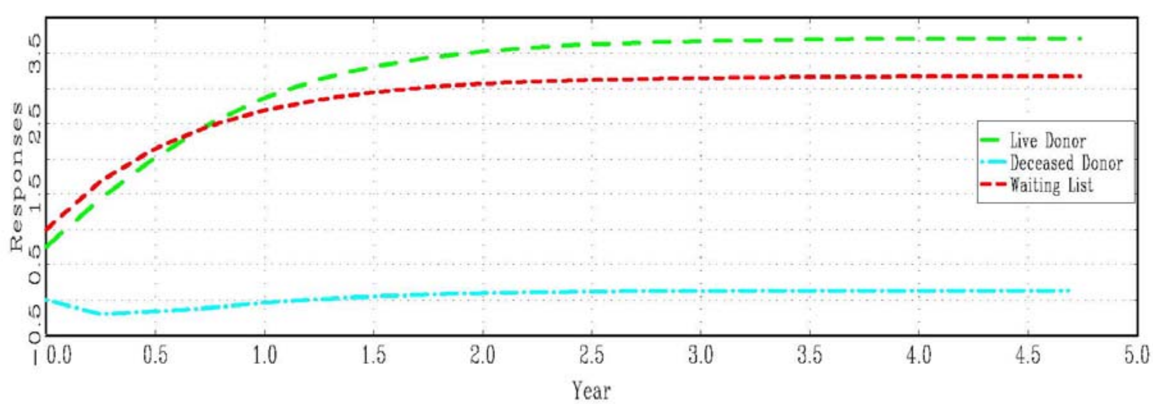
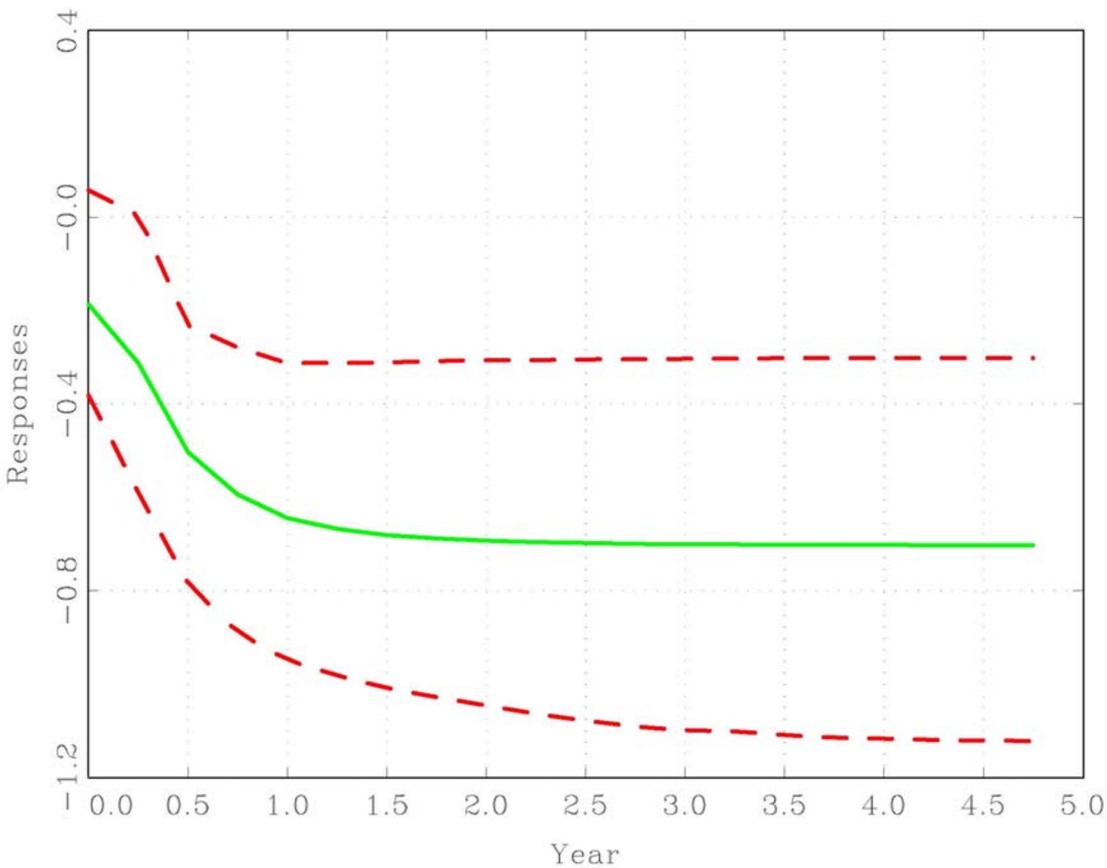
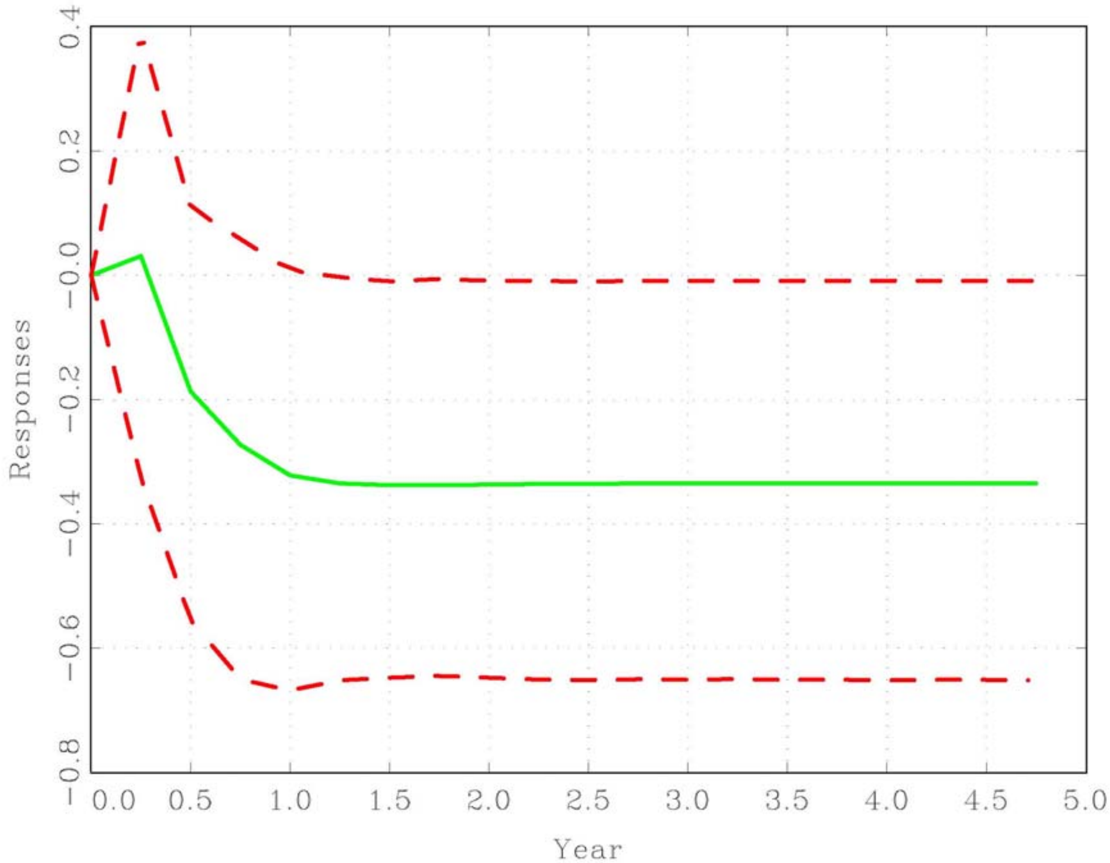


Figure 4. Response of Live Donors to a Deceased Donor Shock



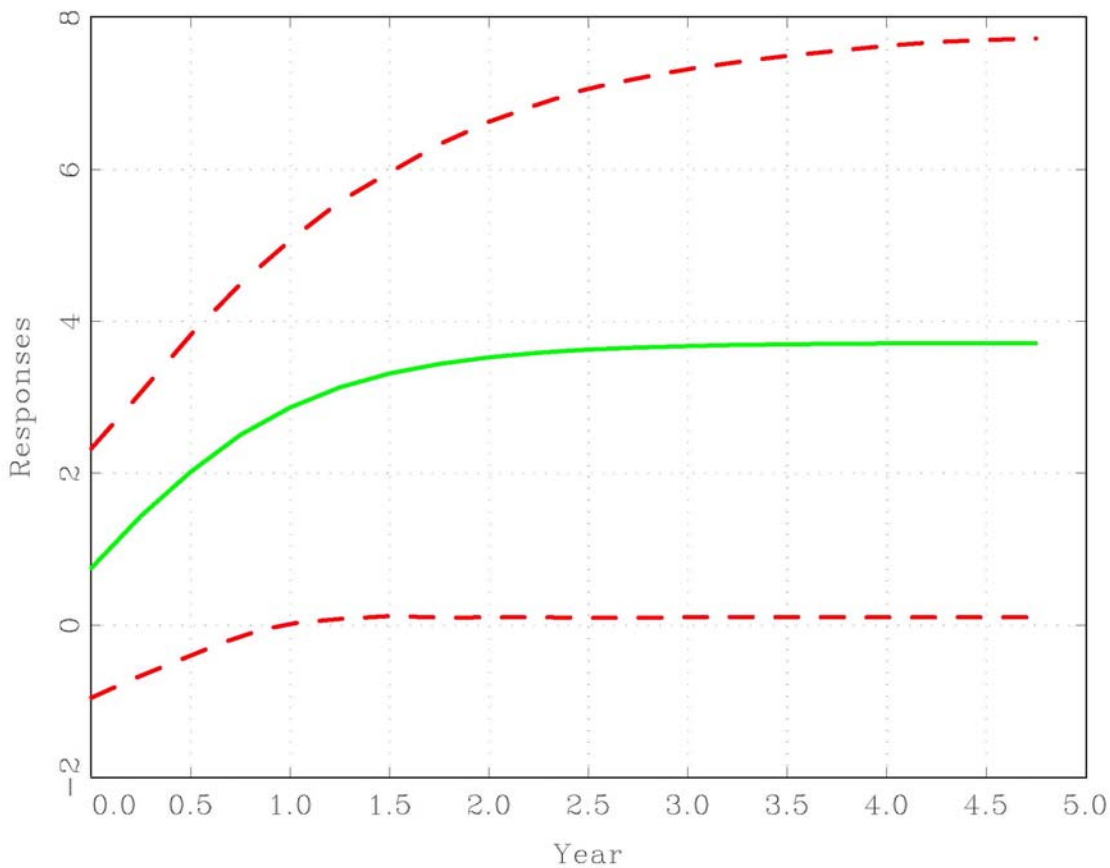
Note: The 95% confidence interval was obtained by 10,000 nonparametric bootstrap simulations from the empirical distribution.

Figure 5. Response of Deceased Donors to a Live Donor Shock



Note: The 95% confidence interval was obtained by 10,000 nonparametric bootstrap simulations from the empirical distribution.

Figure 6. Response of Live Donors to a Waiting List Shock



Note: The 95% confidence interval was obtained by 10,000 nonparametric bootstrap simulations from the empirical distribution.

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