Appendices for **"Enhancing Kidney Supply Through Geographic Sharing in the United States"**

Appendix A: Proofs

Proof of Proposition 1. It follows from (2)-(3) that

$$Q(t) = \exp\left\{-\int_0^t \gamma(s)ds\right\}\left[\lambda - \int_0^t \exp\left\{\int_0^s \gamma(u)du\right\} G'(l(s))\, l'(s)ds\right], t < \tau. \tag{16}$$

Then combining (4) and (16), we see that τ must satisfy

$$\exp\left\{\int_0^\tau \gamma(s)ds\right\}\Delta G(\bar{L}) + \int_0^\tau \exp\left\{\int_0^s \gamma(u)du\right\} G'(l(s))\, l'(s)ds = \lambda. \tag{17}$$

At equilibrium a patient who has waited for t time units must be indifferent between accepting an organ of life years $l(t)$ and waiting. That is, we must have

$$l(t) = W_t + L_t \quad \text{for } t < \tau, \quad l(t) > \underline{L}, \tag{18}$$

where W_t denotes the expected residual waiting time conditional on having waited for t time units, and L_t denotes the expected post-transplant life expectancy associated with waiting (not including the waiting time on the transplant list) of a patient who has waited for t time units.

The following figure shows the various events (and their rates) that can happen to patients who have waited for t time units:

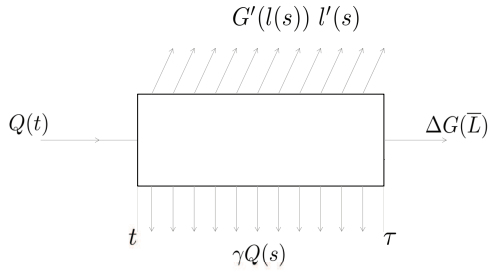

Figure 1: The portion of the transplant waiting list consisting of patients who have waited for t time units or more. Viewing this as a system, patients enter at the rate of $Q(t)$, and can leave the system at time $s \in [t, \tau)$ with rate $G'(l(s))\, l'(s)$ and at time τ with rate $\Delta G(\bar{L})$ by receiving a transplant. Patients can also leave the system by dying at rate $\gamma Q(s)$ (at time s).

Given the system portrayed in Figure 1, we write by Little's Law that

$$W_t = \frac{\int_t^\tau Q(s)ds}{Q(t)}. \tag{19}$$

To compute L_t, consider what happens to the intensity of fluid $Q(t)$ (of those who have been waiting

for t time units) as shown in Figure 1, and interpret the fraction served at various times as the probability density (or mass at time τ) of getting transplanted after waiting for $s \geq t$ time units, denoted by $\phi(s)$. Note that

$$\phi(s) = \frac{G'(l(s))\ l'(s)}{Q(t)} \quad \text{for } t \leq s < \tau \quad \text{and } \phi(\tau) = \frac{\Delta G(\bar{L})}{Q(t)}. \tag{20}$$

Then, we write

$$L_t = \int_t^\tau \phi(s) l(s) ds + \phi(\tau)\bar{L}. \tag{21}$$

Substituting (20) into (21) yields

$$L_t = \int_t^\tau l(s) \frac{G'(l(s))\ l'(s)}{Q(t)}\, ds + \frac{\Delta G(\bar{L})}{Q(t)}\, \bar{L}.$$

Equivalently,

$$L_t = \frac{1}{Q(t)} \int_{l(t)}^{\bar{L}} u dG(u). \tag{22}$$

Substituting (19) and (22) into (18) gives

$$\int_t^\tau Q(s) ds + \int_{l(t)}^{\bar{L}} u dG(u) = Q(t) l(t) \quad \text{for } t < \tau,\ l(t) > \underline{L}. \tag{23}$$

In what follows, we will first ignore the restriction $l(t) > \underline{L}$ in (23) and solve for $f(\cdot)$ that solves (23). Then, we will observe that $f(\cdot)$ is strictly increasing. Therefore, truncating $f(\cdot)$ at $\underline{L}$ from below yields $l(\cdot)$. To this end, consider the equation

$$\int_t^\tau Q(s) ds + \int_{f(t)}^{\bar{L}} u dG(u) = Q(t) f(t) \quad \text{for } t < \tau.$$

Differentiating both sides with respect to t and substituting for $Q'(t)$ (cf. Equation (3)) gives

$$-1 = -\gamma(t) f(t) + f'(t) \quad \text{for } t < \tau. \tag{24}$$

Also, using the boundary condition that $f(\tau) = l(\tau) = \bar{L}$ gives

$$f(t) = \exp\left\{\int_0^t \gamma(s) ds\right\} \left[\bar{L} \exp\left\{-\int_0^\tau \gamma(s) ds\right\} + \int_t^\tau \exp\left\{-\int_0^s \gamma(u) du\right\} ds\right], \quad \text{for } 0 < t < \tau. \tag{25}$$

Then, the patients' strategy is really the truncated function:

$$l(t) = \max\{\underline{L}, f(t)\} \tag{26}$$

which proves (5). Also note that (16) proves (4). To prove (6), we first consider how $f(t)$ and $f'(t)$ change with τ. Note that

$$\frac{\partial f(t)}{\partial \tau} = -(\bar{L}\gamma(\tau) - 1) \exp\left\{-\int_t^\tau \gamma(s) ds\right\} < 0,\ t < \tau, \tag{27}$$

which incidentally proves the last assertion of the proposition. Also note from (24) that

$$\frac{\partial f'(t)}{\partial \tau} = \gamma(t)\frac{\partial f(t)}{\partial \tau} < 0. \tag{28}$$

To establish the uniqueness of τ satisfying (6), consider (17) and define $H(\tau)$ as its left-hand side, i.e.,

$$H(\tau) = \exp\left\{\int_0^\tau \gamma(s)ds\right\}\Delta G(\bar{L}) + \int_0^\tau \exp\left\{\int_0^s \gamma(u)du\right\} G'(l(s))\, l'(s)ds. \tag{29}$$

Differentiating this, substituting $l'(\tau) = \bar{L}\gamma(\tau) - 1$ from (24), and rearranging terms gives

$$\begin{aligned} H'(\tau) = {} & \exp\left\{\textstyle\int_0^\tau \gamma(s)ds\right\}\left[\gamma(\tau)\Delta G(\bar{L}) + G'(\bar{L})(\bar{L}\gamma(\tau) - 1)\right] \\ & + \textstyle\int_0^\tau \exp\left\{\int_0^s \gamma(u)du\right\}\frac{\partial}{\partial \tau}\left[\frac{d}{ds}G(l(s))\right] ds. \end{aligned} \tag{30}$$

Changing the order of differentiation for the integrand of the last term on the right-hand side yields

$$\begin{aligned} H'(\tau) = {} & \exp\left\{\textstyle\int_0^\tau \gamma(s)ds\right\}\left[\gamma(\tau)\Delta G(\bar{L}) + G'(\bar{L})(\bar{L}\gamma(\tau) - 1)\right] \\ & + \textstyle\int_0^\tau \exp\left\{\int_0^s \gamma(u)du\right\}\frac{d}{ds}\left[\frac{\partial}{\partial \tau}G(l(s))\right] ds. \end{aligned} \tag{31}$$

Note that

$$\frac{\partial}{\partial \tau}G(l(s)) = G'(l(s))\frac{\partial l(s)}{\partial \tau}.$$

Then, note from (27) that

$$\frac{\partial}{\partial \tau}G(l(s)) = -G'(l(s))(\bar{L}\gamma(\tau) - 1)\exp\left\{-\int_s^\tau \gamma(u)du\right\}.$$

Thus, we conclude that

$$\begin{aligned} H'(\tau) = {} & \exp\left\{\textstyle\int_0^\tau \gamma(s)ds\right\}\left[\gamma(\tau)\Delta G(\bar{L}) + G'(\bar{L})(\bar{L}\gamma(\tau) - 1)\right] \\ & - \textstyle\int_0^\tau \exp\left\{\int_0^s \gamma(u)du\right\}\frac{d}{ds}\left[G'(l(s))(\bar{L}\gamma(\tau) - 1)\exp\left\{-\int_s^\tau \gamma(u)du\right\}\right] ds. \end{aligned} \tag{32}$$

Integrating the last term on the right-hand side by parts gives

$$\begin{aligned} H'(\tau) = {} & \exp\left\{\int_0^\tau \gamma(s)ds\right\}\left[\gamma(\tau)\Delta G(\bar{L}) + G'(\bar{L})(\bar{L}\gamma(\tau) - 1)\right] \\ & - \exp\left\{\int_0^s \gamma(u)du\right\} G'(l(s))(\bar{L}\gamma(\tau) - 1)\exp\left\{-\int_s^\tau \gamma(u)du\right\}\Big|_0^\tau \\ & + \int_0^\tau \gamma(s)\exp\left\{\int_0^\tau \gamma(u)du\right\} G'(l(s))(\bar{L}\gamma(\tau) - 1)\exp\left\{-\int_s^\tau \gamma(u)du\right\} ds \\ = {} & \exp\left\{\int_0^\tau \gamma(s)ds\right\}\left[\gamma(\tau)\Delta G(\bar{L}) + G'(\bar{L})(\bar{L}\gamma(\tau) - 1)\right] \\ & - \exp\left\{\int_0^\tau \gamma(u)du\right\} G'(l(\tau))(\bar{L}\gamma(\tau) - 1) \\ & + G'(l(0))(\bar{L}\gamma(\tau) - 1)\exp\left\{-\int_0^\tau \gamma(u)du\right\} \end{aligned}$$

$$
\begin{aligned}
&+\int_0^\tau \gamma(s)\exp\left\{\int_0^s \gamma(u)du\right\} G'(l(s))(\bar{L}\gamma(\tau)-1)\exp\left\{-\int_s^\tau \gamma(u)du\right\} ds \\
&>\exp\left\{\int_0^\tau \gamma(s)ds\right\}\left[\gamma(\tau)\Delta G(\bar{L})+G'(\bar{L})(\bar{L}\gamma(\tau)-1)-G'(\bar{L})(\bar{L}\gamma(\tau)-1)\right] \\
&=\exp\left\{\int_0^\tau \gamma(s)ds\right\}\gamma(\tau)\Delta G(\bar{L})>0.
\end{aligned}
$$

Therefore, $H(\cdot)$ is strictly increasing. Also note that $H(0)=\Delta G(\bar{L})$ and $\lim_{\tau\to\infty} H(\tau)=\infty$.

Thus, for every $\lambda > \Delta G(\bar{L})$, there exists a unique τ such that (6) holds. It is also immediate from the monotonicity of $H(\cdot)$ that as λ increases, so does τ, which concludes the proof. ■

Proof of Corollary 1. (8) follows from (5) by direct substitution of $\gamma(t)=\gamma$ for all t. Note also that equation (17) in the Proof of Proposition 1, which pins down τ, becomes

$$
e^{\gamma\tau}\Delta G(\bar{L})+\int_0^\tau e^{\gamma s}G'(l(s))l'(s)ds=\lambda. \tag{33}
$$

Then substituting (8) into (33) and making the change of variable $u=l(s)$ gives

$$
e^{\gamma\tau}\int_{l(0)}^{\bar{L}}\left(u-\frac{1}{\gamma}\right)dG(u)=\lambda\left(\bar{L}-\frac{1}{\gamma}\right).
$$

Similarly, substituting $\gamma(t)=\gamma$ for all t in (16) gives

$$
Q(t)=e^{\gamma t}\left[\lambda-\int_0^t e^{\gamma s}G'(l(s))l'(s)ds\right], t<\tau. \tag{34}
$$

Then substituting (8) into (34) and making the change of variable $u=l(s)$ gives (10), concluding the proof. ■

Appendix B: Details of the Calculation of Variables

Waiting Time to Transplantation in a DSA. Although the donor and recipient data sets do not directly include this kind of information, we calculate this variable by using three variables in the recipient data: *init_date*, *end_date*, and *trr_id_code*. The first two variables represent the date on which the patient is added to the waiting list and on which the patient is removed from the waiting list, respectively. The third variable is the transplant identifier which is only non-missing if a transplantation has occurred.

The *end_date* variable can represent the transplantation date or the date a patient is removed from the waiting list due to other reasons (e.g., death). By using the *trr_id_code*, we can identify all patients who had a transplantation in our data. Hence, we first group the data by each DSA during each quarter by blood type. Then, we calculate the waiting time of each patient who had a transplantation in the recipient data by finding the time difference between his/her *end_date* and *init_date* if this patient was added to the waiting list prior to the beginning of or within the observed quarter and was removed from the list before the last day of the quarter. Finally, the median value (in terms of years) of this variable is calculated for all observations grouped by DSA, blood type, and quarter.

Kidney Donor Risk Index. This index combines a variety of donor factors into a single continuous scale that captures the risk of graft failure after kidney transplantation. There are 10 factors considered in calculating the KDRI. These factors are donor age, height, weight, ethnicity, history of hypertension, diabetes status, serum creatinine level, cause of death, Hepatitis C Virus status, and DCD (donation after circulatory death) status. Note that the lower the KDRI of a donor, the higher is the donor kidney quality. This index was first developed by Rao et al. (2009) by estimating the association between these 10 donor factors and graft survival by using multivariable Cox proportional hazards regression model. The donor characteristics and their estimated coefficients are provided in Table 3 in Appendix E. There is another index called Kidney Donor Profile Index (KDPI) which is a mapping of the KDRI based on the profiles of all deceased donors in the U.S. from whom a kidney was recovered during the prior calendar year. In this study, instead of using this type of mapping we calculate the KDRI value for each donor in our data set and use this variable in the regressions as a proxy for donor kidney quality.

Herfindahl-Hirschman Index. From our recipient data set, we first calculate the total cumulative number of registered patients during our period of study (January 1, 2000 through June 30, 2010) at each transplant center by using the variable *init_date* which indicates the date when a patient is added to the waiting list. Next, we calculate the total cumulative number of registered patients at an OPO by adding the total cumulative number of registered patients at all transplant centers that belong to the observed OPO. Note that we use an HHI for each OPO for each blood type separately. Let λ_{ck} represent the total number of patients registered at transplant center c by blood type k, then the total number of registered patients at each DSA j for each blood type k equals $\sum_{c\in\Omega_j}\lambda_{ck}$ where Ω_j represents the set of transplant centers in each DSA j. The market share of each transplant center c by blood type k then equals to $s_{ck}=\dfrac{\lambda_{ck}}{\sum_{c\in\Omega_j}\lambda_{ck}}$ for transplant center c in DSA j. The Herfindahl-Hirschman Index is calculated as $\sum_{c\in\Omega_j}s_{ck}^2$.

Appendix C: An Additional Figure

Figure 2 indicates that the avarage HHI of all OPOs by year doesn't change significantly over time, but there is heterogeneity across different blood types; i.e., the market seems to be more competitive for blood types A and O; and slightly less competitive for blood types B and AB.

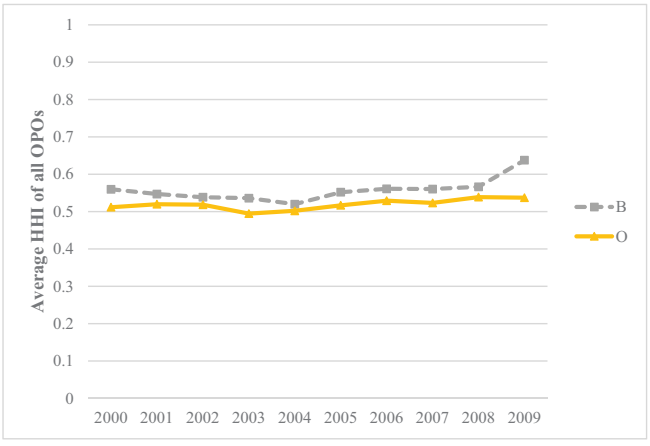

Figure 2: Average HHI of all OPOs by blood types B and O over time.

Appendix D: Endogeneity Correction Method

Two-stage least squares (2SLS) is the common method for testing and eliminating endogeneity in linear models. However, it cannot be easily extended to non-linear models like ours. The control function approach (Rivers and Vuong (1988), Petrin and Train (2010)) is better suited to nonlinear models with continuous variables. The control function approach can be thought of as a two-step procedure to deal with the issue of endogeneity in econometric models. To illustrate the intuition behind this approach, consider a valid instrumental variable (IV), z, and assume only one endogenous variable, x in the model specification. This instrumental variable has to be correlated with the endogenous variable while it should not be correlated with the error term. The control function simply correspond to the estimated residuals of the regression of x on z. Then, since the instrumental variable is not correlated with the original error term, the control function captures the part of x which is correlated with the error in the original model and therefore serves as a control for it.

For the first step, we assume the following functional forms:

$$W_{jkt} = \omega' q_{ij} + \eta_{ij}, \tag{35}$$

$$HHI_{jk} = \psi' q_{ij} + \zeta_{ij}, \tag{36}$$

where $q_{ij} = \left(KDRI_i, \widehat{W_{jkt}}, \widehat{HHI_{jk}}\right)$. The vectors ω and ψ denote the corresponding parameters to be estimated; and η_{ij} and ζ_{ij} are the error terms.

The control function approach further assumes that ε_{ij}, η_{ij}, and ζ_{ij} are one-on-one independent of $KDRI_i$, $\widehat{W_{jkt}}$, and $\widehat{HHI_{jk}}$ where ε_{ij} is the error term in (11). However, ε_{ij} and η_{ij} (also, ε_{ij} and ζ_{ij}) are allowed to be correlated. Consider now the distribution of ε_{ij} conditional on η_{ij}. We can decompose ε_{ij} into its mean, conditional on η_{ij} as follows:

$$\varepsilon_{ij} = \mathbb{E}[\varepsilon_{ij}|\eta_{ij}] + \widetilde{\varepsilon}^W_{ij}, \tag{37}$$

where $\widetilde{\varepsilon}^W_{ij}$ is the error term and by construction $\mathbb{E}[\widetilde{\varepsilon}^W_{ij}|\eta_{ij}] = \mathbb{E}[\widetilde{\varepsilon}^W_{ij}\eta_{ij}] = 0$. The conditional expectation in (37) is called *the control function* used for the variable W_{jkt} for which we assume a linear functional form. Hence, the control function is a function of η_{ij} and is denoted by $\mathbf{CF}_1(\eta_{ij}; \lambda_W) = \lambda_W \eta_{ij}$ where λ_W is a coefficient term to be estimated.[1]

Consider now the distribution of ε_{ij} conditional on ζ_{ij}. Similarly, we can decompose ε_{ij} into its mean, conditional on ζ_{ij} as follows:

$$\varepsilon_{ij} = \mathbb{E}[\varepsilon_{ij}|\zeta_{ij}] + \widetilde{\varepsilon}^C_{ij}, \tag{38}$$

where $\widetilde{\varepsilon}^C_{ij}$ is the error term and by construction $\mathbb{E}[\widetilde{\varepsilon}^C_{ij}|\zeta_{ij}] = \mathbb{E}[\widetilde{\varepsilon}^C_{ij}\zeta_{ij}] = 0$. The control function (i.e., the conditional expectation in (38)) is a function of ζ_{ij} and is denoted by $\mathbf{CF}_2(\zeta_{ij}; \lambda_C) = \lambda_C \zeta_{ij}$ where λ_C is a coefficient term to be estimated.[2] Therefore, since $\varepsilon_{ij} = \mathbf{CF}_1(\eta_{ij}; \lambda_W) + \widetilde{\varepsilon}^W_{ij} =$

[1] Assuming ε_{ij} and η_{ij} are jointly normal with zero mean, by the properties of the multivariate distribution, $\widetilde{\varepsilon}^W_{ij} = \varepsilon_{ij} - \lambda_W \eta_{ij}$ is also normally distributed. Note that $\mathbb{E}[\varepsilon_{ij}|\eta_{ij} = n] = \left(\frac{\mathrm{Cov}(\varepsilon_{ij}, \eta_{ij})}{\mathrm{Var}(\eta_{ij})}\right) \times n$, and hence λ_W reflects a covariance term.

[2] Assuming ε_{ij} and ζ_{ij} are jointly normal with zero mean, by the properties of the multivariate normal distribution, $\widetilde{\varepsilon}^C_{ij} = \varepsilon_{ij} - \lambda_C \zeta_{ij}$ is also normally distributed.

$\mathbf{CF}_2(\zeta_{ij};\lambda_C)+\widetilde{\varepsilon}^C_{ij}$,

$$\varepsilon_{ij}=\left(\frac{\lambda_W}{2}\right)\eta_{ij}+\left(\frac{\lambda_C}{2}\right)\zeta_{ij}+\left(\frac{\widetilde{\varepsilon}^W_{ij}+\widetilde{\varepsilon}^C_{ij}}{2}\right).$$

Then, the utility function $U_{ij}(Y_i)$ becomes

$$U_{ij}(Y_i)=\begin{cases}\beta' x_{ij}+\left(\frac{\lambda_W\eta_{ij}+\lambda_C\zeta_{ij}}{2}\right)+\left(\frac{\widetilde{\varepsilon}^W_{ij}+\widetilde{\varepsilon}^C_{ij}}{2}\right), & \text{if } Y_i=1;\\ 0, & \text{otherwise.}\end{cases} \tag{39}$$

This is an independent probit model with variables $KDRI_i$, W_{jkt}, HHI_{jk}, η_{ij}, ζ_{ij}, and the final term i.i.d. with zero mean.[3] As we assume ε_{ij} has a normal distribution, we implement the two-step approach developed by Rivers and Vuong (1988)[4]: (i) perform two OLS regressions: W_{jkt} on $KDRI_i$, $\widehat{W_{jkt}}$, and $\widehat{HHI_{jk}}$; and HHI_{jk} on $KDRI_i$, $\widehat{W_{jkt}}$, and $\widehat{HHI_{jk}}$ to obtain residuals $\hat{\eta}_{ij}$ and $\hat{\zeta}_{ij}$ respectively; and (ii) perform probit regression of the intent probability on $KDRI_i$, W_{jkt}, HHI_{jk}, $\hat{\eta}_{ij}$ and $\hat{\zeta}_{ij}$. The results of the Step 1 regressions are in Table 1.

Dep. Var.: W_{jkt}		**Dep. Var.: HHI_{jk}**	
Variable (Coefficient)	**Coefficient Estimate**	**Variable** (Coefficient)	**Coefficient Estimate**
Constant (ω_0)	1.266* (0.019)	Constant (ψ_0)	0.168* (0.005)
$KDRI_i$ (ω_1)	0.031* (0.007)	$KDRI_i$ (ψ_1)	0.008* (0.002)
$\widehat{W_{jkt}}$ (ω_2)	0.383* (0.007)	$\widehat{W_{jkt}}$ (ψ_2)	-0.039* (0.002)
$\widehat{HHI_{jk}}$ (ω_3)	-0.520* (0.020)	$\widehat{HHI_{jk}}$ (ψ_3)	0.737* (0.005)
$F(3,75742)=1874.74$, $R^2=0.07$		$F(3,76170)=9664.48$, $R^2=0.28$	

Note: Standard errors are in parenthesis. ($^*p<0.0001$)

Table 1: Control Function Approach Step 1 Results

Step 1 diagnostics such as the F-test and partial R^2 provide a sense of how well IVs perform in our model setting. As can be seen in Table 1, both regression models passed the F-test (i.e., Prob $> F = 0.000$) and 1− Partial R^2 values (0.98 and 0.99 for the waiting time and the competition IVs respectively) show high explanatory power.[5] Additionally, there is reasonably high correlation (i.e., 0.25) between the first endogenous variable (W_{jkt}) and its IV; and there is high correlation (i.e., 0.52) between the second endogenous variable (HHI_{jk}) and its corresponding IV.

We test the evidence of endogeneity in Step 2 of the control function approach in Table 2. As can be seen in Table 2, the coefficient for $\hat{\zeta}_{ij}$ is significant implying that the competition variable

[3]Note that $\text{Var}(\varepsilon_{ij}) > \text{Var}\left(\frac{\widetilde{\varepsilon}^W_{ij}+\widetilde{\varepsilon}^C_{ij}}{2}\right)$; hence, the coefficient estimates need to be normalized. In addition, the probit standard errors and test statistics based on the utility function (39) will not be accurate because this regression will include the residuals from regressions based on functional forms (35) and (36). Therefore, we use bootstrapping for estimating the true standard errors.

[4]Rivers and Vuong (1988) used only one IV in their approach, whereas in our approach we used two IVs and by the properties of the normal distribution we can separate out the part in the error term that correlates with the endogenous variables.

[5]We calculate the partial R^2 (or the coefficient of partial determination) of the variables different form the IVs, which indicates the percentage of variation that is not explained by the IV and is explained by the remaining parameters. Assuming the reduced model includes only the IV and the full model includes all three variables, the partial $R^2=\frac{\text{SSE(reduced)}-\text{SSE(full)}}{\text{SSE(reduced)}}$. 1− Partial R^2 provides a sense of the explanatory power of the IV.

is endogenous. However, the coefficient for $\hat{\eta}_{ij}$ is not significant (i.e., Prob $> t = 0.419$) and hence we conclude that the waiting time until transplantation variable is exogenous. Note that the Wald test of combined exogeneity (Wooldridge (2002), pp. 472-477) for the competition and waiting time variables is rejected, so we have additional evidence of endogeneity in our specification. Note that we also conduct the Wald test of exogeneity only for the competition variable and again find evidence of endogeneity of the competition variable.

Dep. Var.: Prob(Intent)

Variable	**Coefficient Estimate**	**p-value**
Constant	2.851 (0.089)	0.000
$KDRI_i$	-0.988 (0.012)	0.000
W_{jkt}	-0.011 (0.038)	0.773
HHI_{jk}	-0.130 (0.068)	0.056
$\hat{\eta}_{ij}$	0.031 (0.038)	0.419
$\hat{\zeta}_{ij}$	0.215 (0.075)	0.004

Wald test of exogeneity: $\chi^2(2) = 10.19$ (Prob $> \chi^2 = 0.006$)
Note: *Bootstrap* standard errors are in parenthesis.

Table 2: Control Function Approach Step 2 Results

Appendix E: Additional Tables

Donor Characteristic	**Applies to:**	**KDRI Coefficient ("Beta")**	**KDRI "XBeta" Component**
Age (integer years)	All Donors	0.0128	0.0128*(age-40)
	Donors with age < 18	-0.0194	-0.0194*(age-18)
	Donors with age > 50	0.0107	0.0107*(age-50)
Height (cm)	All donors	-0.0464	-0.0464*(hgt-170)/10
Weight (kg)	All donors w/ weight < 80 kg	-0.0199	-0.0199*(wgt-80)/5
Ethnicity	African American donors	0.1790	0.1790
History of Hypertension	Hypertensive donors	0.1260	0.1260
History of Diabetes	Diabetic donors	0.1300	0.1300
Cause of Death	Donors w/ COD=CVA	0.0881	0.0881
Serum Creatinine	All donors	0.2200	0.2200*(creat-1)
	Donors with creat > 1.5 mg/dL	-0.2090	-0.2090*(creat-1.5)
HCV status	HCV positive donors	0.2400	0.2400
DCD status	DCD donors	0.1330	0.1330

Table 3: KDRI Donor Factors Estimated Coefficients

Year	**2000**	**2001**	**2002**	**2003**	**2004**	**2005**
85 Percentile KDRI	1.725	1.735	1.747	1.798	1.828	1.901
Year	**2006**	**2007**	**2008**	**2009**	**2010**	
85 Percentile KDRI	1.886	1.903	1.88	1.888	1.851	

Table 4: 85th Percentile of KDRI Variable by each Year

Dep. Var.: Prob(Intent)
Whole Data

Variable (Coefficient)	**Parameter Estimate** (Blood Type=A)	(Blood Type=O)	(Blood Type=AB)	(Blood Type=B)
Constant	2.824***	2.750***	3.038***	2.733***
(β_0)	(0.067)	(0.056)	(0.253)	(0.086)
$\boldsymbol{KDRI_i}$	-1.005***	-0.988***	-1.002***	-0.942***
(β_{KDRI})	(0.024)	(0.021)	(0.101)	(0.037)
$\boldsymbol{W_{jkt}}$	0.016	0.025*	0.017	0.019
(β_W)	(0.018)	(0.012)	(0.070)	(0.018)
$\boldsymbol{HHI_{jk}}$	-0.082	-0.102	-0.350	-0.061
(β_{HHI})	(0.083)	(0.071)	(0.412)	(0.135)
Num. of Obs.	28,925	36,417	1,514	8,620

Dep. Var.: Prob(Intent)
Bottom 15% Quality Donors

Variable (Coefficient)	**Parameter Estimate** (Blood Type=A)	(Blood Type=O)	(Blood Type=AB)	(Blood Type=B)
Constant	2.634***	2.602***	3.436***	2.626***
(β_0)	(0.142)	(0.117)	(0.753)	(0.231)
$\boldsymbol{KDRI_i}$	-0.943***	-0.980***	-1.237***	-0.896***
(β_{KDRI})	(0.055)	(0.045)	(0.316)	(0.089)
$\boldsymbol{W_{jkt}}$	0.043	0.071***	0.019	0.032
(β_W)	(0.028)	(0.020)	(0.105)	(0.031)
$\boldsymbol{HHI_{jk}}$	-0.015	-0.081	-0.014	-0.076
(β_{HHI})	(0.078)	(0.067)	(0.365)	(0.145)
Num. of Obs.	4,042	5,577	231	1,372

Dep. Var.: Prob(Intent)
Top 85% Quality Donors

Variable (Coefficient)	**Parameter Estimate** (Blood Type=A)	(Blood Type=O)	(Blood Type=AB)	(Blood Type=B)
Constant	2.954***	2.957***	2.937***	2.881***
(β_0)	(0.080)	(0.066)	(0.377)	(0.126)
$\boldsymbol{KDRI_i}$	-1.111***	-1.147***	-0.969***	-1.082***
(β_{KDRI})	(0.033)	(0.037)	(0.181)	(0.073)
$\boldsymbol{W_{jkt}}$	0.009	0.012	0.026	0.015
(β_W)	(0.020)	(0.013)	(0.075)	(0.019)
$\boldsymbol{HHI_{jk}}$	-0.060	-0.073	-0.250	-0.019
(β_{HHI})	(0.110)	(0.076)	(0.443)	(0.210)
Num. of Obs.	25,316	31,319	1,475	7,532

Note: Bootstrap standard errors are in parenthesis.
*** $p < 0.001$, ** $p < 0.01$, * $p < 0.05$, $^+$ $p < 0.10$.

Table 5: Summary of Estimation Models by Blood Type from Whole Data, Bottom 15%, and Top 85% Quality Donors (Endogeneity-corrected Intent Model)

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