Prediabetic Living Kidney Donors Have Preserved Kidney Function at 10 Years After Donation

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Background. Potential living kidney donors with prediabetes are often excluded from donation because of concerns about the development of type 2 diabetes mellitus (DM) and progression to end-stage renal disease (ESRD). This strategy may be unnecessarily restrictive. Previous studies of living kidney donors have not specifically examined subsets with prediabetes.

Methods. We ascertained the vital status and development of ESRD in 143 living kidney donors from 1994 to 2007 with predonation impaired fasting glucose (IFG). We then compared the development of DM, the estimated glomerular filtration rate, and the level of albumin excretion in 45 of these IFG donors to 45 matched controls with normal predonation fasting glucose.

Results. The majority (57.8%) of IFG donors had reverted to normal fasting glucose at a mean follow-up of 10.4 years. Compared with donors with normal fasting glucose, a higher proportion of IFG donors had developed DM (15.56% vs. 2.2%, P=0.06). Predonation characteristics including age, sex, and body mass index did not correlate with the risk of developing DM. At follow- up, estimated glomerular filtration rate by the Modification of Diet in Renal Disease equation (70.7±16.1 mL/min/1.73 m^2 vs. 67.3±16.6 mL/min/1.73 m^2, P=0.21) and albumin excretion (urine albumin/ creatinine 9.76±23.6 mg/g vs. 5.91±11 mg/g, P=0.29) were similar in IFG and normal glucose donors.

Conclusion. Carefully screened prediabetic living kidney donors often revert to normal fasting glucose and do not seem to have a significantly increased risk of impaired kidney function in the short term.

Keywords: Diabetes complications, Donor follow-up, Kidney, Live donor transplantation.

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Kidney transplantation, particularly from a living donor, is the treatment of choice for most patients with end-stage renal disease (ESRD). Epidemiological studies of living donors suggest that their lifetime risk of developing ESRD is minimal (*1–4*). These excellent outcomes are believed to result in some part from careful predonation evaluation and conservative living donor screening practices. However, nearly a quarter of all living kidney donors in the current era are considered medically complex, and more than a fifth have abnormal fasting glucose (*5*). There remain substantial concerns about the acceptability of living donors with medical risk factors for future kidney disease. Most large studies examining outcomes in live kidney donors have not specifically examined subsets with medical complexity, and the long-term consequences of donation in these groups remain unclear (*5, 6*).

The American Diabetes Association describes impaired fasting glucose (IFG) as an intermediate state of hyperglycemia in which glucose levels do not meet criteria for diabetes but are too high to be considered normal (*7*). It defines IFG as a fasting plasma glucose of 100 to 125 mg/dL. The term *prediabetes* is applied in the setting of impaired glucose tolerance or IFG and indicates a higher than normal risk of progression to type 2 diabetes mellitus (DM). By current estimates, one third of the adult population in the United States is prediabetic, and the prevalence is rising (*8*). The detection of abnormal glucose metabolism in a prospective living kidney donor raises concerns about the development of DM (*9–11*) and potentially kidney disease in the future. Transplant societies advise caution in the evaluation and counseling of such prospective donors but have been unable to generate unequivocal practice guidelines in the absence of sound evidence (*12–15*). As a result, potential donors with abnormal glucose metabolism are often discouraged from donating (*16*).

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A strategy of excluding all prediabetic individuals from donation may be unnecessarily restrictive and contribute to the diminution of an already limited pool of living kidney donors. On the other hand, increased acceptance of medically complex donors has brought into question the applicability of previous living donor outcome studies to current practices. The aims of this study were to compare kidney outcomes in living kidney donors with IFG at the time of donation to those in donors with normal fasting glucose.

RESULTS

Predonation Characteristics

Forty-five living kidney donors with IFG and 45 matched controls with normal fasting glucose were enrolled. Compared with the 98 IFG donors who did not participate in the study, the 45 IFG donors who enrolled were older at the time of donation (mean age, 47.1±11.5 vs. 42.8±10.8 yr, P=0.038) but similar in other predonation characteristics including sex, ethnicity, relation to the recipient, body mass index (BMI), systolic and diastolic blood pressures, and mean fasting plasma glucose (Table 1).

Demographic characteristics and predonation medical history and laboratory data for IFG and normal glucose control donors who participated in the study are listed in Table 2. Mean fasting plasma glucose, BMI, and blood pressures before donation were significantly higher in IFG donors as compared with normal glucose controls. Other predonation characteristics and MDRD eGFR at 30 days postdonation were similar between the two groups.

Development of DM and Hypertension

Mean follow-up times postdonation in the IFG and control group were 10.4±3.17 years (range, 5.1–19.5 yr) and 10.0±3.11 years (range, 5.0–15.8 yr), respectively. Based on the questionnaire and laboratory data, seven IFG donors (15.6%) and one control donor (2.2%) had developed DM, yielding a relative risk estimate for IFG versus normal glucose of 7.0 (95% CI, 0.9–54.6, P=0.06) for developing DM. All diabetics were aware of their diagnosis. Of the seven IFG donors who became diabetic, two were being managed by diet alone, and the others were on oral medications; none was taking insulin. Of the remaining 38 IFG donors, 12 (26.7%) continued to have IFG, whereas 26 (57.8%) had normal fasting plasma glucose values at follow-up. In contrast, of the remaining 44 control donors, 2 (4.4%) had developed IFG, and 42 (93.3%) continued to have normal fasting glucose at follow-up.

Impaired fasting glucose donors who developed DM or continued to have IFG were found to have no significant differences in their predonation characteristics when compared with those IFG donors who had reverted to normal fasting plasma glucose (Table 3). Of those who developed DM, 4 (57.1%) of 7 had a fasting plasma glucose greater than 110 mg/dL versus 6 (23.1%) of 26 of those who reverted to normal fasting glucose. However, although a higher proportion of those with predonation fasting plasma

TABLE 1. Predonation characteristics of all identified IFG donors classified by enrollment in the study

Predonation characteristic	IFG donors who enrolled N=45	IFG donors who did not enroll N=98	*P*
Male sex (%)	19 (42.2)	53 (54.1)	0.19
Age (yr)	47.1±11.5	42.8±10.8	0.038
Ethnicity (%)			0.21
White	30 (66.7)	46 (46.9)	
African American	2 (4.4)	8 (8.2)	
Asian	6 (13.3)	13 (13.3)	
Hispanic	6 (13.3)	27 (27.6)	
Pacific Islander	1 (2.2)	4 (4.1)	
Relation to the recipient (%)			0.37
Unrelated	19 (42.2)	30 (30.6)	
Related	26 (57.8)	68 (69.4)	
Family history of diabetes mellitus (%)			
Type I	N=45	N=52	0.34
	5 (11.1)	3 (3.1)	
Type II	N=45	N=60	<0.0001
	15 (33.3)	43 (43.9)	
Body mass index (kg/m^2)	28.1±3.95	27.8±4.08	0.65
Blood pressure (mm Hg)	N=43	N=85	
Systolic	130±14.6	126±13.4	0.23
Diastolic	75.6±9.61	74.9±9.43	0.75
Fasting plasma glucose (mg/dL)	109±9.39	108±11.4	0.36
2 hour glucose on oral glucose tolerance test (mg/dL)	n=16	n=54	0.21
	110±19.5	106±25.1	
MDRD eGFR 30 d postdonation (mL/min/1.73 m^2)	60.8±10.8	63.8±15.4	0.41

TABLE 2. Predonation characteristics of IFG and control donors

Predonation characteristic	IFG donors N=45	Control donors N=45	*P*
Male sex (%)	19 (42.2)	20 (44.4)	0.83
Age (yr)	47.1±11.5	47.5±10.4	0.81
Ethnicity (%)			0.61
Caucasian	30 (66.7)	26 (57.8)	
African American	2 (4.4)	2 (4.4)	
Asian	6 (13.3)	4 (8.9)	
Hispanic	6 (13.3)	12 (26.7)	
Pacific Islander	1 (2.2)	1 (2.2)	
Relation to the recipient (%)			0.33
Unrelated	19 (42.2)	26 (57.8)	
Related	26 (57.8)	19 (42.2)	
Family history of diabetes mellitus (%)			
Type I	5 (11.1)	2 (4.4)	0.24
Type II	15 (33.3)	19 (42.2)	0.38
Body mass index (kg/m^2)	28.1±3.95	25.3±4.54	0.0018
Blood pressure (mm Hg)	n=43	n=40	
Systolic	130±14.6	123±14.8	0.047
Diastolic	75.6±9.61	70.2±10.3	0.025
Fasting plasma glucose (mg/dL)	109±9.39	87.1±7.05	<0.0001
2-hr glucose on oral glucose tolerance test (mg/dL)	N=16	N=13	0.0003
	110±19.5	83.9±13.5	
MDRD eGFR 30 d postdonation (mL/min/1.73 m^2)	60.8±10.8	59.3±11	0.41

glucose greater than 110 mg/dL developed DM as compared with staying IFG or reverting to normal fasting glucose (28.57% vs. 9.68%), this difference was not statistically significant (Fisher's exact test, two-tailed *P*=0.18). Additionally, in a logistic regression analysis, the level of predonation fasting plasma glucose did not predict the

TABLE 3. Predonation characteristics of IFG donors subdivided according to current fasting plasma glucose

Variable	Normal fasting plasma glucose (<100 mg/dL) n=26	Impaired fasting plasma glucose (100–125 mg/dL) n=12	Diabetes (≥126 mg/dL) n=7	*P*
Male sex (%)	8 (30.8%)	7 (58.3%)	4 (57.1%)	0.19
Age at donation (yr)	46.7±12.2	50.4±11.9	43.1±7.2	0.25
Ethnicity (%)				0.45
White	18 (69.2)	7 (58.3)	5 (71.4)	
African American	1 (3.8)	1 (8.3)	0	
Asian	5 (19.2)	0	1 (14.3)	
Hispanic	2 (7.7)	3 (25)	1 (14.3)	
Pacific Islander	0	1 (8.3)	0	
Relation to the recipient (%)				0.13
Unrelated	10 (38.5%)	7 (58.3%)	2 (28.6%)	
Related	16 (61.5%)	5 (41.7%)	5 (71.4%)	
Family history of DM (%)				
Type I	4 (15.4%)	1 (8.3%)	0 (0.0%)	0.48
Type II	8 (30.8%)	5 (41.7%)	2 (28.6%)	0.77
Predonation BMI (kg/m^2)	28±4.14	27.4±3.98	29.8±3.11	0.41
Predonation BP (mm Hg)				
Systolic	130±16.2	133±11	124±14.6	0.44
Diastolic	74.8±11.3	77.3±7.29	75.4±7.16	0.62
Predonation fasting plasma glucose (mg/dL)	109±11.4	109±7.06	110±3.78	0.35
MDRD eGFR 30 d postdonation (mL/min/1.73 m^2)	63.6±11	55.9±10.1	58.6±8.7	0.09

TABLE 4. Current health status of IFG and control donors

Variable	IFG donors n=45	Control donors n=45	*P*
Age (yr)	57.4±12.1	57.4±10.9	0.90
Time since donation (yr)	10.4±3.2	10.0±3.1	0.65
Development of diabetes (%)	7 (15.56%)	1 (2.2%)	0.06
Development of hypertension (%)	16 (35.6%)	10 (22.2%)	0.16
Body mass index (kg/m^2)	27.9±4.86	25.9±5.09	0.048
Fasting plasma glucose (mg/dL)	104.7±33.2	90.0±6.5	0.0045
Hemoglobin A1c (%)	5.97±1.26	5.58±0.233	0.027
Cholesterol (mg/dL)	190±36.3	199±38.3	0.11
Triglycerides (mg/dL)	131±81.4	111±55.3	0.35
LDL (mg/dL)	106±29	113±30.8	0.19
HDL (mg/dL)	60.3±23	63.6±25.3	0.54
MDRD eGFR (mL/min/1.73 m^2)	70.7±16.1	67.3±16.6	0.21
Albumin/ creatinine ratio (mg/g)			
Mean	9.76±23.6	5.91±11	0.29
Median	3 (0.0–150)	2 (0.0–54)	

development of DM versus the remaining IFG or reverting to normal glucose (odds ratio 1.008 per 10 mg/dL change in plasma glucose; 95% CI, 0.929–1.093, *P*=0.85). Hemoglobin A1c values were available only for a subset of donors and were not statistically different between IFG donors (5.46±0.38%, n=20) and control donors (5.50±0.32%, n=12; *P* =0.40). Other predonation characteristics such as age, sex, race, whether the donor was related to the recipient, family history of DM, and body mass index also did not predict the development of DM. We also examined the mean change in BMI from predonation to follow-up in the IFG donors who developed DM compared with those who reverted to normoglycemia and found no significant difference (–1.24±2.97 kg/m^2 vs. –0.15±3.44 kg/m^2 respectively; *P*=0.49). Interestingly, of the seven patients who developed diabetes in the IFG group, five patients had lost weight and had a decrease in their BMI (range, –0.25 to –5.54 kg/m^2), and two had only a modest increase (+1.86 and +2.49 kg/m^2). On the other hand, the single donor who had developed DM in the control group had the highest gain in BMI (+10.6 kg/m^2) of all the study participants.

Medical history was positive for a diagnosis of hypertension since donation in 35.6% of IFG donors and 22.2% of healthy controls; this difference was not statistically significant (*P*=0.16).

GFR and Albuminuria

Table 4 shows the health status of IFG and control donors at follow-up. Mean MDRD eGFR (70.7±16.1 ml/min/1.73 m^2 vs. 67.3±16.6 ml/min/1.73 m^2, *P*=0.21) and albumin excretion at follow-up (urine ACR 9.76±23.6 mg/g vs. 5.91±11 mg/g, *P*=0.29) were similar in IFG and healthy glucose donors. Microalbuminuria (urine ACR, 30–300 mg/g) was seen in three IFG and three healthy glucose donors. None of the donors had ACR greater than 150 mg/g. Of the three IFG donors with microalbuminuria, two were diabetic. Mean MDRD eGFR and the presence or level of albuminuria were not significantly different in those who developed DM versus those who remained impaired or regained normal fasting glucose (Table 5). In addition, we did not find a significant correlation between the change in BMI following donation and the level of albuminuria or

TABLE 5. Current health status of IFG donors subdivided by current fasting plasma glucose

Variable	Normal fasting plasma glucose (<100 mg/dL) n=26	Impaired fasting plasma glucose (100–125 mg/dL) n=12	Diabetes (≥126 mg/dL) n=7	*P*
Current age (yr)	57.5±12.5	60.6±12.4	51.6±8.28	0.16
Time since donation (yr)	11.15±3.56	9.97±2.77	8.63±1.92	0.20
Current BMI (kg/m^2)	27.8±5.52	27.8±4	28.5±4.02	0.82
Hemoglobin A1c (%)	5.63±0.253	5.63±0.352	7.77±2.59	0.0006
Cholesterol (mg/dL)	195±33.1	178±30.9	191±53.9	0.32
Triglycerides (mg/dL)	110±52.3	118±55.2	229±135	0.028
LDL (mg/dL)	111±24.8	92.6±21.4	112±50.6	0.12
HDL (mg/dL)	62.4±24.2	64.2±23.8	45.7±9.27	0.09
MDRD eGFR (mL/min/1.73 m^2)	72.1±17.6	64.5±12.4	76.3±14	0.25
Albumin/creatinine ratio (mg/g)				
Mean	6.85±10.7	4.01±4.16	30.4±54.6	0.70
Median	3 (0–50)	3 (0–13)	11 (0–150)	

eGFR at follow-up in IFG donors (Spearman rank correlation 0.26 for urine ACR; 95% CI, –0.05 to 0.51, *P*=0.08; and 0.15 for MDRD eGFR; 95% CI, –0.15 to 0.43, *P*=0.32).

Survival and Risk of ESRD in IFG Donors

As of March 1, 2013, approximately 84 IFG donors were documented to be alive, and 4 were documented as deceased. The causes of death listed in the NDI were hypertensive heart disease, megacolon, and homicide by handgun discharge in three donors who died at the ages of 73, 52, and 51 years, respectively. The cause of death for one donor, listed in the SSDMF as having died at the age of 76 years, was not found in the NDI. The remaining 55 IFG donors were unable to be contacted, but they were not listed as deceased in the Social Security Death Master File or in the National Death Index. ESRD requiring dialysis or transplantation had not developed in any IFG donor to date.

DISCUSSION

Our results indicate that donors with IFG have similarly preserved GFR and low prevalence of albuminuria as donors with normal fasting glucose at a mean of 10 years postdonation. More than half (57.8%) of IFG donors had reverted to normal fasting glucose, and only a minority (15.6%) had developed DM. However, this incidence of DM in IFG donors is much higher compared with healthy controls (2.2%), and although statistical significance was limited by our sample size, the best estimate of increased risk associated with baseline IFG of developing overt diabetes within 10 years of donor nephrectomy is seven-fold compared with normal fasting glucose at the time of nephrectomy.

Concerns about the acceptability of living kidney donor with abnormal glucose metabolism revolve around the perceived risk of developing DM postdonation and potentially diabetic nephropathy with progression to ESRD. Although IFG falls under the category "prediabetes" (*7*), reported estimates of DM development in this group vary widely (9.1%–72.7%) depending on the population studied and the duration of follow-up (*17*). Also, within the population with IFG, it has been observed that there is a continuum of increasing risk for every 1 mg/dL increase in the fasting glucose, with progression to DM occurring more commonly and more rapidly in those with fasting glucose greater than 110 mg/dL versus those with fasting plasma glucose 100 to 110 mg/dL (*18*). We did not find a statistically significant difference in the mean fasting plasma glucose between IFG donors with and without incident diabetes, possibly because of the small sample size. However, more than half (57.1%) of those who progressed had a fasting plasma glucose greater than 110 mg/dL as compared with only 23.1% of those who reverted to normal fasting glucose, so it is possible that given a larger study population, this cutoff might identify a subgroup of prospective IFG donors at higher risk who merit closer evaluation and counseling. It should be noted that, although the 2003 American Diabetes Association Expert Committee report reduced the lower limit of fasting plasma glucose to define IFG from 110 to 100 mg/dL, the World Health Organization and many other diabetes organizations have not adopted this change in the definition of IFG (*19*).

A hemoglobin A1c value of 5.7% to 6.4% corresponds to a fasting plasma glucose greater than 110 mg/dL and can also be used to define prediabetes (*7*). Hemoglobin A1c may even be a superior diagnostic tool, given the greater convenience (because fasting is not required), likely greater preanalytical stability, and less day-to-day perturbations during periods of stress and illness. Unfortunately, predonation hemoglobin A1c was available in fewer than half (44.4%) of the IFG group and in only 2 of those who later developed DM (values of 5.1% and 5.5%) so that we could not perform any meaningful analysis of it as a predictive variable. Similarly, the 2-hr oral glucose tolerance test had been performed in only a small minority of IFG donors before donation. Given its endorsement by the American Diabetes Association as a diagnostic parameter for DM and prediabetes, it is important that future studies explore the role of hemoglobin A1c in the screening of potential living kidney donors.

Additionally, we did not find an association between the family history of DM or BMI (whether predonation or at follow-up) and the later development of DM. Other known risk factors for the development of DM besides family history and BMI or adherence to appropriate lifestyle measures were not studied.

Our results do not indicate reduced kidney function in IFG donors who developed DM. In these donors, one concern is that compensatory hyperfiltration from donating a kidney will combine with the hyperfiltration observed in DM and lead to a rapidly progressive deterioration of kidney function. A few animal studies have indicated faster progression of diabetic nephropathy in the setting of renal mass reduction (*20–25*). However, small studies comparing diabetic humans with one or two kidneys have not demonstrated differences in GFR or histologic changes of diabetic nephropathy (*26–28*). In a retrospective review of 71 Japanese living kidney donors with impaired glucose tolerance at the time of donation, no increase in the incidence of ESRD or mortality was seen, but no information was provided on their level of kidney function or protein excretion (*29*). Another study found higher rates of albuminuria in living kidney donors who subsequently developed DM versus nondiabetic donors, but no excess risk for accelerated kidney disease was seen in the first decade of DM development (*30*).

In our study, the mean time from donation to follow-up in those IFG donors who had developed DM was 7.6 years. It takes many years to develop diabetic kidney changes, and our conclusions are limited to the first 5 years after DM development. Also, the higher level of MDRD eGFR seen in diabetic donors, although statistically not significant, may represent early hyperfiltration, and it is possible that later reduction in GFR will occur. Therefore, longer follow-up is certainly needed.

Our study has a few limitations. The most important of these is the small sample size. As a retrospective study, which included only a small number of donors with available contact information, our analysis is subject to response bias. On the other hand, it is reassuring that except for being slightly older, IFG donors who participated in the study seem to have similar predonation characteristics as those who did not enroll. Another limitation pertains to the racial

and ethnic composition of our donor population. Although more than a third of our donors belong to minority populations, the majority is white, and because of the small sample size, our results may not be generalizable to minority donors with IFG, particularly African Americans (only two in each group). We used the MDRD study equation to estimate the GFR for the comparison of current and baseline rates; however, this formula was developed in people with two kidneys and a GFR of less than 60 mL per minute per 1.73 m^2, so the usefulness of the equation in kidney donors with GFR around or above 60 m/min may be limited. Serum creatinine measurements before or 30 days postdonation were not calibrated, a factor that may have resulted in imprecise estimates of the change in the estimated GFR after donation. Although the outcomes are reassuring, the relatively short duration of follow-up in our study limits the conclusions we can draw about long-term kidney survival and safety in IFG donors.

Another significant limitation of our study is the effect of selection bias on our results. Our study lacks an effective control group, which would ideally consist of prospective donors with IFG who were accepted as donors but did not donate a kidney for nonmedical reasons. Such a control group would have allowed an assessment of the risk attributable to donation itself and adjust for selection bias, as kidney donors are carefully screened and are healthier, on average, than the general population. Therefore, it is possible that the population we studied is not representative of the general population with prediabetes.

In conclusion, our study demonstrates that living kidney donors with IFG developed DM at a much higher rate than matched controls, but the majority remained with IFG or reverted to normal fasting glucose. Impaired fasting glucose donors in our study had a preserved GFR, and their rates of albuminuria were similar to those of matched controls at 10 years. They did not have an excessive risk of ESRD. Although these results are encouraging, our optimism is tempered with caution because of the limited sample size and short duration of follow-up. Although limited in its ability to draw firm conclusions about the long-term safety of kidney donation in this group, this is the first objective report of renal outcomes in a set of prediabetic living kidney donors and will hopefully lead to prospective long-term studies in this growing population of potential donors.

MATERIALS AND METHODS

Study Population

From January 1, 1994, through December 31, 2007, approximately 1423 living donor nephrectomies were performed at the University of California–San Francisco Medical Center. Donors provided a complete medical history, underwent a physical examination, and were subject to a comprehensive laboratory assessment to rule out kidney disease, systemic illnesses, or active infections. Potential donors were required to have a creatinine clearance greater than 80 mL/minute to be eligible for donation. No potential donor with significant albuminuria (defined as a urinary albumin/ creatinine ratio (ACR) >30 mg/g) was accepted.

An electronic database containing the laboratory information on donors was queried to identify donors who had at least one fasting plasma glucose value greater than 100 mg/dL (IFG) before donation. Approximately 143 such donors were identified; 82 donors had only one fasting glucose measurement. Of the remaining 61 donors who had had more than one fasting glucose measurement, the higher value was used to determine inclusion in the study and for statistical analysis.

Data Collection

We attempted to contact donors using telephone numbers listed in the medical chart at the time of donation. Donors who were successfully contacted by telephone and provided written informed consent were administered a medical history questionnaire by phone. Study participants were mailed a laboratory order form and asked to submit a fasting morning blood and urine sample to a local Quest laboratory for analysis. Serum samples were analyzed for electrolytes, blood urea nitrogen, creatinine, glucose, albumin, total protein, transaminases, alkaline phosphatase, total bilirubin, total cholesterol, lipid fractions, and hemoglobin A1C. A single spot urine specimen was analyzed for creatinine and albumin. Predonation clinical and laboratory data were obtained using retrospective chart review.

Up to three potential living kidney donors who had normal fasting plasma glucose at the time of donation matched for age, sex, race or ethnic group, and year of donation to successfully enrolled IFG donors were identified through an electronic database query. For each IFG donor, the controls were approached alphabetically till one control provided consent to participate in the study. No further controls were then contacted for that IFG donor. In this way, 1:1 matched control donors with normal fasting glucose were enrolled in the study.

We ascertained the vital status of all IFG donors as of March 1, 2013, through a search of the Social Security Death Master File as well as the National Death Index. The presence of ESRD was ascertained through reports by the donors themselves and by querying databases of the Organ Procurement and Transplantation Network and the Center for Medicare and Medicaid Services.

The study was approved by the institutional review board at UCSF (CHR 11-07024).

Statistical Analysis

Continuous variables are expressed as mean±SD and were compared using the Mann-Whitney test. Categorical variables are expressed as a percentage and were compared using the χ^2 test. We performed single predictor regression modeling between each of the outcomes (development of DM, development of hypertension, estimated GFR by the Modification of Diet in Renal Disease equation (MDRD eGFR) (*31*) at follow-up, ACR) and each of the following predonation characteristics: age, sex, race, whether the donor was related to the recipient, family history of DM, fasting glucose, body mass index, and MDRD eGFR 30 days postdonation. Linear regression was used for continuous outcomes and logistic regression for binary outcomes. Predonation fasting glucose was analyzed as both a continuous and as a categorical independent variable (≤ or >110 mg/dL). We then repeated the regression modeling but added in age, sex, and race in each of the models as additive covariates. We also performed a Spearman correlation analysis between the change in BMI and the level of albuminuria and eGFR in the control and IFG groups at follow-up. $P<0.05$ were considered statistically significant. The software used for the analysis was SAS Version 9.2.

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