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Measured Glomerular Filtration Rate After Kidney Donation: No Evidence of Accelerated Decay

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Abstract

Background—The rate of measured GFR change in kidney donors years after donation has not been adequately addressed. Whether this change is accelerated in the setting of 1 kidney is also understudied.

Methods—214 randomly selected donors underwent serial GFR measurements of nonradioactive iohexol. eGFR at each visit was calculated using the CKD EPI and MDRD study equations.

Results—GFR visits were 4.8 ± 1.3 years apart and the second occurring 16.9 ± 9.1 years after donation. The majority (97.7%) were White; 60.8% female and 78.5% were related to their recipient. Most, 84.6%, had a GFR ≥ 60 ml/min/1.73m², 14.0% had a GFR between 45–60 ml/min/1.73m² and 1.4% had a GFR < 45 ml/min/1.73m². Between visit 1 and 2, 56.5% had a GFR decline, 36.0% increase, and in 7.5% there was no change. Overall, GFR declined at a rate of -0.42 ml/min/1.73m² per year. Of GFR estimating models, only CKD Epidemiology Collaboration - Creatinine equation produced a slope that was steeper than measured GFR.

Conclusion—Nearly 2 decades postdonation GFR declined at a rate similar to that seen in the general population and in one-third GFR continues to increase.

Introduction

Living kidney donation results in an immediate 50% reduction in renal mass and also glomerular filtration rate (GFR), that subsequently reaches approximately 60–70% of the predonation value.^{1–4} This new GFR appears to remain stable in the ensuing months and

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even years after donation.^{1,5-7} These data have come mainly from the early eras of live donation and have generally utilized less than optimal methods of measuring kidney function such as creatinine clearance. Moreover, we are not aware of studies addressing serial and late changes of measured GFR. Whether kidney donors loss of kidney function with aging is similar to people with 2 kidneys has not been studied utilizing measured GFR either.

We previously reported on measured GFR (mGFR) in a representative cohort of living donors from our institution.⁷ Since our initial report, these same donors were invited to return for a second measurement. Our goals with this analysis were 3-fold: 1) Describe the mGFR change decades following living donor nephrectomy, 2) Assess if the loss of GFR with aging is accelerated in donors, and 3) Evaluate the ability of contemporary GFR estimating models to capture true GFR change over time.

Methods

Study Population

In December 2003, we initiated a comprehensive effort to contact all persons who had donated a kidney after November 1963. We consulted telephone and internet directories and asked recipients for their specific donor's contact information. We asked donors we located to provide us with updates on their health status and to report the results, if available, of urinalysis and serum creatinine testing. At the beginning of this effort in 2003, we generated lists of donors who were known to be alive (as of December 2003) and stratified them according to sex and the number of years since donation (in 3-year intervals). A random starting point within each stratified list was used to generate random numbers to select 5 to 10% of donors from each stratum who would be asked to undergo measurements of the GFR. Eighty percent of the donors who were designated in this way were successfully contacted, and 260 donors underwent measurement of the GFR. If the selected donor refused to participate, the same method was used to contact a new donor from the same stratum.

GFR was determined on the basis of the plasma clearance of nonradioactive iohexol.⁸ We chose this method because it does not require timed urine collections, which may result in incomplete bladder emptying and lead to significant variability in GFR measurement, and in view of its excellent correlation with inulin clearance; the gold standard of measuring GFR. The coefficient of variation (CV) of the iohexol GFR method at our center is consistently < 10%.⁹ On the day of GFR measurement, we also measured albumin to creatinine in an early-morning urine sample. We defined albuminuria by a urinary albumin excretion rate (ACR) >30 mg/g creatinine. Complete details regarding these tests were previously published.¹⁰ Blood pressure was measured 3 times while the donor was seated, and the mean value was recorded. We considered hypertension to be present when a donor required antihypertensive medications. This study was approved by the University of Minnesota Institutional Review Board (HSC #0301M39762 and 0905M66501).

In May 2008, the creatinine assay at University of Minnesota laboratories changed from Jaffé/CXR Synchron method to the IDMS-traceable creatinine. All Jaffé creatinines were converted to IDMS-traceable creatinine using the following formula: IDMS creatinine

[mg/dL] = $-0.111 + 0.964 \times \text{Jaffé creatinine [mg/dL]}$. We have previously shown, in 50 serum samples randomly selected, that this formula provided serum creatinine values identical to the directly measured value and thus we used the values obtained from using the formula for this analysis.¹¹ Creatinine measurements at the time of the second mGFR were all IDMS-traceable. We used the standardized serum Cystatin C traceable to the International Federation of Clinical Chemistry Working Group for standardization of Serum Cystatin C and the Institute for Reference Materials and Measurements certified reference materials.^{12,13} CysC was measured using the PENIA cystatin C kit on a ProSpec nephelometer (Siemens) from blood specimens stored at -70°C as described elsewhere.¹⁴ We evaluated eGFR slope using 4 models: 1) IDMS-traceable re-expressed MDRD study equation, 2) CKD-EPI-Creatinine, 3) CKD-EPI-Cystatin C, and 4) CKD-EPI-Creatinine and Cystatin C equation.

Statistical analysis

Descriptive statistics are expressed as frequencies and percent, or mean $\pm$ standard deviation (SD), as appropriate. For unadjusted analysis, the p value was calculated from one-way ANOVA for continuous variables, and Fisher's exact test for categorical variables. To investigate the change of mGFR and eGFR over time, a linear mixed model was used accounting for within-subject correlation. Slope of GFR change with age and its 95% confidence interval were reported, and compared between different eGFR models. To determine risk factors for albuminuria and hypertension at visit 1, we fitted logistic-regression models using a backward selection method with covariates of age, age at the time of donation, sex, time since donation, systolic blood pressure, diastolic blood pressure, body-mass index, smoking status, and baseline MDRD eGFR. For inclusion in the logistic-regression models, P values of less than 0.10 were considered to indicate statistical significance. Association between ratio of the most recent MDRD eGFR/donation MDRD eGFR were investigated by a linear model adjusting for age at donation and time to GFR from donation. Factors that may affect the association, ie gender, race, BMI at donation, blood pressure at donation and creatinine at donation were examined in the model. All analyses were done using the SAS system (v. 9.3; SAS Institute, Cary, NC). Graphs were plotted in R (<http://www.r-project.org>). P values were two-sided with <0.05 considered statistically significant.

Results

Originally, 260 randomly selected donors underwent iothexol GFR measurement. The donors who completed the iothexol GFR were older than the donors who did not, lived in geographic proximity to the metropolitan Minneapolis area, donated more recently, and a higher proportion were White/non-Hispanic, unrelated to their recipient, and nonsmokers; the 2 groups were otherwise similar (Table 1). These same donors were invited to return in 3-year intervals for serial mGFR measurements. The majority of donors, 214 (82%), returned for a second measurement and constitute our study cohort.

Predonation characteristics of donors with 2 mGFRs are shown in Table 2. The mean age at donation was 40.8 ± 0.7 years, 97.7% White, 60.8% female, 78.5% related to their recipient,

and 23.8% smoked at donation. Their predonation serum creatinine was 0.91 ± 0.14 mg/dL; predonation body mass index (BMI) 26.0 ± 4.1 kg/m². Donors who returned for a second mGFR visit were comparable to those who did not. None of the donors studied died prior to the offering of the second mGFR visit.

Serial mGFR

The second mGFR visit occurred 16.9 ± 9.1 years from donation; 4.8 ± 1.3 years (range 2.1 to 8.5) after visit 1. The average age at visit 2 was 57.7 ± 9.3 years. The mean mGFR at visit 1 was 72.1 ± 11.9 ml/min/1.73m²; 17 (7.9%) donors had a value ≥ 90 ml/min/1.73m², 168 (78.5%) 60–90 ml/min/1.73m², 26 (12.2%) 45–60 ml/min/1.73m², and 3 (1.4%) 30–45 ml/min/1.73m². mGFR at visit 2 was 69.8 ± 11.3 ml/min/1.73m²; 9 (4.2%) donors had a value ≥ 90 ml/min/1.73m², 172 (80.4%) 60–80 ml/min/1.73m², 30 (14.0%) 45–60 ml/min/1.73m², and 3 (1.4%) 30–45 ml/min/1.73m². At neither visit did any donor have an eGFR < 30 ml/min/1.73m² (Table 3).

Figure 1 shows the longitudinally measured glomerular filtration rate by age at study visit after adjusting for sex and age at donation. mGFR declined -0.42 (95% CI $-0.54, -0.31$) ml/min/1.73m² with every increasing year of age, $p < 0.0001$. The individual donor mGFR values are represented as dots with longitudinal values from the same donor connected by a black line. The red dotted line is a smooth spline to show the trend.

Correlates of mGFR change

Between visit 1 and visit 2, 121 (56.5%) donors experienced an mGFR decline, while 77 (36.0%) had an increase and 16 (7.5%) had no change. Unadjusted analysis showed no difference in predonation characteristics between donors with an mGFR decrease, no change, or increase (Table 4). Donors whose mGFR decreased (vs donors with an mGFR increase) had a significantly higher mGFR at visit 1 (75.0 ± 11.3 vs 67.8 ± 11.5 , $p < 0.0001$) and a significantly lower mGFR at visit 2 (67.8 ± 10.9 vs 72.9 ± 11.4 , $p < 0.01$). At visit 1, time since donation was significantly associated with the development of albuminuria (odds ratio, 1.12, 95% CI, 1.05 to 1.20; $P < 0.001$) while albuminuria was less likely to develop in women (odds ratio, 0.31, 95% CI, 0.12 to 0.79; $P = 0.01$). Increasing age was significantly associated with development of hypertension requiring medication (odds ratio per year, 1.09, 95% CI 1.04 to 1.13; $P < 0.001$) as well as increasing body-mass index, per unit (odds ratio, 1.12, 95% CI 1.04 to 1.21; $P = 0.003$). At visit 2, there was no statistically significant difference in the proportion of donors treated for hypertension across the groups: 36.4% of donors with an mGFR decrease, 12.5% of those with no change, and 27.3% of those with an increase, $p = 0.11$. Albuminuria was present in 6.5% of all donors at visit 1 and 2.3% at visit 2; the proportion with albuminuria was not different across the different GFR change groups at either visit (Table 5).

Estimating GFR compensation

The mean eGFR (MDRD) predonation was 84.8 ± 14.1 ml/min/1.73m²; at visit 2 the eGFR was 65.9 ± 12.7 ml/min/1.73m². The ratio of the eGFR at visit 2 compared to predonation (long-term GFR compensation) was 0.79 ± 0.13 (min 0.44, max 1.34). After adjusting for sex, age at donation, predonation serum creatinine, and years from donation, a higher serum

creatinine at donation was associated with greater compensation (estimate +0.05 per every 0.1 mg/dL increase, SE 0.07), $p < 0.0001$. Males (vs females) had less GFR compensation (estimate -0.10, SE 0.02), $p < 0.0001$. Age at donation was not significantly associated (estimate -0.001 per 1 year increase, SE 0.001), $p = 0.18$. Race, BMI, SBP, and DBP were considered in the model but found not to be significant.

Ability of estimating equations to capture GFR change

eGFR at visit 1 and 2 were calculated using the 3 CKD Epidemiology Collaboration and MDRD study equations (Table 6). Adjusting for sex and age at donation, the eGFR slopes using these 4 estimating equations were calculated and are shown in Table 6 and Figure 2. MDRD, CKD-Epi Cystatin C, and CKD-Epi Creatinine and Cystatin C all produced a slope similar to mGFR. Using MDRD, eGFR declined $-0.43 \text{ ml/min/1.73m}^2$ per year of age (95% CI -0.58, -0.28); using CKD-EPI Cystatin C, eGFR declined $-0.43 \text{ ml/min/1.73m}^2$ per year of age (95% CI -0.61, -0.24); using CKD-EPI Creatinine and Cystatin C, eGFR declined $-0.53 \text{ ml/min/1.73m}^2$ per year of age (95% CI -0.67, -0.39). Only the CKD-EPI Creatinine did not produce a slope similar to mGFR; -0.71 (95% CI -0.88, -0.56).

Discussion

In this longitudinal study of living kidney donors many years after donation, we found most had a GFR $\geq 60 \text{ ml/min/1.73m}^2$, prevalence of albuminuria was minimal and GFR declined at a rate similar that observed in the general population. Assessment of common GFR estimating models showed the MDRD study and CKD-Epi equations utilizing Cystatin C alone or in conjunction with creatinine to be suitable tools to capture GFR change in the absence of formal testing.

Many studies have documented hyperfiltration in the early months and years following living donor nephrectomy.¹⁻⁷ In a recent prospective study, mGFR returned to approximately 70% of baseline by 6 months and over the next 3 years, GFR continued to increase at $0.84 \text{ ml/min/1.73m}^2$ per year.⁶ In our previous cross-sectional analysis of this cohort, a longer time from donation was associated with a higher mGFR (slope +0.20 ml/min/yr).⁷ Now, at 17 ± 9 years postdonation and an interim of 5 years between GFR measurements, we again found the majority (85%) had an mGFR $\geq 60 \text{ ml/min/1.73m}^2$ and in 44% mGFR either increased or remained unchanged. While predonation mGFR values were not available, eGFR at visit 2 remained at $79 \pm 13\%$ of baseline function, indicating ongoing long-term compensation. Numerous studies have instead shown an annual improvement, albeit small, in GFR during the first few years after donation (15–20). For example, in an analysis of serial serum creatinine level measurements up to 25 years postdonation ($n = 2002$ donors since 01/01/1990), we modelled eGFR change and showed a small annual increase in eGFR for about the first 15 years postdonation (20). For a typical 55-year-old donor, eGFR then stabilized for a few years; subsequently, it slowly began to fall. For donors < 55 years old, eGFR stabilized after increasing for about 15 years postdonation, and has yet to decrease.

Many have expressed concern for possible long-term hyperfiltration injury as a direct result of living donor nephrectomy. However, decades after donation, we found no evidence for

this. Albuminuria, the hallmark of hyperfiltration damage, was minimal (2.3%) and was not associated with an increasing or decreasing mGFR. Overall, GFR declined an average of $-0.42 \text{ ml/min/1.73m}^2$ per year of age. This rate is similar to what has been reported in the general population although the lack of nondonor controls in our study is certainly a limitation. In the general population GFR declines anywhere from 0.4 to 2.5 ml/min per year of age.^{21–24} The Baltimore Longitudinal Study measured creatinine clearance in men 40–80 years of age without renal disease or hypertension and showed GFR declined at a rate of $-1.51 \text{ ml/min/year}$.²¹ In a more recent longitudinal study of healthy Chinese participants, eGFR (CKD-Epi creatinine) declined by approximately $-1.66 \text{ ml/min/year}$ and the degree of eGFR change significantly increased with age; eGFR in participants aged 45–59 years declined at $-5.5 \pm 11.6\%$ over 5 years; age 60–74 declined $-11.2 \pm 13.8\%$; age 75 declined $-12.1 \pm 12.1\%$.²² The average age of our donors studied was 52 years at visit 1 and 58 years at visit 2; whether a more rapid decline will occur in the ensuing decades remains uncertain. Of some reassurance, a small physiologic study of living donors with a median follow-up of 6.1 years demonstrated the observed hyperfiltration following kidney donation was fully explained by hyperperfusion and renocortical hypertrophy without any component of glomerular hypertension.²⁵

The MDRD and CKD-Epi equations utilizing Cystatin C all produced a slope similar to mGFR. The CKD-Epi creatinine significantly overestimated the slope. While iothexol and iothalamate based methods are considered the ‘gold standard’ for GFR measurement these tests are costly and labor intensive to perform and thus are infrequently used. Unfortunately, performance of the estimating models is quite variable and which model should be used varies depending on age and true GFR.^{14,26} We previously showed CKD-Epi Creatinine Cystatin C to be the most precise of the CKD Epidemiology Collaborations or MDRD. However, it also missed many with an mGFR $< 60 \text{ ml/min/1.73m}^2$ and because of this, the MDRD study equation should be used for detecting GFR below this threshold.¹⁴ In older populations, the CKD Epidemiology Collaboration formulas are less biased and more accurate than the MDRD. However, the MDRD is superior for estimating GFR $< 60 \text{ ml/min/1.73m}^2$.²⁶ This is an important issue as GFR estimating models may misclassify donors as having an eGFR $< 60 \text{ ml/min}$ while in fact their true GFR is higher than 60 ml/min as shown by Tan et al and also from our previous work in this area.^{9,11,14,27} Studies in type 1 and 2 diabetics have also reported poor association between mGFR slope and eGFR slope.^{28,29} Thus the applicability of our findings is limited to healthy living donor population.

There are limitations to our findings: The overwhelming majority of donors were White and further validation in other ethnic groups is needed. However White donors make up 75% of all donors in the United States and thus this information is important for this population.³⁰ The donors studied were generally healthy; nonobese with normal blood pressure and glucose control and whether these results translate to overweight or hypertensive donors is unknown and warrants further investigation.

Conclusions

In a cohort of living kidney donors, nearly 2 decades post donation, mGFR declined at 0.42 ml/min/year. The MDRD or CKD-Epi Cystatin C equations are useful alternative tools for monitoring long-term GFR change in living kidney donors.

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Index words

ACR	urine albumin to creatinine ratio
BMI	body mass index
CKD-EPI	Chronic Kidney Disease Epidemiology Collaboration
CI	confidence interval
Cr	serum creatinine
CV	coefficient of variation
DBP	diastolic blood pressure
eGFR	estimated glomerular filtration rate
GFR	glomerular filtration rate
mGFR	measured glomerular filtration rate
HTN	hypertension
MDRD	modification of Diet in Renal Disease
SD	standard deviation
SE	standard error
SBP	systolic blood pressure

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Figure 1.

Longitudinally measured glomerular filtration rate by age at study visit. mGFR significantly declined -0.42 (95% CI $-0.54, -0.31$) ml/min/1.73m² with every increasing year of age, $p < 0.0001$. The unique mGFR values are represented as dots with longitudinal values from the same donor connected by a black line. The red dotted line is a smooth spline to show the trend. Analysis adjusted for sex and age at donation.

Figure 2. Estimated GFR change with age at study visit. MDRD and CKD-Epi equations utilizing Cystatin C produced a slope similar to mGFR. CKD-Epi creatinine overestimated the slope decline.

Table 1

Predonation characteristics of donors with and without postdonation mGFR.

	Donors with mGFR n=260	Donors without mGFR n=3070	P value
White/Non-Hispanic	97.3%	91.3%	<0.001
Female	61.2%	55.7	0.09
Related to recipient	79.6%	88.1%	<0.001
Age (years)	40.9 ± 11.1	38.0 ± 11.7	<0.001
Serum cr (mg/dL)	0.90 ± 0.14	0.91 ± 0.17	0.31
eGFR MDRD (ml/min/1.73m ²)	85.3 ± 14.7	88.0 ± 17.3	0.02
BMI (kg/m ²)	25.9 ± 4.1	25.6 ± 4.5	0.39
SBP (mmHg)	119.5 ± 13.4	118.6 ± 12.9	0.32
DBP (mmHg)	72.9 ± 9.6	73.1 ± 9.8	0.83
Smoker	25.4%	36.3%	<0.001
Hypertension	2.7%	1.4%	0.11
Years from donation	23.8 ± 9.1	28.8 ± 10.5	<0.0001

BMI, body mass index; diastolic blood pressure; eGFR, estimated glomerular filtration rate; SBP, systolic blood pressure.

Table 2

Characteristics of donors with serial mGFR (n=214).

<u>Predonation</u>	
White	97.7%
Female	60.8%
Related to recipient	78.5%
Age (years)	40.8 ± 10.7
Serum cr (mg/dL)	0.91 ± 0.14
eGFR MDRD (ml/min/1.73m ²)	84.8 ± 14.1
BMI (kg/m ²)	26.0 ± 4.1
SBP (mmHg)	118.6 ± 13.2
DBP (mmHg)	72.7 ± 9.5
Smoker	23.8%
Hypertension	2.3%
<u>First GFR Visit</u>	
Years from donation	12.1 ± 8.9
mGFR (ml/min/1.73m ²)	72.1 ± 11.9
Age (years)	52.9 ± 9.4
Fasting glucose (mg/dL)	91.2 ± 11.7
ACR (mg/g Cr)	18.2 ± 111.1
SBP (mmHg)	122.0 ± 14.5
BMI (kg/m ²)	28.2 ± 4.9
Diabetes	2.8%
Hypertension	22.4%
Albuminuria	6.5%

ACR, albumin creatinine ratio; BMI, body mass index; diastolic blood pressure; eGFR, estimated glomerular filtration rate; mGFR, measured glomerular filtration rate; SBP, systolic blood pressure.

Table 3

mGFR at visit 1 and visit 2.

mGFR ml/min/1.73m ²	Visit 1	Visit 2
Mean ± SD	72.1 ± 11.9	69.8 ± 11.3
90	17 (7.9%)	9 (4.2%)
60 to 90	168 (78.5%)	172 (80.4%)
45 to 60	26 (12.2%)	30 (14.0%)
30 to 45	3 (1.4%)	3 (1.4%)
<30	0	0

Table 4

Predonation characteristics by mGFR change from visit 1 to visit 2.

	<i>Decrease n= 121</i>	<i>No Change n= 16</i>	<i>Increase n= 77</i>	P value
Female	63.6%	62.5%	55.8%	0.55
Age (years)	40.9 ± 11.2	39.7 ± 9.1	40.9 ± 10.1	0.90
White	97.5%	100.0%	97.4%	0.73
BMI (kg/m ²)	25.9 ± 3.8	25.1 ± 2.8	26.3 ± 4.8	0.51
SBP (mmHg)	119.5 ± 13.4	113.0 ± 10.4	118.5 ± 13.3	0.18
DBP (mmHg)	72.5 ± 9.6	70.2 ± 8.7	73.5 ± 9.4	0.43
Serum Cr (mg/dL)	0.89 ± 0.1	0.89 ± 0.1	0.93 ± 0.2	0.23
MDRD eGFR	85.3 ± 14.0	85.9 ± 13.7	83.9 ± 14.5	0.77

BMI, body mass index; diastolic blood pressure; eGFR, estimated glomerular filtration rate; MDRD, Modification of Diet in Renal Disease Study equation; SBP, systolic blood pressure.

Table 5

Characteristics at second visit by mGFR change from visit 1 to visit 2.

	<i>Decrease n= 121</i>	<i>No Change n= 16</i>	<i>Increase n= 77</i>	P etc.value
Years from donation	17.2 ± 9.4	20.5 ± 10.4	15.6 ± 8.3	0.13
Age (years)	58.1 ± 8.9	60.1 ± 10.8	56.5 ± 9.5	0.27
mGFR (ml/min/1.73m ²)	67.8 ± 10.9	70.1 ± 12.1	72.9 ± 11.4	< 0.01
BMI (kg/m ²)	29.0 ± 4.7	28.1 ± 4.2	28.3 ± 5.7	0.52
SBP (mmHg)	124.4 ± 14.1	121.0 ± 14.9	121.9 ± 14.7	0.40
DBP (mmHg)	73.2 ± 8.8	69.6 ± 9.6	72.1 ± 8.9	0.26
Serum cr	1.01 ± 0.2	1.00 ± 0.2	1.02 ± 0.2	0.93
Cystatin C	0.96 ± 0.2	1.02 ± 0.2	0.94 ± 0.2	0.35
Serum glucose	88.2 ± 13.8	87.6 ± 12.7	87.1 ± 11.6	0.85
Diabetes	8.3%	12.5%	2.6%	0.13
Hypertension	36.4%	12.5%	27.3%	0.11
Albuminuria	3.3%	6.3%	0%	0.10
ACR (mg/gCr)	5.7 ± 15.2	6.1 ± 12.1	3.2 ± 2.8	0.35
Mean ± SD (range)	(2.0 to 100.00)	(2.0 to 48.7)	(1.6 to 11.6)	

BMI, body mass index; DBP, diastolic blood pressure; mGFR, measured glomerular filtration rate; SBP, systolic blood pressure.

Table 6

mGFR and eGFR change by age at visit.

GFR model	Visit 1		Visit 2		Slope (95% CI)	SE
	Mean (SD); Median (IQR)	Mean (SD); Median (IQR)	Mean (SD); Median (IQR)	Mean (SD); Median (IQR)		
mGFR	72.1 (11.9); 71.0 (64.0, 81.0)	69.8 (11.3) 68.5 (62.0, 78.0)			-0.42 (-0.54, -0.31)	0.06
CKD-EPI Cystatin C	78.8 (17.1) 78.5 (66.0, 92.5)	82.4 (18.1) 82.8 (69.6, 96.5)			-0.43 (-0.61, -0.24)	0.09
CKD-EPI Cr cystatin C	78.1 (14.8) 77.7 (68.6, 88.3)	76.2 (14.7) 75.8 (67.4, 86.9)			-0.53 (-0.67, -0.39)	0.07
CKD-EPI Cr	78.7 (16.1) 78.0 (66.6, 89.6)	71.4 (14.4) 69.6 (61.0, 82.4)			-0.71 (-0.88, -0.56)*	0.08
MDRD	71.8 (14.7) 69.9 (61.2, 82.0)	65.9 (12.7) 64.5 (56.8, 74.5)			-0.43 (-0.58, -0.28)	0.08

* Significantly different compared to mGFR (P<0.05). Slope is adjusted for sex and age at donation.

CI, confidence interval; CKD-EPI, Chronic Kidney Disease Epidemiology Collaboration equation; mGFR, measured glomerular filtration rate; SE, standard error; SD, standard deviation.