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Income Level and Kidney Disease Severity and Progression Among Children and Adolescents With CKD: A Report From the Chronic Kidney Disease in Children (CKiD) Study

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Abstract

Background—Among adults, lower socioeconomic status (SES) is a risk factor for chronic kidney disease (CKD), progression to end stage renal disease, and poor health outcomes, but the effect among young people with CKD is not well known.

Study Design—Prospective cohort study.

Settings & Participants—572 children and adolescents aged 1 to 16 years with mild to moderate CKD residing in the United States and Canada who were enrolled in the Chronic Kidney Disease in Children (CKiD) Study, a multicenter prospective cohort.

Predictor—Self-reported annual household income category as a proxy measure for SES: ≥ $75,000 (high income), $30,000 to <$75,000 (middle income) and<$30,000 (low income).

Outcomes & Measurements—Clinical characteristics and CKD severity at baseline (GFR; comorbidities related to disease severity and management) and longitudinally (GFR decline; changes in blood pressure *z* scores and height *z* scores per year).

Results—At baseline, low and middle household incomes, compared to high income, were associated with minority race (39% and 20% vs. 7%), lower maternal education (28% and 5% vs.

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1%), abnormal birth history (34% and 32% vs. 21%), and having at least one clinical comorbidity (66% and 64% vs. 55%).Baseline median GFRs were similar across income categories(between 43 and 45 ml/min/1.73m^2). After adjusting for baseline differences, the average GFR declines per year for the high, middle and low income categories were −1.9%, −2.7%, and −2.3%, respectively, and were not statistically significantly different between groups. Blood pressure control tended to improve in all groups (*z* score, between −0.10 and −0.04), but this was not associated with income. Height deficits diminished over time for subjects from high income families but not among subjects from low income families(z scores for height per year, 0.05 and −0.004, respectively; $P = 0.03$ for comparison of high and low income).

Limitations—Statistical power to detect associations by income level is limited; income is an imperfect measure for SES; CKiD participants are not representative of children and adolescents with CKD who are uninsured or not receiving care.

Conclusions—GFR decline and blood pressure control were comparable across income groups. Children and adolescents with CKD from lower income households are at higher risk of impaired growth.

Keywords

socioeconomic status; children; adolescents; chronic kidney disease; progression; growth

Low socioeconomic status (SES) is associated with poor outcomes in a variety of health domains, including chronic kidney disease (CKD).[1–3] Previous studies have shown that low SES is independently associated with poor end-stage renal disease (ESRD) management, longer wait times for kidney transplantation, and ESRD complications among children with CKD.[4] This may be at least partially explained by financial and societal burdens on a family with a child with CKD, particularly among low income households. Furthermore, limited resources for medications and accessing health care among low income families may lead to increased disease severity and accelerated disease progression. However, how SES level affects disease progression and normal growth and development in young people with CKD prior to ESRD has not been well studied. Understanding the effect of SES on disease progression would help clinicians identify high risk groups and target resources towards potential interventions to delay the onset of ESRD.

In this study, we sought to determine the impact of individual-level SES, as measured by parent-reported household income, on CKD disease severity at the time of entry into the Chronic Kidney Disease in Children (CKiD) study. Additionally, using longitudinal follow-up over time, we assessed the effect of low SES on CKD progression, blood pressure (BP) control, and growth. For the cross sectional analysis at study entry, we hypothesized that lower SES is associated with higher disease severity, defined as a higher number of comorbid conditions (hypertension, anemia, abnormalities of bone-mineral metabolism and growth). For the longitudinal analysis, we hypothesized that children and adolescents with CKD from families with lower SES have more rapid GFR decline, worse BP control, and poor linear growth.

METHODS

Study Design and Population

The CKiD study is a multicenter cohort study based in the United States and Canada designed to investigate and describe the natural history and progression of CKD in children and adolescents. Between April 2005 and September 2009, a total of 586 children and adolescents with mild to moderate CKD were enrolled at 48 pediatric nephrology centers, of which 572 had available income data. Details of the CKiD study design have been

previously described.[5] In brief, eligible participants were between the ages of 1 and 16 years, with a diagnosis of CKD and an estimated GFR of between 30 and 90 ml/min/1.73m^2 using the Schwartz formula.[6] Data collection visits occurred annually after study entry (baseline). All protocols and study design were approved by institutional review boards at each participating site. All participants and their families enrolled in the study provided informed consent.

Income as a Proxy for SES

Household income information for each subject was collected at baseline and used in this analysis as the primary exposure and a proxy measurement for SES. Since the CKiD study includes children and adolescents from both the United States and Canada, and low income is a risk factor for poor health outcomes in both countries[7], we assumed equivalence in currency between the two countries. Categories of income were defined as household incomes ≥$75,000 (high income), $30,000 to <$75,000 (middle income) and <$30,000 (low income).As a sensitivity analysis, income was also investigated as a continuous variable and the inferences remained unchanged.

Clinical Characteristics and Comorbidities at Baseline

Demographic, clinical characteristics and comorbid conditions were analyzed at baseline. Clinical characteristics included birth history (premature birth, small for gestational age or low birth weight), underlying diagnosis of CKD (glomerular or non-glomerular cause), age at diagnosis, and urine protein-creatinine ratio (PCR; mg of protein per 1 mg of creatinine). Comorbidities were selected as conditions of particular clinical importance for treatment and management of CKD. These comorbidities were uncontrolled hypertension (systolic BP [SBP] or diastolic BP [DBP]>95th percentile based on age, sex, and height)[8], anemia (defined by sex- and age-defined abnormally low hemoglobin levels)[9], elevated calcium-phosphorus product (i.e., ≥65 mg^2/dl^2 for<12 years of age and ≥55 mg^2/dl^2 for ≥12 years)[10], hyperphosphatemia (age-defined abnormally high phosphate level)[10], and growth failure (i.e., height <3rd percentile). The cumulative presence of these comorbidities (i.e., ≥1, ≥2, and ≥3 comorbidities) were also included as variables of interest. All laboratory measurements were obtained from the CKiD central biochemistry laboratory (University of Rochester), except for hemoglobin which was obtained from the local clinical site laboratory.

Longitudinal Markers of Disease Progression

All markers of disease progression (GFR, BP and height) were measured annually. Direct measurements of GFR were obtained at baseline, one year later, the nat every other annual visit by plasma disappearance of iohexol.[11] When visits did not include an iohexol-based GFR, estimated GFR (eGFR) by the validated CKiD eGFR equation[12] was used in its place (throughout this article, this combination is referred to as “GFR”), using a valid approach for CKiD data to characterize GFR decline.[13] BP was measured by aneroid sphygmomanometry three times at each visit and averaged as the final BP value. The SBP and DBP values were converted to *z* scores based on the normal population for age, gender and height.[8] Height was determined at each annual visit as the average of three measurements by stadiometer and converted to z-scores based on the normal population, standardized to age and gender.[14] BP z-scores and height z-scores are defined as standard deviation units in the normal population.

Statistical Analyses

For cross-sectional analyses, demographics, clinical characteristics and comorbidities were described by medians and interquartile ranges or percentages. Wilcoxon rank-sum and

Fisher's exact tests were used to determine differences by SES categories for continuous and categorical variables, respectively. There were two components to the longitudinal analyses. First, using all available data for each subject, individual linear regressions empirically estimated the baseline level and the annual change in the four outcomes (GFR, SBP, DBP, and height z-scores). These regressions used time (in years) from study entry as the continuous independent variable and each subject contributed an intercept (baseline level) and slope (change), described in Table 2. GFR was log-transformed to stabilize variance; BP and height z-scores were not transformed. For GFR in the log-scale, the average change per year was described as a percent difference, and calculated as follows: (exp(slope) − 1)*100. A similar approach with CKiD data has been previously published.[13]

The second component of the longitudinal analysis was to estimate the effect of income on baseline levels and change over time of the dependent variables in a unified setting, with adjustment for confounders. Linear mixed models were used for each outcome (GFR in the log scale) with random intercepts and slopes for each subject. The models included time (in years from study entry) and income categories as fixed effects, interaction terms with time and income categories, and adjustment for confounders. These confounders were age, sex, race, Hispanic ethnicity, type of CKD diagnosis (classified as either an underlying glomerular or non-glomerular cause, as previously published[15]), and PCR at baseline in the log scale. Interaction terms between confounders and time were evaluated by step-wise inclusion, and included if the model fit was improved, using Aikaike's information criterion. For the BP and height z-score models, GFR at baseline was also included as a confounder (centered at 45 ml/min/1.73m^2). All models included continuous independent variables centered on the median: age at 11 years and PCR at 0.4. In assessing the effect of income on height, abnormal birth history (i.e., parental reported premature birth, small gestational age or low birth weight) was also considered a confounder, since these subjects were expected to have a significant deficit in height and different growth trajectories than those without an abnormal birth history.

The adjusted mean baseline values were estimated for each level of income (with 95% confidence intervals [CIs]). The estimated baseline values were interpreted as the expected level at study entry for a hypothetical average subject: 11 year old non-black, non-Hispanic male with a non-glomerular diagnosis and average disease severity (GFR, 45 ml/min/1.73m^2; PCR, 0.4 mg/mg). Adjusted baseline values were compared across income categories (high income as the reference). The average change over time for each SES category was estimated with the null hypothesis that the average change per year was equal to 0. Pair-wise contrasts compared the effect of time across income categories (i.e., the null hypothesis is the average change per year in lower SES category is equal to the average change per year in the highest income group).

Statistical significance was defined by $P<0.05$. All statistical analyses were performed with SAS 9.2 statistical software (SAS Institute Inc., Cary, NC).

RESULTS

Baseline Characteristics

Table 1 describes the demographic and clinical characteristics stratified by income. Children and adolescents from lower income families were younger at study entry (median age, 10 vs. 12 years), were less likely to be male(58% vs. 68%)and were more likely to be of black race (39% vs. 7%) or Hispanic ethnicity (25% vs. 6%) compared to those from high income families. Maternal education was strongly related to income: only 1% of mothers in a high income household had less-than-high-school level education versus 28% of those in the lowest income household.

Abnormal birth history (premature birth, small for gestational age or low birth weight) was more common among low and middle income households compared to the highest income category (34% and 32% vs. 21%; $P = 0.01$), but BMI did not differ by income category ($P = 0.8$). The proportion of children and adolescents with an underlying glomerular cause of CKD and CKD diagnosed within the first year of life was not different across income categories. Similarly, there were no differences observed in proportions of children and adolescents in each income category treated with anti-hypertensive agents. In investigating co-morbidities at baseline, estimates suggested slightly higher proportions of individuals affected by co-morbid conditions among households with lower income levels, although these associations were non-significant (Table 1). The median GFR level was about the same across income categories (between 43 and 45 ml/min/1.73m^2; $P = 0.3$). BP z-scores ($P = 0.1$ for SBP z-scores; $P = 0.1$ for DBP z-scores) and proteinuria ($P = 0.2$) were not statistically different between groups. For the listed comorbidities, there were also no associations related to income based on logistic regression models adjusting for confounders (results not shown).

Longitudinal Description of Markers of Disease Progression

Table 2 describes the longitudinal data, stratified by household income level. With an average follow-up time of about 3 years, most subjects contributed at least 3 observations. The distribution of observations and follow-up time did not differ by income category (χ^2 $P = 0.7$), indicating that drop-out was not related to income.

Based on individual regressions fit to the data from each subject, the median change in GFR per year was smaller among those with high income (−2.1% per year) compared to those in the middle and low income groups (−3.8% and −3.6% per year, respectively), suggesting accelerated GFR decline in the lower income groups. The correlation between the estimated baseline levels and annual change had a range between −0.06 and 0.12.

Overall, the estimated baseline SBP and DBP z-scores indicated that most subjects had improved BP control over time. Despite lower SBP among the high income group at baseline (z scores, 0.25 vs. 0.51 and 0.37 for the middle and low income groups, respectively), there was a similar z-score decrease for those in the high income group (z score, −0.07 per year) compared to the lower income groups (z scores, −0.06 and −0.05 per year, respectively). A similar effect was observed for DBP. The correlation between baseline level and change was moderately strong and negative: those with higher BP at baseline had a larger negative slope (i.e., those with worse BP control at baseline improved most over time).

The empirically estimated baseline height z-scores demonstrated substantial height deficits in this study population (−0.75 to −0.62). Among the high income group, height z scores increased on average by 0.02 per year. In contrast, among the low income group, height z-scores decreased on average by 0.02 per year.

Adjusted Longitudinal Results From Linear Mixed-Effects Models

Table 3 presents the results from the linear mixed effects models, whereby the data of all individuals were analyzed in a unified setting, in order to estimate and test the baseline levels and changes over time, adjusted by covariates. Baseline levels were estimated for a hypothetical reference subject: an 11 year old non-black, non-Hispanic male, with a non-glomerular diagnosis and average baseline proteinuria (and without an abnormal birth history, for the height z-score model). Based on model fit statistics, the model with GFR included all interactions between confounders and time, but the models with SBP, DBP and height z-scores did not.

The adjusted baseline GFR levels, estimated for the reference group, for the high, middle and low income groups were 44, 40 and 43 ml/min/1.73m^2, respectively. The middle income group was statistically different from the high income group ($P = 0.03$). The adjusted average annual percent changes in GFR for the high, middle and low income categories were −1.9% (95% CI, −4.0% to 0.3%), −2.7% (95% CI, −4.7% to −0.8%), and −2.3% (95% CI, −4.5% to −0.1%), respectively. The declines for the low and middle income groups were significantly different from zero, while the decline in the high income group was borderline significant. Overall, most subjects had substantial disease progression, as measured by GFR. The point estimate for GFR decline was greater among the lower income groups relative to the high income group (−2.7% and −2.3% vs. 1.9%), however these declines were not significantly different ($P = 0.4$ and $P = 0.7$).

The baseline adjusted mean SBP z-scores were not significantly different across income categories. The estimates of mean SBP z-scores were similar between the high income category and the low income category (adjusted means of 0.20 [95% CI, −0.01 to 0.41] and 0.18 [95% CI, −0.03 to 0.39, respectively). The middle income category had higher SBP z-scores (adjusted mean, 0.35; 95% CI, 0.16 to 0.54), but this was not significantly different from the high income category ($P = 0.2$). For the high and middle income categories, there was a significant improvement in SBP z-scores (adjusted means of −0.10 [95% CI, −0.15 to −0.04]and −0.06[95% CI, −0.11 to −0.01], respectively), but not in the low income group (adjusted mean, −0.04; 95% CI, −0.09 to 0.01).

A similar trend was observed for DBP z-scores: the high and middle income groups had significant improvement per year in DBP z-scores, but this was not the case for the low income group. The difference between the change in DBP z-scores per year comparing the low income group and the high income group approached but did not reach significance ($P = 0.05$).

The baseline adjusted mean height z-score for the lowest income category was −0.98 (95% CI, −1.23 to −0.73) and was −0.80 for the highest income category (95% CI, −1.05 to −0.56) and this effect was not significant ($P = 0.2$).There was a significant increase in height z-score per year for the high income category (adjusted mean, 0.05; 95% CI, 0.01–0.08), and height deficits (relative to the normal population) in this group decreased over time. In contrast, the middle income category had a non-significant increase of 0.01 in height z-scores per year (95% CI, −0.02 to 0.03), and the lowest income group had essentially no change per year (adjusted mean, −0.004; 95% CI, −0.03 to 0.03). The difference between the middle and high income group for the change in height z-score per year was borderline significant ($P = 0.07$). The change per year in the low income group was significantly different from that in the high income group ($P = 0.03$).

DISCUSSION

The present analysis of the CKiD cohort describes the differences in comorbidities and disease severity by income categories. Overall, we found that children and adolescents from families with lower income were more likely to be female, black or Hispanic; have low maternal education; lack private health insurance; and have higher rates of abnormal birth history. Additionally, a higher proportion of children and adolescents from low income families had at least 1 comorbid condition. However, for many comorbidities there were no differences by income categories, a reassuring finding in this high risk population. Longitudinally, there was a similar decline in kidney function across all income categories.

While BP baseline levels and changes per year were comparable for each group, it is important to compare baseline levels and expected change. Based on the SBP model, for the

high income category, it would take about 2 years to observe an average SBP at a normal level (i.e., z-score of 0): the average z-score at baseline was 0.20 with an expected decrease per year of 0.10. However, for the low income group, the same model estimates that it would take more than twice as long to achieve a normal SBP level, or 4.5 years, assuming an average decline in z score of 0.04 per year with a starting level of 0.18. Nonetheless, since BP control is strongly related to CKD progression with a direct impact on cardiovascular outcomes[16–18], the observed BP improvement across all groups is encouraging.

This analysis also showed that children and adolescents in the lowest income category were shorter than those in the highest income group at baseline (adjusted height z-scores, −0.98 vs. −0.80).Height deficits present in the highest income group significantly decreased over time, but this effect was not observed for the lower income categories; height deficits in the lower income categories did not improve over time and remained on the same diminished height trajectory.

In a supplementary analysis, we assessed prescribing patterns of growth hormone by income category. It was possible that factors associated with high income (such as access to primary care physicians or ability to afford medications) might lead to increased growth hormone use and account for height improvements in this group. To investigate whether high income was associated with increased growth hormone use, we analyzed data on self-reported growth hormone prescription (ever) and incident growth hormone prescription (after baseline) over the course of follow-up. Those in the lowest income category reported higher ever- and incident growth hormone use (23% and 10%, respectively) relative to the higher income categories (15% and 6% in the middle income group, respectively; and 18% and 6% in the highest income group, respectively). The differences by income categories were not significant for ever growth hormone use($P = 0.1$) or incident growth hormone use ($P = 0.2$), suggesting that prescription of growth hormone therapy was not driving the growth improvements observed among children in higher income categories. While the data do not indicate whether those who needed growth hormone were appropriately prescribed this medication, these results show that the proportion of patients with growth hormone prescriptions did not differ by income status. Perhaps most importantly, the group identified as highest risk for poor growth (i.e., the low income group) had higher proportions of growth hormone prescriptions.

These findings are consistent with prior studies showing SES in children with CKD and ESRD.[4,19–21] However, our study results are in contrast with an analysis of adults with CKD in the REGARDS (Reasons for Geographic And Racial Differences in Stroke)) study which found that household income was independently associated with GFR. [22] The present study did not find statistically significant differences by income in the decline of kidney function in these children and adolescents.

This study has several limitations. It is underpowered to detect statistical significance of these even moderately large effect sizes. In addition, income alone is an imperfect measure of SES. In our study, we considered low income as a surrogate for low SES, although we acknowledge that other important components are likely not captured in this variable. It is possible that mediating factors associated with low income, but not income per se, explain the observed associations with height, such as low parental education, ability to afford health care and medications, and/or exposure to psychosocial or environmental stressors. Also, since subjects entered the study with moderate to severe CKD, we cannot assess whether low income is a cause or a consequence of having a sick child. Additionally, we were unable to capture information on dietary factors that may significantly impact disease trajectory. Specifically, we did not collect 24-hour urine sodium or urea, so we could not assess

whether sodium or protein intake differed by income strata or were associated with BP control or GFR decline. Lastly, adherence data for medication use including growth hormone use was not available. It is possible that those in the lowest income category, while prescribed growth hormone, were unable to afford the medications or were otherwise non-adherent. This is a reasonable hypothesis given a body of literature suggesting low SES is associated with medication non-adherence, leading to poor health outcomes, at least among adult populations.[23]

It is also important to highlight that this study sample is only representative of a population receiving regular care from a pediatric nephrology clinic. Indeed, nearly all subjects had some form of health insurance. Therefore, we are unable to generalize these results to populations who are undiagnosed, not receiving sub-specialty care or who are uninsured: such children would likely have higher disease severity and progression. This form of selection bias would diminish the effect of SES on disease severity in this analysis. With insurance coverage expected to expand in coming years, it is nonetheless encouraging that children and adolescents in low income households, practically all of whom are insured (mostly with non-private insurance) and receiving regular specialized care, are not at substantially higher risk for accelerated GFR decline or poor BP control.

Despite few differences in co-morbidities and disease progression among children and adolescents with CKD, we found that those from lower income households may be at higher risk of impaired growth in contrast to those from higher income households, whose deficits diminished over time. Among high risk populations of children and adolescents with CKD, low SES should be an important clinical consideration for aggressive interventions, especially to promote growth.

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Table 1

Study sample characteristics, stratified by income category

	Annual Household Income			P-value*
	≥$75,000 (n = 158)	$30,000 to <$75,000 (n = 212)	<$30,000 (n = 202)	
Age (y)	12 [8, 14]	10 [7, 14]	10 [6, 13]	0.05
Male sex	68% (108)	61% (130)	58% (118)	0.2
Black race	7% (11)	20% (42)	39% (79)	<0.001
Hispanic ethnicity	6% (10)	9% (20)	25% (50)	<0.001
US resident	89% (140)	88% (187)	93% (188)	0.2
Health insurance				
Any	99% (157)	96% (203)	96% (193)	0.06
Private based	94% (148)	81% (164)	33% (64)	<0.001
Maternal education				<0.001
< high school	1% (2)	5% (10)	28% (55)	
High school or some college	41% (64)	67% (139)	63% (125)	
≥ College	57% (89)	29% (60)	9% (17)	
Abnormal birth history[a]	21% (33)	32% (68)	34% (69)	0.01
Body mass index (kg/m^2)	18.5 [16.5, 20.8]	18.2 [16.1, 21.3]	17.8 [15.9, 22.5]	0.8
CKD				
Glomerular	19% (30)	22% (46)	23% (47)	0.6
Diagnosed within 1 y of birth	57% (89)	47% (98)	55% (109)	0.1
Urine protein (mg/mg of Cr)	0.40 [0.15, 1.15]	0.43 [0.16, 1.11]	0.58 [0.18, 1.54]	0.2
Medication use				
Systemic corticosteroid	8% (13)	5% (10)	6% (13)	0.4
ACE inhibitor	50% (79)	48% (101)	47% (94)	0.8
ARB	13% (21)	9% (19)	11% (22)	0.4
Any antihypertensive	62% (98)	61% (130)	62% (126)	0.9
Comorbidities and outcomes				
Anemia	36% (57)	34% (71)	42% (84)	0.2
Hypertension	16% (25)	24% (49)	23% (44)	0.2
Elevated Ca x P	2% (3)	2% (4)	4% (8)	0.4
Hyperphosphatemia	11% (17)	18% (37)	17% (34)	0.2
Growth failure	13% (20)	14% (29)	17% (34)	0.5
No. of comorbities[b]				
1+	55% (80)	64% (126)	66% (123)	0.09
2+	20% (29)	22% (44)	27% (51)	0.3
3+	5% (7)	5% (10)	10% (19)	0.09
GFR (ml/min|1.73m^2)	45 [35, 61]	43 [33, 55]	44 [32, 58]	0.3
z Scores				

	Annual Household Income			P-value*
	≥$75,000 (n = 158)	$30,000 to <$75,000 (n = 212)	<$30,000 (n = 202)	
SBP	0.30 [−0.33, 0.88]	0.43 [−0.30, 1.30]	0.52 [−0.30, 1.26]	0.1
DBP	0.32 [−0.19, 1.13]	0.59 [0.02, 1.30]	0.56 [−0.05, 1.12]	0.1
Height	−0.71 [−1.33, 0.13]	−0.64 [−1.32, 0.11]	−0.71 [−1.63, 0.06]	0.4

Note: N = 572. Values for categorical variables are given as number (percentage); values for continuous variables, as median [interquartile range].P < 0.05 is statistically significant. Missing data: Hispanic ethnicity, n = 7; Private insurance, n = 19; Maternal education, n = 11; CKD diagnosed within 1 year of birth, n = 8; Urine protein, n = 22; Hypertension, n = 21; Elevated Ca × P, n = 14; Hyperphosphatemia, n = 14; Growth failure, n = 16; Comorbidities, n = 44.

*P-values based on Fisher's exact test for categorical variables, and Wilcoxon multiple comparisons rank-sum test for continuous variables.

[a]Premature birth, small for gestational age or low birth weight.

[b]Having anemia, hypertension, elevated Ca x P, hyperphosphatemia or growth failure.

Abbreviations: ACE, angiotensin-converting enzyme; ARB, angiotensin receptor blocker; GFR, glomerular filtration rate. CKD, chronic kidney disease; Ca x P, calcium-phosphorus product; Cr, creatinine; SBP, systolic blood pressure; DBP, diastolic blood pressure

Table 2

Descriptive statistics of longitudinal measurements, stratified by annual household income category.

	≥$75,000 (n = 158)	$30,000 to <$75,000 (n = 212)	<$30,000 (n = 202)
Follow-up (y)	3.12 [2.05, 4.07]	3.16 [2.17, 4.21]	3.13 [1.46, 4.17]
No. of observations			
1	11% (17)	9% (19)	15% (30)
2	13% (21)	11% (23)	13% (26)
3	16% (25)	20% (42)	16% (32)
4	28% (44)	28% (59)	23% (46)
5	22% (35)	18% (39)	21% (42)
6	9% (15)	13% (27)	12% (23)
7	1% (1)	1% (2)	0% (0)
Total no.	582	798	710
Empirical estimates			
GFR			
Median intercept *	46[36, 59]	43[33, 57]	46[34, 60]
Median annual change	−2.1%[−9.2%, 2.7%]	−3.8%[−10.8%, 1.4%]	−3.6%[−12.1%, 1.9%]
Correlation **	0.10	−0.06	0.12
SBP *z* scores			
Median intercept *	0.25[−0.25, 0.83]	0.51[−0.38, 1.13]	0.37[−0.32, 1.13]
Median annual change	−0.07[−0.31, 0.15]	−0.06[−0.29, 0.18]	−0.05[−0.24, 0.21]
Correlation **	−0.47	−0.51	−0.40
DBP *z* scores			
Median intercept *	0.32[−0.20, 1.03]	0.60[−0.06, 1.20]	0.50[−0.08, 1.11]
Median annual change	−0.07[−0.33, 0.13]	−0.07[−0.27, 0.15]	−0.07[−0.23, 0.20]
Correlation **	−0.58	−0.50	−0.51
height *z* scores			
Median intercept *	−0.73[−1.31, 0.11]	−0.62[−1.34, 0.14]	−0.75[−1.55, 0.002]
Median annual change	0.02[−0.08, 0.14]	0.01[−0.10, 0.13]	−0.02[−0.13, 0.11]
Correlation **	−0.29	−0.20	−0.18

Note: Unless otherwise indicated, values for categorical variables are given as number (percentage); values for continuous variables, as median [interquartile range].Empirical estimates of variables based on separate regressions fit to the data of each individual subject(log-linear regressions for GFR; linear regression for *z* scores for SBP, DBP, and height).

Abbreviations: GFR, glomerular filtration rate (in ml/min/1.73 m2); SBP, systolic blood pressure; DBP, diastolic blood pressure.

* Baseline level.

** Between Intercept and change.

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Table 3

Baseline levels and longitudinal changes using mixed-effects regression models.

	Income ≥ $75,000	Income $30,000 to <$75,000		Income <$30,000	
		Value	P*	Value	P*
GFR[a]					
Baseline level (95% CI)	44 (41, 47)	40 (38, 43)	0.03	43 (40, 46)	0.5
Annual Change (95% CI)	−1.9% (−4.0%, 0.3%)	−2.7% (−4.7%, −0.8%)	0.4	−2.3% (−4.5%, −0.1%)	0.7
SBP *z* scores[b]					
Baseline level (95% CI)	0.20 (−0.01, 0.41)	0.35 (0.16, 0.54)	0.2	0.18 (−0.03, 0.39)	0.9
Annual Change (95% CI)	−0.10 (−0.15, −0.04)	−0.06 (−0.11, −0.01)	0.4	−0.04 (−0.09, 0.01)	0.2
DBP *z* scores[b]					
Baseline level (95% CI)	0.33 (0.17, 0.50)	0.47 (0.32, 0.62)	0.1	0.30 (0.13, 0.45)	0.7
Annual Change (95% CI)	−0.10 (−0.15, −0.05)	−0.06 (−0.10, −0.01)	0.2	−0.04 (−0.08, 0.01)	0.05
Height *z* scores[c]					
Baseline level (95% CI)	−0.80 (−1.05, −0.56)	−0.77 (−1.00, −0.54)	0.8	−0.98 (−1.23, −0.73)	0.2
Annual Change (95% CI)	0.05 (0.01, 0.08)	0.01 (−0.02, 0.03)	0.07	−0.004 (−0.03, 0.03)	0.03

*P for comparison with ≥ $75,000 reference category.

[a]Adjusted for age, gender, race (black/not black), ethnicity (Hispanic/not Hispanic), glomerular/non-glomerular diagnosis, urine protein-creatinine ratio at baseline, having either anemia, hypertension, elevated Ca x P, hyperphosphatemia or growth failure at baseline, and all interactions with time.

[b]Adjusted for age, race (black/not black), ethnicity (Hispanic/not Hispanic), glomerular/non-glomerular diagnosis, GFR at baseline, urine protein-creatinine ratio at baseline, and having either anemia, hypertension, elevated Ca x P, hyperphosphatemia or growth failure at baseline.

[c]Adjusted for age, race (black/not black), ethnicity (Hispanic/not Hispanic), glomerular/non-glomerular diagnosis, abnormal birth history, GFR at baseline, urine protein: creatinine ratio at baseline, and having either anemia, hypertension, elevated Ca x P, hyperphosphatemia or growth failure at baseline.

Abbreviations: GFR, glomerular filtration rate(in ml/min/1.73 m2); SBP, systolic blood pressure; DBP, diastolic blood pressure. CI, confidence interval; Ca × P, calcium-phosphorus product.