Item S1.

Supplemental Methods

Design and Validation of the NGS Nephropathy Panel. Two different custom target enrichment kits were tested in the validation process (Agilent and Illumina). These kits were designed to cover the exonic region of a total of 301 genes including 265 clinical genes that have known association with nephropathies as well as 36 'experimental' genes that are suspected to be involved in nephropathy but have not been formerly documented to result in human disease. For the purposes of clinical testing, the assay will be divided into multiple smaller panels including those for steroid resistant nephrotic syndrome/FSGS, Alport syndrome, cystic kidney disease, ciliopathies, developmental disorders with renal manifestations, thrombotic microangiopathy, C3 glomerulonephritis, and storage diseases with renal manifestations. The full list of genes targeted is included in table *a*. In the end, the Agilent SureSelect kit was selected based on the increased reproducibility of the results as well as the significantly shorter processing time. The Agilent kit was then optimized to ensure maximum coverage of the targeted region to minimize co-capture of non-target or homologous pseudogene sequences as much as possible.

We utilized the well characterized HapMap reference sample NA12878 for validation. This genome was selected due to the availability of a highly confident "gold standard" set of reference calls through the National Institutes of Standards and Technology (NIST), Genome in a Bottle Consortium and Illumina.[1] Thus, assay performance could be easily determined across the target region. The DNASTAR software utilized facilitates validation using this genome through comparison of variant calls generated in our laboratory.

Paired-end sequencing was performed on the MiSeq system using the PE v3 150 cycle kit. A batch size of 8 samples was selected in order to provide the optimum depth of coverage across the target region. We ran a total of 8 validation runs using this setup which included 9 of the NA12878 reference sample. These validation reference samples had a mean depth of 237X. Based on the NA12878 accuracy data we determined that the sensitivity and specificity are optimized at a minimum depth of 16X and therefore set 20X as a minimum depth to consider an exon adequately covered in order to provide a margin of safety. DNA from five cases that had a known pathogenic variant within a gene in the panel were tested during the validation process and the pathogenic variant was correctly identified in all cases.

Utilization of assay on a series of renal biopsy samples. After assay validation, we sequenced extracted DNA from renal biopsy samples under an IRB-approved protocol (Schulman Associates IRB) in order to test the performance of the assay utilizing patient DNA. Since the current study was retrospective in nature and the study subjects were completely de-identified, the IRB waived the need for written consent from the patients. We sought to determine how commonly an identifiable genetic etiology of a patient's kidney disease can be detected in cases where the pathogenesis is undetermined after renal biopsy . We included biopsies from patients <40 years of age with either idiopathic focal segmental glomerulosclerosis or cases without FSGS that show at least moderate chronic injury with no discernable etiology either by biopsy or clinical parameters. A relatively young cutoff age was chosen for inclusion in an effort to enrich study population for cases with a monogenic etiology. Idiopathic focal segmental glomerulosclerosis was defined as biopsy evidence of FSGS in which the clinical indication for the biopsy was proteinuria. Patients with secondary etiologies for the FSGS were excluded. This would include biopsy evidence of other forms of glomerulopathy which commonly result in FSGS lesions (e.g. IgA nephropathy) or clinical evidence of a disease known to result in secondary FSGS. Cases without FSGS lesions were included if at least moderate chronic injury was present by renal biopsy (>30% interstitial fibrosis in the renal cortex) and there was no known clinical or pathologic etiology of the chronic injury.

A total of 50 consecutive renal biopsies with adequate DNA that met inclusion criteria were sequenced. DNA for each case was extracted from the frozen tissue leftover after immunofluorescence evaluation was complete.

The fastq files generated by the MiSeq were streamed to Illumina BaseSpace where the data was assembled with the BWA Genome Alignment Software and the variants called by GATK Variant Caller. This produced a VCF which can be imported into Illumina Variant Studio. The fastq files were also imported into DNASTAR's SeqMan NGen (SM NGen) assembler and the data interrogated in DNASTAR ArrayStar and SeqMan Pro. We found high correlation between the variant calls provided by these two software pipelines (>99.9%). Both the Illumina and DNASTAR programs allow filtering for rare variants. We filtered for coding variants with a minor allele frequency (MAF) less than <1% and also manually looked for coding variants in APOL1 and NPHS2 which are known to be pathogenic but have a MAF above the cutoff. Variants were considered disease causing under strict criteria as previously described[2] and in accordance with the recently published ACMG guidelines for the interpretation of sequence variants.[3] The FSGS cases were classified according to the Columbia Classification.[4]

Works Cited

1. Zook JM, Chapman B, Wang J, et al. Integrating human sequence data sets provides a resource of benchmark SNSNP and indel genotype calls. *Nat Biotechnol.* 2014; 32: 246-251.
2. Sadowski CE, Lovric S, Ashraf S, et al. A single-gene cause in 29.5% of cases of steroid-resistant nephrotic syndrome. *J Am Soc Nephrol.* 2015; 26: 1279-1289.
3. Richards S, Aziz N, Bale S, et al. Standards and guidelines for the interpretation of sequence variants: a joint consensus recommendation of the American College of Medical Genetics and Genomics and the Association for Molecular Pathology. *Genet Med.* 2015; 17: 405-424.
4. D'Agati VD, Fogo A, Bruijn JA, Jennette JC. Pathologic classification of focal segmental glomerulosclerosis: a working proposal. *Am J Kidney Dis.* 2004; 43: 368-382.

Table *a*. Genes included in the NGS nephropathy panel.

Location	Gene	Disease
1p21.3	ABCD3	Zellweger syndrome 2
17q23.3	ACE	Renal tubular dysgenesis
19q13	ACTN4	FSGS
9q34	ADAMTS13	TTP
1q42.13	ADCK3	SRNS
1q24	ADCY10*	Absorptive hypercalciuria type 2
1q42.2	AGT	Absence or paucity of proximal tubules, fetal anuria
3q24	AGTR1	Renal adysplasia
2q37.3	AGXT	Type I primary hyperoxaluria
6q23.3	AHI1	Joubert syndrome
2p13	ALMS1	Alstrom syndrome
9q22.33	ANKS6	Nephronophthisis
11q23-q24	APOA1	Amyloidosis
1q23.3	APOA2	Amyloidosis
19q13.2	APOE	Lipoprotien glomerulopathy
22q13.1	APOL1	FSGS
16q24	APRT	2-8 DHA deficiency
12q12-q13	AQP2	Bartter syndrome, type 4b
4q22.1	ARHGAP24*	Murine FSGS
Xq28	ARHGAP4	Nephrogenic diabetes insipidous
17q25.3	ARHGDIA	Congenital nephrotic syndrome
3q11.1	ARL13B	Joubert syndrome 8
3q11.2	ARL6	Bardet-Biedl
7q34	ATP6V0A4	Primary distal renal tubular acidosis
2q13.1	ATP6V1B1	Primary distal renal tubular acidosis
22q13.31	ATXN10	Nephronophthisis
Xq28	AVPR2	Nephrogenic diabetes insipidous
15q21-q22.2	B2M	Amyloidosis
17p11.2	B9D1	Dysplastic kidney, meckel's syndrome
19q13.2	B9D2	Cystic kidneys, meckel's syndrome
11q13	BBS1	Bardet-Biedl
12q21.2	BBS10	Bardet-Biedl
4q27	BBS12	Bardet-Biedl
16q21	BBS2	Bardet-Biedl
15q22.3-q23	BBS4	Bardet-Biedl
2q31.1	BBS5	Bardet-Biedl
4q27	BBS7	Bardet-Biedl
7p14	BBS9	Bardet-Biedl
2q33	BCS1L	Early onset Toni-Debré-Fanconi Syndrome
14q22-q23	BMP4	Renal hypodysplasia
1p32.1	BSND	Bartter syndrome with sensorineural deafness
1p36.12	C1QA	Systemic lupus erythematosus and glomerulonephritis
1p36.12	C1QB	Systemic lupus erythematosus and glomerulonephritis
1p36.11	C1QC	Systemic lupus erythematosus and glomerulonephritis
12p13	C1S	Atypical HUS
6p21.3	C2	Complement component 2 deficiency
19p13.3-p13.2	C3	Atypical HUS/C3 GN

12p13.31	C3AR1*	Atypical HUS
5p13.2	C5orf42	Joubert syndrome 17
1p32.1	C8A	Complement C8 alpha-gamma deficiency
5p14-p12	C9	C9 deficiency
8q22	CA2	Primary proximal renal tubular acidosis
3q13	CASR	Familial hypocalciuric hypercalcemia and neonatal severe primary hyperparathyroidism
4p15.32	CC2D2A	Joubert syndrome, meckel's syndrome
12q22	CEP83 (CCDC41)	Nephronophthisis
11p15.5	CD151	Nephropathy with pretibial epidermolysis bullosa and deafness
6p12	CD2AP	FSGS
1q32	CD46	Atypical HUS and C3 GN
9q22.33	CDC14B*	Zebrafish ciliopathy
1p36.1	CDC42*	Zebrafish ciliopathy
11q23.3	CEP164	Nephronophthisis 15
12q21.32	CEP290	Joubert syndrome, meckel's syndrome
7q323	CEP41	Joubert Syndrome 15
6p21.3	CFB	Atypical HUS/C3 GN
1q32	CFH	Atypical HUS/C3 GN
1q32	CFHR1	Atypical HUS/C3 GN
1q31.3	CFHR2	Atypical HUS/C3 GN
1q32	CFHR3	Atypical HUS/C3 GN
1q32	CFHR4	Atypical HUS/C3 GN
1q31.3	CFHR5	Atypical HUS/C3 GN
4q25	CFI	Atypical HUS/C3 GN
8q12.2	CHD7	CHARGE syndrome
7q35	CLCN1	recessive generalized myotonia congenita (Becker) and dominant myotonia (Thomsen)
Xp11.23-p11.22	CLCN5	Dent disease
1p36	CLCNKA	Bartter syndrome type 3, Bartter syndrome, type 4b
1p36	CLCNKB	Bartter syndrome, type 3
3q28	CLDN16	Familial hypomagnesemia with hypercalciuria and nephrocalcinosis
1p34.2	CLDN19	Familial hypomagnesemia with hypercalciuria and nephrocalcinosis
16p13.3	CLUAP1*	Zebrafish ciliopathy
2q36-q37	COL4A3	Alport
2q35-q37	COL4A4	Alport
Xq22	COL4A5	Alport
Xq22	COL4A6	Alport
4q21.23	COQ2	COQ2 nephropathy
9q34.11	COQ4*	No renal manifestations reported
14q24.3	COQ6	Steroid resistant nephrotic syndrome
16q21	COQ9	Coenzyme Q10 deficiency
17p12	COX10	Proximal tubulopathy
1p32	CPT2	Canntitine palmitoyltransferase II deficiency (myoglobinuria)
1q32	CR2	complement abnormalities/immunodefficiency
9q33.3	CRB2	Steroid res nephrotic syndrome
17p13	CTNS	Cystinosis
20q13.1	CTSA	Galactosialidosis
10p12.31	CUBN*	Megaloblastic anemia

8q21-q22	CYP11B2	Corticosterone methyl oxidase deficiency
2p25.1	CYS1*	murine cystic kidneys
5p13.1	DAB2*	Dent's disease
13q22	DACH1	Renal hypodysplasia
Xp22.11	DDX53	Nephrotic syn (ASN 2013)
17q22	DGKE	TMA and FSGS
2p13	DGUOK	recurrent rhabdomyolysis
1q32.1	DSTYK	CAKUT
11q21-q22.1	DYNC2H1	Multicystic kidneys
11q13.1	EFEMP2	cutis laxa
3q26.3-q28	EHHADH	Renal fanconi
4q25	ENPEP*	Kidney development in zebrafish
15q23-q25	ETFA	Glutaric acidemia type 2
19q13.3	ETFB	Glutaric acidemia type 2
4q32-q35	ETFDH	Glutaric acidemia type 2
4p16	EVC	Ellis van Creveld syndrome
4p16.2-p16.1	EVC2	Ellis van Creveld syndrome
14q22.3	EXOC5*	Pronephros development
8q13.3	EYA1	Branchio-oto-renal syndrome
5q35.3	F12	Heriditary angioedema
15q13.2-q13.3	FAN1	Karyomegalic nephropathy
4q35	FAT1*	Nephrophthisis and fsgs (ASN 2013)
14q32.1	FBLN5	Autosomal recessive cutis laxa type 1B
9q34	FCN1	Atypical HUS
4q28	FGA	Amyloidosis
12p13.3	FGF23	Hereditary hypophosphatemic rickets
8p11.23-p11.22	FGFR1	Kallmann syndrome 2 and hypophosphataemic rickets
2q34	FN1	Fibronectin glomerulopathy
5q13.2	FOXD1*	Kidney development in zebrafish
4q21.21	FRAS1	Fraser syndrome/CAKUT
13q13.3	FREM2	Fraser syndrome/CAKUT
11q23	FXYD2	Renal Hypomagnesemia-2
17q21	G6PC	Glycogen storage disease type I
2q24-q31	GALNT3	Tumoral calcinosis, hyperphosphatemic,
1q21	GBA	Gaucher disease
Xq22	GLA	Fabrys disease
7p21.3	GLCCI1*	Murine FSGS
16p13.3	GLIS2	Nephronophthisis
12q23.2	GNPTAB	I-cell/ mucolipidosis II
Xq26.1	GPC3	Simpson golabi behmel syndrome
13q32	GPC5*	Susceptibility to nephrotic syndrome
9q12	GRHPR	Primary hyperoxaluria
9q33.3	GSN	amyloidosis
10q24	HIF1AN*	Zebrafish ciliopathy
17q12	HNF1B	Renal cysts and diabetes syndrome, Glomerulocystic kidney disease
Xq26.1	HPRT1	Lesch-Nyhan syndrome
16q22	HSD11B2	Apparent mineralcorticoid excess syndrome
3q21	IFT122	Cranioectodermal dysplasia-1

16p13.3	IFT140	Nephronophthisis
17q11.2	IFT20*	Murine nephonophthisis
14q24.3	IFT43	Cranioectodermal dysplasia-3
3q25.33	IFT80	Asphyxiating thoracic dystrophy 2
13q12.1	IFT88*	Murine nephonophthisis
14q32.33	INF2	FSGS
9q34	INPP5E	Nephronophthisis
9q31	INVS	Nephronophthisis type 2
3q13.33; 3q21.1	IQCB1	Senior-Loken syndrome type 5
17q21.33	ITGA3	Nephrotic syndrome
10p13	ITGA8	Bilateral renal agenesis
10p11.2	ITGB1*	Murine cystic kidneys
17q25.3	ITGB4	Congenital FSGS
20p12.1-p11.23	JAG1	Alagille syndrome
Xp22.32	KAL1	Kallmann syndrome
11q24	KCNJ1	Bartter syndrome
17p11.2	KCNJ18	Thyrotoxic hypokalemic periodic paralysis
11p15.5	KCNQ1	Jervell and Lange-Nielsen syndrome and familial atrial fibrillation
5q31	KIF3A*	Murine cystic kidneys
15q26.1	KIF7	Nephronophthisis-medullary cystic disease
1q32	KISS1	Kallmann syndrome
13q12.1	KL	Hyperphosphatemic familial tumoral calcinosis
3p21	LAMB2	Pierson syndrome
16q22.1	LCAT	Lecithin cholesterol acyltransferase deficiency
9q33.3	LMX1B	Nail-patella
2q31.1	LRP2	Facio-oculoacoustico-renal syndrome
8q24.22	LRRC6*	Primary ciliary dyskinesia-19
12q15	LYZ	Amyloidosis
3q27-q28	MASP1	Atypical HUS
1p36.3-p36.2	MASP2	Atypical HUS
16p13.3	MEFV	Familial Mediterranean Fever
20p12.1-p11.23	MKKS	Bardet-Biedl syndrome type 6
17q22	MKS1	Meckel syndrome type 1
1p34.1	MMACHC	Cobalamin C deficiency
3q23	MRPS22	COMBINED OXIDATIVE PHOSPHORYLATION DEFICIENCY 5
1q21	MUC1	Medullary cystic kidney disease-1
6p12.3	MUT	Methylmalonic acidemia
12q24	MVK	AA amyloidosis, hyper IgD syndrome
22q13.1	MYH9	MYH9-related disorder
15q21-q22	MYO1E	FSGS
2p13.2	NAT8*	GWAS CKD suspected gene
2p13.1	NAT8B*	GWAS CKD suspected gene
15q24.2	NEIL1*	Candidate in SRNS patient
4q33	NEK1*	Murine cystic kidneys
17q11.1	NEK8*	Nephrophthisis
6p21.3	NEU1	Sialidosis
1q44	NLRP3	Muckle-Wells syndrome
1p13-p11	NOTCH2	Alagille syndrome

2q13.1	NPHP1	Senior-Loken syndrome type 1
3q22.1	NPHP3	Nephronophthisis type 3
1p36	NPHP4	Nephronophthisis type 4
19q13.1	NPHS1	Finnish nephropathy
1q25.2	NPHS2	Steroid resistant nephrotic syndrome
4q31.1	NR3C2	Pseudohypoaldosteronism type 1
Xq22	NXF5	FSGS
Xq25	OCRL	Lowe's/Dent
Xp22	OFD1	Oral-facial-digital syndrome
2p24.1	OSR1*	Kidney size and function
8q22.2	OSR2*	Murine kidney development
12q24	P2RX7*	Zebrafish polycystic disease model
10q24	PAX2	Oligmeganephronia
10p12.1	PDSS1*	Coenzyme Q10 deficiency and murine kidney disease
6q21	PDSS2	Coenzyme Q10 deficiency
7q21.2	PEX1	Zellweger's syndrome
1p36.32	PEX10	Zellweger's syndrome
17q12	PEX12	Zellweger's syndrome
2p16.1	PEX13	Zellweger's syndrome
1p36.22	PEX14	Zellweger's syndrome
11p11.2	PEX16	Zellweger's syndrome
1q23.2	PEX19	Zellweger's syndrome
8q21.1	PEX2	Zellweger's syndrome
22q11.21	PEX26	Zellweger's syndrome
6q24.2	PEX3	Zellweger's syndrome
12p13.31	PEX5	Zellweger's syndrome
6p21.1	PEX6	Zellweger's syndrome
6q23.3	PEX7	Zellweger's syndrome
Xp22.2-p22.1	PHEX	Hereditary hypophosphatemic rickets
10p13	PHYH	Refsum's disease
16p13.3	PKD1	ADPKD
4q22.1	PKD2	ADPKD
6p12.2	PKHD1	ARPKD
8q23	PKHD1L1	ARPKD
10q23	PLCE1	FSGS
6q26	PLG	Plasminogen deficiency
7q32-q33	PODXL*	FSGS
2p21	PREPL	Hypotonia-cystinuria syndrome
20p12.3	PROKR2	Kallmann syndrome
Xq22.3	PRPS1	5-PPS Superactivity and Arts Syndrome
12p13.3	PTPRO	FSGS
1q32	REN	Renal dysplasia
16q12.2	RPGRIP1L	Joubert syndrome type 7
8q23.1	RRM2B	Proximal Tubulopathy
16q12.1	SALL1	Townes-brocks
19q13.2	SARS2	Tubulointerstitial disease with salt wasting and hypomagnesaemia
4q21.1	SCARB2	FSGS
12p13	SCNN1A	Pseudohypoaldosteronism type 1

16p12.2-p12.1	SCNN1B	Pseudohypoaldosteronism type 1
16p12.2-p12.1	SCNN1G	Pseudohypoaldosteronism type 1
1q43	SDCCAG8	Retinal-renal ciliopathy
14q23.1	SIX1	Branchio-oto-renal syndrome
2p21	SIX2	Anomalous Kidney Development
19q13.32	SIX5	Branchio-oto-renal syndrome
15q15-q21.1	SLC12A1	Bartter syndrome
16q13	SLC12A3	Gitelman syndrome
6p21.3	SLC17A3*	Gout
6q13	SLC17A5	Salla disease
9p24	SLC1A1	Dicarboxylic aminoaciduria
11q13.1	SLC22A12	Renal hypouricemia type 1
3q26.1-q26.2	SLC2A2	Fanconi-Bickel
4p16.1	SLC2A9	Renal hypouricemia
5q35.3	SLC34A1	Fanconi renotubular syndrome
9q34	SLC34A3	hypercalciuria
11q23.2	SLC37A4	Glycogen storage disesease 1b
2p16.3	SLC3A1	Cystinuria
1q32.1	SLC41A1	Nephronophthisis
17q21.31	SLC4A1	Primary distal renal tubular acidosis
4q21	SLC4A4	Primary proximal renal tubular acidosis
16p11.2	SLC5A2	Glucosuria
5p15.33	SLC6A19	Hartnup's disease
14q11.2	SLC7A7	Lysinuric protein intolerance
19q13.1	SLC7A9	Cystinuria
17q25.1	SLC9A3R1	Hypophosphatemic nephrolithiasis/osteoporosis type 2
2q35-q37	SMARCAL1	Schimke immunoosseous dysplasia (SIOD)and renal dysfunction
9q34.2	SURF1	Leigh disease with cytochrome c oxidase deficiency
12q24.11	TCTN1	Joubert syndrome
12q24.31	TCTN2	Meckel syndrome type 8
10q24.1	TCTN3	Joubert syndrome 18
20p11.2	THBD	Atypical hemolytic uremic syndrome
11q12.2	TMEM138	Joubert Syndrome
11q13.1	TMEM216	Meckel-Gruber Syndrome Type 2
16q23.1	TMEM231	Joubert
2q33.2	TMEM237	Joubert syndrome-14
8q22.1	TMEM67	Meckel syndrome type 3 and Joubert syndrome type 6
12p13.2	TNFRSF1A	Amyloidosis, familial periodic fever
16p13.3	TRAP1	CAKUT and VACTERL
99q33.1	TRIM32	Bardet-biedel
11q22.1	TRPC6	Steroid resistant nephrotic syndrome
15q21	TRPM7*	Zebrafish kidney function
9q34	TSC1	Tuberous sclerosis
16p13.3	TSC2	Tuberous sclerosis
2q24.3	TTC21B	Nephronophthisis and fsgs
14q31.3	TTC8	Bardet-Biedl syndrome
18q12.1	TTR	Amyloidosis
16p12.3	UMOD	Medullary cystic kidney disease-2

22q13.31	UPK3A	Renal adysplasia
6p12	VEGFA*	Murine kidney disease
3p25.3	VHL	Von Hippel-Lindau
14q24.3-q31	VIPAS39	Arthrogryposis, renal dysfunction and cholestasis syndrome
15q26.1	VPS33B	Arthrogryposis-renal dysfunction-cholestasis
12p13.3	VWF	Von willebrand's disease
2p15	WDPCP	Bardet-Biedl syndrome 15
4p14	WDR19	Jeune syndrome
2p24.1	WDR35	Cranioectodermal dysplasia
11p13	WT1	Steroid resistant nephrotic syndrome
3q23-q24	WWTR1*	Zebrafish kidney development
22q13.2	XPNPEP3	Nephronophthisi
1p34	ZMPSTE24	Mandibuloacral dysplasia and restrictive dermopathy
3p21.3	ZMYND10	Primary ciliary dyskinesia
16q12	ZNF423	Nephronophthisis-14 and Joubert syndrome-19

*designates experimental gene

Abbreviations: ADPKD, autosomal dominant polycystic kidney disease; ARPKD, autosomal recessive polycystic kidney disease; CAKUT, congenital anomalies of the kidney and urinary tract; CKD, chronic kidney disease; FSGS, focal segmental glomerulosclerosis, GN, glomerulonephritis; SRNS, steroid resistant nephrotic syndrome; TTP, Thrombotic Thrombocytopenic Purpura

Table *b*. NA12878 variant call precision and reproducibility at a depth of 20X	
True positive rate	100%
False negative rate	0%
True negative rate	99.94%
False positive rate	0.018%
Variant reproducibility	99.6%

Table *c*. Percent of cases with an identified monogenic etiology by race.

Race	Mean age (range)	M/F	Total	Genetic etiology	No genetic etiology	% Genetic
Caucasian	22.0 (5-35)	7/8	15	5	10	33.3%
African American	24.5 (2-38)	11/10	21	16	5	76.2%
Hispanic	25.6 (3-37)	5/6	11	2	9	18.2%
Other	27.7 (21-37)	0/3	3	0	3	0%

Table *d*. Clinical and genetic findings in 50 consecutive renal biopsies studied.

Patient #	Clinicopathologic diagnosis	Serum Cr (mg/dL)	Proteinuria (grams/day)	Age	Gender	Ethnicity	Gene	Variant	Zygosity	Classified*	dbSNP ID
1	CKD	2.5	2.3	31	M	Af Am	APOL1	c.1024A>G (G1)	Homozygous	Variant pathogenic	rs73885319
2	CKD	3.7	1.8	37	F	Af Am	APOL1	c.1164delTTATAA (G2), c.1024A>G (G1)	Compound heterozygous	Variant pathogenic	rs73885319 and rs143830837
3	CKD	4	3.2	33	F	Af Am	APOL1	c.1024A>G (G1)	Homozygous	Variant pathogenic	rs73885319
4	CKD	3	4	38	M	Af Am	APOL1	c.1024A>G (G1)	Homozygous	Variant pathogenic	rs73885319
5	CKD		1.5	24	F	Af Am	APOL1	c.1024A>G (G1)	Homozygous	Variant pathogenic	rs73885319
6	CKD	1	2.7	15	F	Cau	COL4A6	c.2422G>A	Heterozygous	VUS	rs143895379
7	CKD	13.4		21	M	Af Am	CRB2	c.3373G>T and c.2519C>G	unknown	VUS	rs61740212 and rs543547663
8	CKD	2.4	0.4	25	F	Cau	NPHP1	whole gene deletion	Homozygous	Variant pathogenic	none
9	CKD	2.6	0.5	21	M	His				none	
10	CKD	1	1	12	F	His				None	
11	CKD	3.5	4.4	35	M	His				None	
12	CKD	1.5	2.8	32	F	His				None	
13	CKD	3.5	3	25	F	Mid East				None	
14	CKD	4.1	1	19	F	Cau				None	
15	FSGS	1.7	29	14	M	Af Am	APOL1	c.1164delTTATAA (G2)	Homozygous	Variant pathogenic	rs143830837
16	FSGS	2.7	12	36	M	Af Am	APOL1	c.1164delTTATAA (G2), c.1024A>G (G1)	Heterozygous	Variant pathogenic	rs73885319 and rs143830837
17	FSGS	0.5	3.3	10	F	Af Am	APOL1	c.1024A>G (G1)	Homozygous	Variant pathogenic	rs73885319
18	FSGS	1	2.5	17	F	Af Am	APOL1	c.1164delTTATAA (G2), c.1024A>G (G1)	Compound heterozygous	Variant pathogenic	rs73885319 and rs143830837
19	FSGS	0.7	2.2	28	F	Af Am	APOL1	c.1024A>G (G1)	Homozygous	Variant pathogenic	rs73885319
20	FSGS	1	4	29	F	Af Am	APOL1	c.1164delTTATAA (G2), c.1024A>G (G1)	Compound heterozygous	Variant pathogenic	rs73885319 and rs143830837

21	FSGS	0.5	5	2	M	Af Am	APOL1	c.1024A>G (G1)	Homozygous	Variant pathogenic	rs73885319
22	FSGS	17.7	5.4	31	M	Af Am	APOL1	c.1024A>G (G1)	Homozygous	Variant pathogenic	rs73885319
23	FSGS	1.8	5	37	F	Af Am	APOL1	c.1164delTTATAA (G2)	Homozygous	Variant pathogenic	rs143830837
24	FSGS	4.1	27	22	F	Af Am	APOL1	c.1164delTTATAA (G2), c.1024A>G (G1)	Compound heterozygous	Variant pathogenic	rs73885319 and rs143830837
25	FSGS	3.2	5.7	22	M	Af Am	APOL1	c.1164delTTATAA (G2), c.1024A>G (G1)	Compound heterozygous	Variant pathogenic	rs73885319 and rs143830837
26	FSGS	3.7	1.1	32	M	His	CFI	c.148C>G	Heterozygous	Variant Likely Pathogenic	rs144082872
27	FSGS	1.2	2.1	33	M	Cau	COL4A3	c.3829G>A	Heterozygous	VUS	rs190598500
28	FSGS	7.1	5	35	F	Cau	COL4A4	c.2563G>A	Heterozygous	Variant Likely Pathogenic	none
29	FSGS	8.2	3	24	M	Cau	COL4A5	c.4298G>T	Hemizygous	Variant pathogenic	rs281874735
30	FSGS	0.7	1.2	13	F	His	COL4A5	c.834+8C>A	Heterozygous	VUS	rs201717817
31	FSGS	0.9	9.4	5	M	Cau	COQ2	c.683A>G	Homozygous	Variant pathogenic	rs121918232
32	FSGS	0.8	5	5	F	Af Am	COQ6	c.145G>T and c.1184C>T	unknown	VUS	rs61743884 and rs34746680
33	FSGS	0.6	4	6	M	Cau	CRB2	c.1828C>T	Homozygous	VUS	rs145286619
34	FSGS	1.1	5	18	F	Cau	CRB2	c.471C>T and c.2680G>A	unknown	VUS	rs113157023 and rs144714250
35	FSGS	1.9	4.2	34	F	Cau	INF2	c.628G>A	Heterozygous	Variant Likely Pathogenic	none
36	FSGS	2	2.5	37	F	His	INF2	c.653G>A	Heterozygous	Variant pathogenic	rs267607183
37	FSGS	9.9		32	M	Cau	INF2	c.820G>A	Heterozygous	VUS	none
38	FSGS	7.7	6	37	F	Asian	PKD2	c.734A>G	Heterozygous	VUS	none
39	FSGS	11.1	6.8	31	M	His				none	

40	FSGS	1.4	6.3	21	F	Asian	None
41	FSGS	5	4.4	18	M	Af Am	None
42	FSGS	5	4	26	M	Af Am	None
43	FSGS	5.7	10	34	M	Af Am	None
44	FSGS	0.8	21	33	M	His	none
45	FSGS	0.3	6	3	F	His	none
46	FSGS	2.3	4	33	F	His	None
47	FSGS	14	2	26	M	Cau	None
48	FSGS	2.2	5	23	M	Cau	None
49	FSGS	10	4.5	21	M	Cau	None
50	FSGS	0.5	6	14	F	Cau	None

Abbreviations: Af Am, African American; Cau, Caucasian; CKD, chronic kidney disease; FSGS, focal segmental glomerulosclerosis; His, Hispanic; Mid East, Middle Eastern; VUS, variant of uncertain significance.

*Variant classification according to ACMG guidelines[1]

[1] Richards S, Aziz N, Bale S, et al. Standards and guidelines for the interpretation of sequence variants: a joint consensus recommendation of the American College of Medical Genetics and Genomics and the Association for Molecular Pathology. *Genet Med.* 2015; 17: 405-424.